REPRODUCTIVE HEALTH

Unit one Content..

Topic one: Standards of Reproductive health care in Kenya, Policies and initiatives related to Reproductive Health

**Definition of terms**

* ·         **Reproductive Health**is a state of complete physical, mental, emotional, and social well-being, and not merely the absence of disease or infirmity, in all matters relating to the reproductive system and its functions and processes.
* ·         **Reproductive Health Care**is the appropriate constellation of methods, technologies and services that will ensure reproductive health and well-being by preventing and solving problems related to human reproduction and sexuality.

**Basic Elements in Reproductive Health**

* ·         Ability - to reproduce, regulate fertility and enjoy healthy relationships
* ·         Success - result in child survival, growth and health development
* ·         Safety - Fertility regulation, pregnancy and child health

**Components of Reproductive Health**

* + ·         Family Planning
  + ·         Safe motherhood (Maternal and Newborn Health)
  + ·         Management of STIs/RTIs and HIV/AIDS
  + ·         Promotion of Adolescent and Youth Sexual and Reproductive Health
  + ·         Management of Infertility
  + ·         Gender Issues, Sexual and Reproductive Rights
  + ·         Reproductive Health Research
  + ·         Monitoring and Evaluation
  + ·         Community Reproductive Health
  + ·         Reproductive Tract Cancers
  + ·         Reproductive Needs of the Elderly (Andropause/Menopause)

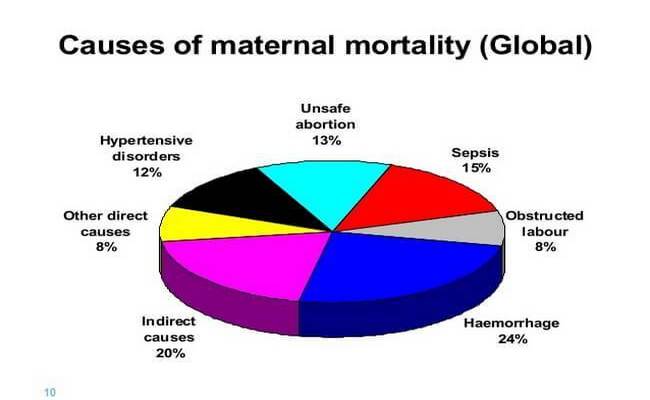
**Significance of Studying RH**

* + ·         Reproductive health is a right
  + ·         Lead a responsible and satisfied sex life.
  + ·         To reproduce and freedom to decide when and how often to do so
  + ·         To be informed about advantages, possible risks and side effects of contraceptives
  + ·          To have free, equal access to safe, effective, affordable, and acceptable method of fertility regulation.
  + ·          To get access to appropriate health service of good quality to go through safe pregnancy and child
* ·   Kenya has made steady progress in improving reproductive, maternal and child health outcomes in the last decade.
* ·   Child mortality has declined by over 20 percent since 2008 and the country achieved a total fertility rate of less than four.
* ·   Stunting, which remained stubbornly high over the past two decades has started to decline.
* ·   Six out of ten pregnant women now receive skilled care at child birth and over half receive postnatal care
* ·   However, despite this progress, Kenya could not achieve maternal and child health Millennium Development Goals (MDGs).
* ·   In Kenya today, many women, neonates, children, and adolescents continue to experience morbidity or die from preventable conditions that have proven and cost-effective interventions.
* ·   Access to quality Reproductive Maternal Newborn Child and Adolescent Health (RMNCAH) services remains a challenge across all levels of care, and inequities continue to persist among population sub-groups, and between rich and the poor.
* ·   The key supply side challenges include:
  + 1. sub-optimal functioning of the health system with uneven distribution of the health workforce as well as constraints in competency and motivation of the health care providers to provide quality care;
    2. insufficient financing and weak supply chain management resulting in missing critical inputs required for service delivery, especially essential commodities; and
    3. poor quality and utilization of routine data for evidence-based decision making.
  + · Sociocultural and economic barriers and constraints in physical access to health services continue to limit demand
  + ·  Globally, there is also a renewed momentum and support for RMNCAH as part of the Sustainable Development Goals (SDGs) and the updated Global Strategy for Women’s Children’s and Adolescent’s Health (2016-2030) which aims to achieve the highest attainable standard of health for all women, children and adolescents, and ensures that every newborn, mother and child not only survives, but thrives.
  + ·  Such growing national and international commitments provide an opportune time to enhance both domestic and external support for RMNCAH in Kenya to ensure smart, scaled-up, and sustained financing.
  + ·  Improving coverage for RMNCAH services is a priority for the Government of Kenya as is reflected in its Vision 2030, the Constitution of 2010 and the Health Sector Strategic and Investment Plan 2014-18.
  + ·   The Government has introduced new policies as well as initiatives such as Free Maternity Services, Elimination of User Fee for Primary Care and the Beyond Zero campaign to address the critical barriers.

### Status of Key Indicators

|  |  |  |  |
| --- | --- | --- | --- |
| **Key indicators** | **KDHS 2008/09** | **KDHS 2014** | **Sub-Saharan Africa Region** |
| NMR (per 1,000 Live births) | 31 | 22 | 31.1 |
| Infant mortality rate (per 1,000 Live births) | 52 | 39 | 61.1 |
| Under-five mortality rate (per 1,000 Live births) | 74 | 52 | 92.4 |
| Maternal mortality ratio (per 100,000 live births) | 488 | 360 | 510 |
| Total fertility rate (per women) | 4.6 | 3.9 | 5.0 |
| Teen pregnancy (%) | 18 | 18 | - |
| Children under-five stunted (%) | 35 | 26 | - |
| Deliveries attended by a skilled provider (%) | 43 | 62 | 48.6 |
| Pregnant women received any antenatal care (%) | 92 | 96 | 77 |
| Children received all basic vaccines (%) | 65 | 71 | - |
| Children under 6 months exclusively breastfed (%) | 32 | 61 | 37.7 |
| Contraceptive prevalence rate (any method) among  currently married women (%) | 46 | 58 | 23.6 |
| Unmet need for family planning (%) | 25 | 18 | 24.4 |

**Maternal Mortality**

* "A maternal death is defined as the death of a woman while pregnant or within 42 days of termination of the pregnancy, irrespective of the duration and site of pregnancy, from any cause related to or aggravated by the pregnancy or its management, but not from accidental or incidental causes." 

**Direct Causes of Maternal Mortality**

* These result from obstetric complications of pregnancy, labour and the puerperium and from interventions or any after effects of these events.
* The Five major causes of direct maternal deaths in order of frequency are:

o   Hemorrhage,

o   Sepsis,

o   Hypertensive disorders,

o   Complications of abortion and

o   obstructed labour

* **Incidental/Coincidental causes**
* are deaths that were neither due to direct nor indirect obstetric causes. E.g. car accident, fire burn, bullet injury, etc.
* **Indirect Causes of Maternal Mortality**
* They result from previously existing disease or disease that develops during pregnancy which was not due to direct obstetric causes, but which was aggravated by physiologic effects of pregnancy.
* These include:
  + - * o   Malaria,
      * o   HIV/AIDS
      * o   Anemia.
* **Maternal Morbidity**
* For every woman who dies another 30 suffer long term injuries and illness due to pregnancy and childbirth related complications.
* Maternal morbidity is any symptom or condition resulting from or made worse by pregnancy.
* **Severe maternal morbidity (Near Miss)** is defined as: “*any pregnant or recently delivered woman (within six weeks after termination of pregnancy or delivery), in whom immediate survival is threatened and who survives by chance or because of the hospital care she receives*.”

**Causes of Maternal Morbidity**

* **Infection**: There is high risk of infection of the genital organs (cervix, uterus, tubes, Ovaries and peritoneum) after prolonged labor.
* **Fistula:**are holes in the birth canal that allow leakage from the urethra, bladder or rectum into the vagina. Women present with continuous leakage of urine or feces or both. The commonest cause in our country is obstructed labor as opposed to surgery and cancer in the developed world.
* **Incontinence:**is involuntary leakage of urine.
* **Uterine prolapse**: the falling or sliding of the uterus from its normal position into the vaginal canal. Commonest predisposing factors include prolonged labor, heavy exercise, multiple childbirths, etc.
* **Infertility:**inability to become pregnant for a year despite unprotected sexual intercourse.
* **Nerve Damage:**As a result of prolonged labor, there may be compression or damage of the nerves in the pelvis (Sciatic nerve) may result in foot drop and contractures.
* **Psychosocial problems:**such as anxiety, depression, and psychosexual problem. Others Include pain during intercourse, low back pain, anemia, etc

* **Underlying Causes of Maternal & Neonatal Mortality (The Three Delays)**
* There are three distinct levels of delay which contribute to maternal morbidity and mortality: (Thadaseus and Maine, 1994):
  + **Delay in deciding to seek appropriate care**. This could be due to: socio-cultural barriers, Failure to recognize danger signs, failure to perceive severity of illness, and cost considerations
  + **Delay in reaching an appropriate health care facility**. This is due to: long distance to a facility, poor condition of roads, lack of transportation and cost considerations
  + **Delay in receiving adequate emergency care at the facility**. This may be due to: Shortage of staff, supplies and basic equipment; unskilled personnel, user fees among others

* **Risk factors affecting maternal health**
  + **Socio-cultural factors:**lack of literacy, early marriage, early childbirth, harmful traditional practices including female genital mutilation, etc.
  + **Economy:**Socio economic status affects women’s status by affecting their decision- making roles in the community, educational status, health service accessibility, prostitution, etc.
  + **Inadequate Health Service Coverage:**More than 70% mothers do not get care during pregnancy and more than 90 % of deliveries are unattended. This can be due to lack of transportation, distance from health facilities, small number of health facilities, lack of knowledge about importance of health services, lack of money, and lack of family support
  + **Psychological factors**: Fear of childbirth, too many pregnancies, death of other children illness in family. Women are at great risk of depression after sexual abuse, divorce as a result of marriage without consent and after Vesico-Vaginal/Recto- Vaginal fistula (VVF/RVF).
  + **Health and nutrition services:**The health status of women who are not getting adequate nutrients and proper reproductive health services could be affected
  + **Interaction with providers:**Some health care providers are not empathetic and caring. They do not respect women's cultural preferences for privacy, birth position, or the preference for treatment by women providers.
  + **Gender Discrimination**:  The family may give more attention to a male child with regard to education, nutrition; may want the mother to produce boys. Policy decisions, inheritance etc.
* **Maternal Morbidity**
* For every woman who dies another 30 suffer long term injuries and illness due to pregnancy and childbirth related complications.
* Maternal morbidity is any symptom or condition resulting from or made worse by pregnancy.
* **Severe maternal morbidity (Near Miss)** is defined as: “*any pregnant or recently delivered woman (within six weeks after termination of pregnancy or delivery), in whom immediate survival is threatened and who survives by chance or because of the hospital care she receives*.”

**Causes of Maternal Morbidity**

* **Infection**: There is high risk of infection of the genital organs (cervix, uterus, tubes, Ovaries and peritoneum) after prolonged labor.
* **Fistula:**are holes in the birth canal that allow leakage from the urethra, bladder or rectum into the vagina. Women present with continuous leakage of urine or feces or both. The commonest cause in our country is obstructed labor as opposed to surgery and cancer in the developed world.
* **Incontinence:**is involuntary leakage of urine.
* **Uterine prolapse**: the falling or sliding of the uterus from its normal position into the vaginal canal. Commonest predisposing factors include prolonged labor, heavy exercise, multiple childbirths, etc.
* **Infertility:**inability to become pregnant for a year despite unprotected sexual intercourse.
* **Nerve Damage:**As a result of prolonged labor, there may be compression or damage of the nerves in the pelvis (Sciatic nerve) may result in foot drop and contractures.
* **Psychosocial problems:**such as anxiety, depression, and psychosexual problem. Others Include pain during intercourse, low back pain, anemia, etc

* **Underlying Causes of Maternal & Neonatal Mortality (The Three Delays)**
* There are three distinct levels of delay which contribute to maternal morbidity and mortality: (Thadaseus and Maine, 1994):
  + **Delay in deciding to seek appropriate care**. This could be due to: socio-cultural barriers, Failure to recognize danger signs, failure to perceive severity of illness, and cost considerations
  + **Delay in reaching an appropriate health care facility**. This is due to: long distance to a facility, poor condition of roads, lack of transportation and cost considerations
  + **Delay in receiving adequate emergency care at the facility**. This may be due to: Shortage of staff, supplies and basic equipment; unskilled personnel, user fees among others

* **Risk factors affecting maternal health**
  + **Socio-cultural factors:**lack of literacy, early marriage, early childbirth, harmful traditional practices including female genital mutilation, etc.
  + **Economy:**Socio economic status affects women’s status by affecting their decision- making roles in the community, educational status, health service accessibility, prostitution, etc.
  + **Inadequate Health Service Coverage:**More than 70% mothers do not get care during pregnancy and more than 90 % of deliveries are unattended. This can be due to lack of transportation, distance from health facilities, small number of health facilities, lack of knowledge about importance of health services, lack of money, and lack of family support
  + **Psychological factors**: Fear of childbirth, too many pregnancies, death of other children illness in family. Women are at great risk of depression after sexual abuse, divorce as a result of marriage without consent and after Vesico-Vaginal/Recto- Vaginal fistula (VVF/RVF).
  + **Health and nutrition services:**The health status of women who are not getting adequate nutrients and proper reproductive health services could be affected
  + **Interaction with providers:**Some health care providers are not empathetic and caring. They do not respect women's cultural preferences for privacy, birth position, or the preference for treatment by women providers.
  + **Gender Discrimination**:  The family may give more attention to a male child with regard to education, nutrition; may want the mother to produce boys. Policy decisions, inheritance etc.

### The Kenya Maternal and Newborn Health Model (2009)

The Kenya Maternal and Newborn Health (MNH) Pillars

1. **Family planning and pre-pregnancy care**: To  ensure  that  individuals  and  couples  have  the information and services to plan the timing, number and spacing of pregnancies.
2. **Focused Antenatal Care** – To prevent complications where possible and ensure that complications of pregnancy are detected early and treated appropriately.
3. **Essential Obstetric Care**  –  To  ensure  that  essential  care  for  the  high-risk  pregnancies  and complications is made available to all women who need it.
4. **Essential Newborn Care** – To ensure that essential care is given to newborns from the time they are born up to 28 days in order to prevent complications that may arise after birth
5. **Targeted Postpartum Care**– To prevent any complication occurring after childbirth and ensure that both mother and baby are healthy and there is no transmission of infection from mother to child.
6. **Post Abortion Care  –**  to  provide  clinical  treatment  to  all  women  and  girls  seeking  care,  for complications of incomplete abortion and miscarriage as well as counselling and contraceptives.

(Note that HIV services  are  now  integrated  into  ALL  the  pillars  of  MNH  and  clean  and  safe delivery is part of Essential Obstetric Care)

**Skilled Attendance**

* Evidence has shown that there are 2 key interventions that improve maternal health and reduce maternal mortality, namely:
  + Skilled attendance at delivery (skills, numbers, enabling environment) and
  + availability of Emergency Obstetric Care.

 The term "skilled attendant" refers exclusively to ***people with midwifery skills (e.g. doctors, midwives, nurses, clinical officers) who have been trained to proficiency in the  skills  necessary  to  manage  normal  deliveries  and  diagnose  or  refer obstetric  complications.***

**Enabling Environment**

* To ensure effective and efficient service delivery, the skilled attendant requires an enabling environment.
* There is need for appropriate infrastructure as well as ensuring that the continuum of care is connected by an effective  referral  system,  and  supported  by  adequate  supplies, equipment, drugs,  good management and  supportive  supervision.

**Referral Systems**

* A key aspect in  ensuring  a  good  maternal  health  service  is  a  functional  referral  system.
* Access  to  a telephone and/or vehicle, with emergency funds or fuel to transfer urgent cases day or night is extremely important.
* Good record keeping and use of detailed referral letters will assist in reducing delay in the care for women with obstetric emergencies and severely ill newborns.
* The referring unit should be aware of the capacity of the referral point to manage the client being referred.

**Community Action, Partnerships**

* Involving  community  members  (particularly  women  and  their  families,  health  care  providers,  and  local leaders) in efforts to improve maternal health helps to ensure programme success;
* Community education about obstetric complications and when and where to seek medical care is important to ensure birth planning/ use of birth preparedness cards, early recognition of complications and prompt care-taking behaviour

**Male Involvement and Participation**

* it is evident that for successful programme implementation, male participation in  MNH  results  in  good  outcomes  for  both  mother  and  baby.
* Male involvement and participation  is  critical  in  addressing  the  first  and  second  delay.
* In the  Kenyan  context,  men  have  the resources and are the main decision makers in the families and communities on issues relating to MNH.

**Equity for All**

* Rights based perspective helps legitimize prioritization of women’s health.
* It focuses attention on social, economic and geographic inequities.
* Strong political  support  and  national  ownership  are  essential  to create  enabling  policies  to  attract  resources  for  maternal  and  newborn  health  and  to  ensure  those resources reach groups with the highest maternal mortality and morbidity.

**Reproductive Rights**

* Health care providers should appreciate that most maternal  and  neonatal  deaths  are  avoidable,  and therefore  maternal  and  newborn  health  must  be  given  its  due  prominence.
* Safe Motherhood is  a  basic human right as women are entitled to enjoy a safe pregnancy and childbirth.

**Emergency Obstetric Care**

* Emergency Obstetric Care refers to a set of minimal health care elements, which should be availed to all women during pregnancy and delivery.
* It includes both life-saving and emergency measures e.g. Caesarean section, manual removal of placenta, etc, as well as non-emergency measures (e.g. use of the partograph  to  monitor  labour,  active  management  of  the  third  stage  of  labour,  etc.).
* Emergency  Obstetric  Care functions  are  generally  categorized  as  Basic  Emergency  Obstetric  Care  (BEmOC)  and  Comprehensive Emergency Obstetric care (CEmOC).
* Basic Emergency Obstetric Care Include:
  + - Administration of IV antibiotics.
    - Administration of magnesium sulphate.
    - Administration of parental oxytocics.
    - Performing manual removal of the placenta.
    - Performing removal of retained products.
    - Performing assisted vaginal delivery (e.g. by vacuum extraction).
    - Performing newborn resuscitation
* Comprehensive Emergency Obstetric Care
  + - includes all the seven above, PLUS:
    - Performing surgery (Caesarean section), including provision of emergency obstetric anaesthesia.
    - Administration of blood transfusion.

**Client Rights**

* Right to Information
  + All members of the community have a right to information on the benefits of reproductive health  including Maternal and Newborn health for themselves and their families. They also have a right to  information on how to access the services.
* Right to Access
  + All members of the community have a right to receive services from reproductive health / MNH programs,  regardless  of  their  socio-economic  status,  political  affiliations,  religious  beliefs,  ethnic origin,  marital  status  or  geographical  location.    Access  includes  freedom  from  barriers  such  as policies, standards and practices, which are not scientifically justifiable.
* Right of choice
  + Individuals and couples have the right to decide freely where to obtain RH /MNH services.
* Right to safety
  + Clients have a right to safety in the practice of MNH
* Right to Privacy
  + Clients  have  a  right  to  privacy  while  holding  conversation  with  service  providers  and  while undergoing physical examination.
* Right to Confidentiality
  + The client should be assured that any information she/he provides or any details of the service received will not be communicated to other parties without her/his consent.
* Right to Dignity
  + Reproductive Health /MNH clients have a right to be treated with courtesy, consideration, and attentiveness and with full respect of their dignity regardless of their level of education, social status or any other characteristics, which would single them out or make them vulnerable to abuse.
* Right to Comfort
  + Clients have a right to comfort when receiving services. This can be ensured by providing quality services in hygienically safe and conveniently located service delivery sites.
* Right to Continuity of Care
  + Clients have a right to receive services and reliable supply of RH /MNH commodities and drugs for as long as they need them.
* Right of Opinion
  + Clients have a right to express their views freely on the services they receive.
* **Providers’ Rights**
* Training
  + To continuously have access to the knowledge and skills needed to perform all the tasks required of them.
* Information
  + To be kept informed on issues related to their duties
* Infrastructure
  + To have appropriate physical facilities and organization to provide services at an acceptable level of quality.
* Supplies
  + To receive continuous and reliable supplies and materials required for providing reproductive health services at acceptable standards of quality.
* Guidance
  + To receive clear, relevant and objective guidance.
* Back up
  + To be reassured that whatever the level of care at which they are working they will receive support from other individuals or units.
* Respect
  + To receive recognition of their competence and potential, and respect for their human needs.
* Encouragement
  + To be given stimulus in the development of their potential, initiative and creativity.
* Feedback
  + To receive feedback concerning their competence and attitudes as judged by others.
* Self–expression
  + To express their views freely, concerning the quality and efficiency of the reproductive health program.

### Policies and initiatives related to Reproductive health

**THE SAFE MOTHERHOOD INITIATIVE (SMI)**

* **Safe motherhood** is women’s ability to have a safe and healthy pregnancy and delivery
* The Global Safe Motherhood Initiative launched in Nairobi in 1987 aimed at reducing the burden of maternal deaths and ill health in developing countries.
* The Safe Motherhood Initiative differed from other health initiatives in that it focused on the well-being of women as an end in itself.
* In the SMI, the prevention of the death of a pregnant woman is considered to be the key objective, not because death adversely affects children and other family members but because women are intrinsically valuable (Thaddeus and Maine 1994).
* It underscored the fact that Safe motherhood is a basic human right

**Summary of SMI Events**

* International Conference for Population and Development (ICPD)
* The 1994 International Conference on Population and Development in Cairo recommended to the international      community      a      set      of      important      population      and      development      objectives.
* The Programme of Action was striking for the attention it devoted to the issue of women’s health.
* It also included goals with regard to education, especially for girls, and for the further reduction of infant, child and maternal mortality levels.

**International Conference For Population and Development (ICPD)**

* For Kenya, the ICPD recommendations were then translated  into  the  National  Reproductive  Health Strategy (NRHS 1997 – 2010) and implementation plan whose goal was to reduce maternal, perinatal and neonatal morbidity and mortality.
* Another  event  that  followed  the  ICPD  was  the  Millennium  Declaration  in  2000  and  the  development  of  goals  (MDGs)  with  indicators.

**National Reproductive Health Policy 2007**

* The goal of the RH policy is to enhance the Reproductive Health status of all Kenyans through:
  + Increased equitable access to RH services
  + Improved quality, efficiency and effectiveness of service delivery at all levels
  + Improved responsiveness to clients’ needs
* The main objective for Safe Motherhood in this RH Policy is to reduce maternal, peri-natal and neonatal morbidity and mortality in Kenya
* The National Health Sector Strategic Plan (NHSSP II)-2005-2010
* The aim of NHSSP II was to reverse the decline in the health status of Kenyans through an efficient, high quality health care system that is accessible, equitable and affordable for every Kenyan household.
* A major feature of the NHSSP is the introduction of the Kenya Essential Package for Health (KEPH), which focuses on the health needs of individuals through the six stages of the human life cycle.
* The strategic plan emphasizes strong community involvement in health care through the community Strategy
* National Reproductive Health Strategy (NRHS): 2009- 2015
* This is a revision of the NRHS  1997-2010 and includes issues and challenges that had not been incorporated in the original strategy.
* The revision was also necessary in order to align it to the National RH Policy - 2007.

**The KEPH Life-Cycle Cohorts**

* ·         They are delineated in the NHSSP II as follows

o   Cohort 1: -Pregnancy, delivery and the newborn child (up to 2 weeks of age)

o   Cohort 2: - Early childhood (3 weeks to 5 years)

o   Cohort 3: - Late childhood (6-12 years)

o   Cohort 4: - Adolescence (13-24 years)

o   Cohort 5: - Adulthood (25-59 years)

o   Cohort 6: - Elderly (60 years and over)

**Levels of Care in KEPH**

* ·         The KEPH approach is not only limited to a definition of the target groups in terms of life-cycle cohorts.
* ·         It also defines where the health services will be delivered.
* ·         Under KEPH, promotive, preventive and curative services are provided at six levels of care:

o   Level 6 : tertiary hospitals

o   Level 5: secondary hospitals

o   Level 4: primary hospitals

o   Level 3: health centres, maternity, nursing homes

o   Level 2: dispensaries / clinics

o   Level 1: villages/household/families/individuals

NB

* ·         As a result of the implementation of the constitution of Kenya 2010, health functions have been devolved to the county.
* ·         In the structure, County Health Services are organized around three levels of care:

o   Community,

o   Primary care, and

o   Referral services.

**Annual Operational Plans (AOPs)**

* The Annual Operational Plans (AOPs) translate Kenya Essential Package for Health and the National Health Sector Strategic Plan II 2005-2010 into ‘actionable’ operational plans.
* AOPs also improve the planning process within the Ministry in particular highlighting the need for
  + - * + o   Improved coordination and decision-making
        + o   Elimination of duplication of activities and
        + o   More efficient use of available resources

**The Community Strategy**

* The community-based approach, is the mechanism through which households and communities take an active role in health and health-related development issues.
* Initiatives outlined in the approach target the major priority health and related problems affecting all cohorts of life at the community and household levels – level 1 of the KEPH-defined service delivery.

**Vision 2030**

* Kenya Vision 2030 is the country’s new development blueprint covering the period 2008 to 2030.
* The vision is based on three “pillars” namely;
  + - o   the economic pillar,
    - o   the social pillar and
    - o   the political pillar.
* Health is part of the social pillar.
* To improve the overall livelihoods of Kenyans, the country aims to provide an efficient and high-quality health care system with the best standards.
* This is in order to reduce health inequalities and improve indicators in key areas where Kenya is lagging, especially in lowering infant and maternal mortality.
* Specific strategies include:
  + - o   provision of a robust health infrastructure network;
    - o   improving the quality of health service delivery to the highest standards and promotion of partnerships with the private sector.
* In-addition the Government has put in place health financing mechanisms to make quality MNH services affordable and accessible to all especially the poor and vulnerable women.
* These include the provision of free MNH /FP services at the lower levels, National Health Insurance Fund (NSSF), Health Sector Support Fund (HSSF), Hospital Management Support Fund (HMSF), FIF, Voucher system /Output Based Aid (OBA).
* The government is also encouraging initiatives that promote community-based health financing.

**Assignment: study the following policies/initiatives**

* The Kenya Constitution (2010) calls for the highest attainable standard for health including reproductive health for all Kenyans
* The Kenya Health Policy (2014-30) commits to strengthening the health care system and service delivery.
* Kenya Health Sector Strategic and Investment Plan (2014-18) (MOH  2014A, B, MOH  2012).
* Free Maternity Care
* Elimination of User Fee for Public Primary Health Care Services,
* Beyond Zero campaign.
* Kenya RMNCAH investment framework
* Universal Health Coverage

**Millennium Development Goals (MDGs)**

* MDGs were set by all Government leaders at the UN Millennium Summit, September 2000)
* All UN organisations decided to be guided by MDGs in their future action: unity of purpose, coherent action, synergies and strategic approaches by the UN system as a whole (guided by CEB)
* Leaders pledged to strive, individually and collectively, towards these goals through international, regional and national action, concerted by the UN.

**Why the MDGs ?**

* The 1990s was a decade of faltering progress on:
* Under-5 mortality rate
* Maternal mortality rate
* Child malnutrition
* Water and sanitation
* Income poverty
* Primary education
* **MDGs were meant to accelerate progress**

**Millennium Development Goals (MDGs)**

·         Goal 1. Eradicate extreme poverty and hunger

·         Goal 2. Achieve universal primary education

·         Goal 3. Promote gender equality and empower women

·         Goal 4. Reduce child mortality

·         Goal 5. Improve maternal health

·         Goal 6. Combat HIV/AIDS, malaria and other diseases

·         Goal 7. Ensure environmental sustainability

·         Goal 8. Develop a Global Partnership for Development

**MDGS related to RH**

·         Goal 3 Promote gender equality & empower women

·         Goal 4 Reduce child mortality by 2015 the no at 1990

·         Goal 5 Improve maternal health

**Progress of MDGs Related To RH 2014**  
**Infant Mortality Rate**

* The KDHS  2014 shows   a   decrease   in the   infant mortality rate from 52 to 39 per 1,000 live births, and a decrease in the under-five mortality rate from 74   to   52   per   1,000   live   births between 2008 and 2014.
* These declines have been driven mainly by:
  + o   Enhanced use of mosquito nets,
  + o   Increases in antenatal care,
  + o   skilled attendance at childbirth,
  + o   Postnatal care,
  + o   Contraceptive use,
  + o   exclusive breastfeeding practices and
  + o   a decrease in unmet family planning (FP) needs,
  + o   as well as overall improvements in other social indicators such as education and access to water

**Neonatal Mortality Rate**

* The NMR declined from 31 per 1,000 births to 22 per 1,000 live births between 2008-2014.
* However, during the past decade, the NMR exhibited the slowest decline.
* Further reductions in infant and child mortality require steeper decline in the NMR, which is closely linked to improvements in maternal health services including intrapartum care.

**Maternal Health**

* The MMR of 362 per 100,000 live births estimated by KDHS in 2014 is still high
* Coverage/utilization indicators also show some improvements but much more needs to be done to address inequities and to reach Universal Health Care (UHC).
* The contraceptive prevalence rate (CPR, any method) among married women has increased to 58% in 2014 from 46% in 2008/9 with a decline in unmet need for FP.
* Nearly two thirds (61%) of births took place in a health facility and 62% of pregnant women were delivered by a skilled attendant.
* Postnatal care (PNC) increased from 42% in 2009 to 51% in 2014
* The total fertility rate has declined from 4.6 in 2008/9 to 3.9 in 2014; however, there has been no change in teen pregnancy with one in five (18%) adolescents in the 15-19 years age group having started child bearing due to early marriage, high unmet need for contraception and poor access to FP services.
* Nutritional status of children under-five has improved with a decline in stunting from 35% in 2008/9 to 26% in 2014.
* However, one out of every four children still remain shorter for their age, a factor that adversely affects their future health, well-being and economic productivity.
* Nearly half (48%) of households have access to an insecticide treated net;
* 53% of women and 46% of men were tested for HIV  in  the past 12 months and received the test results.
* Overall immunization coverage for basic vaccines   increased   from   65.3 percent 2008/9 to 71.3 percent in 2014 and coverage for measles, pentavalent and pneumococcal vaccine remained high.
* Prevalence of exclusive breastfeeding among children under 6 months has nearly doubled from 32 percent to 61 percent

**SUSTAINABLE DEVELOPMENT GOALS (SDGs) - The 2030 Agenda for Sustainable Development**

* A set of 17 goals for the world’s future, through 2030
* Backed up by a set of 169 detailed Targets
* Negotiated over a two-year period at the United Nations
* Agreed to by nearly all the world’s nations, on 25 Sept 2015

**SDGs:**

#1: End poverty in all its forms everywhere

#2: End hunger, achieve food security and improved nutrition and promote sustainable agriculture

#3: Ensure healthy lives and promote well-being for all at all ages

#4: Ensure inclusive and quality education for all and promote lifelong learning

#5: Achieve gender equality and empower women and girls

#6: Ensure access to water and sanitation for all

#7: Ensure access to affordable, reliable, sustainable and modern energy for all

#8: Promote inclusive and sustainable economic growth, employment and decent work for all

#9: Build resilient infrastructure, promote sustainable industrialization and foster innovation

#10: Reduce inequality within and among countries

#11: Make cities inclusive, safe, resilient and sustainable

#12: Ensure sustainable consumption and production patterns

#13: Take urgent action to combat climate change and its impacts\*

#14: Conserve and sustainably use the oceans, seas and marine resources

#15: Sustainably manage forests, combat desertification, halt and reverse land degradation, halt biodiversity loss

#16: Promote justice, peaceful and inclusive societies

#17: Revitalize the global partnership for sustainable development

**What is New and Different about the 17 SDGs?**

* These Goals apply to *every* nation … and every sector. Cities, businesses, schools, organizations, *all* are challenged to act. This is called **Universality**
* The Goals are all inter-connected, in a system. We cannot aim to achieve just one Goal. We must achieve them all. This is called I**ntegration**
* Achieving these Goals involves making very big, fundamental changes in how we live on Earth. This is called **Transformation**
* The vision is for the goals to promote sustainable development and poverty eradication.

The first 16 goals address priority areas that:

* o   Increase the ambition/ improving and sustaining current achievements on existing MDG goals (poverty, health education, gender) with added dimensions on
* o   Economic sustainability (inclusive growth, jobs, infrastructure, industrialization)
* o   Environmental sustainability (climate change, oceans and land-based ecosystems, sustainable consumption and production)
* o   All held together by the glue of ‘peaceful and inclusive societies for sustainable development’ (governance agenda, rule of law, violence).

The 17thgoal covers means of implementation (finance, trade, technology, capacity building, partnerships, and data)

**The SDGs versus MDGs**

* *Key Strengths of the proposed SDGs*include: -
* + The notion of **leaving no one behind**– with many targets aspiring zero/fully coverage (raising the ambition of the MDGs)
  + **Stand-alone goal of Inequality (**within and between countries)
  + **Stand-alone goal on** **gender inequality**, including ending of all forms of violence, discrimination, child marriages, and female genital mutilations

**The SDGs versus MDGs**

* **Environmental issues are strongly represented**– fulfilling ***a long-sought marriage***between development and environment (climate change, marine and land base ecosystems, and sustainable consumption and production)
* **Governance - for the first time**– incorporating a goal and targets on ***governance and peaceful societies***(legal identity, tackling corruption and bribery etc)
* **Participatory/Inclusiveness Process in formulation of the SDGs**: The participation and buy in of a wide range of stakeholders including member states and non-governmental organizations
* The broad nature of the SDG is also a reflection of the nature of challenges facing the world today

Top of Form

**Kenya SDG Targets For RH**

* Kenya RMNCAH Investment Framework (January 2016)
* The Kenya RMNCAH investment framework sets ambitious targets to increase:
  + - * o   skilled deliveries to 87%,
      * o   4+ ANC visits to 69%,
      * o   full immunization to 76 %,
      * o   contraceptive use by currently married women in reproductive age to 73%, and
      * o   pregnant women tested for HIV who received results and post-test counseling to 75% by 2020 from the baselines of the Kenya Demographic and Health Survey 2014 with enhanced focus on quality of services.
* It also aims to reduce stunting to 19%, teenage pregnancy to 11%, and contribute to decrease in neonatal mortality to 18%.
* The absolute number of deaths of children under-five years is projected to reduce from 77,761 to 48,590 and maternal from 5,453 to 3,276 between 2014/15 and 2019/20.
* Finally, the framework aims to ensure that at least three out of four births will be registered, thereby providing more robust denominators to effectively plan and monitor RMNCAH service delivery.
* Progress on improvements in quality, productivity and efficiency will be tracked through strengthened routine data, independent surveys, and implementation research of innovations.
* Impact and outcome level indicators in reducing neonatal, infant, and under-five mortality rates, and maternal mortality ratio (MMR) will be tracked through population-based surveys such as the KDHS.
* The proposed Maternal Death Surveillance and Response (MDSR) helps to identify and register maternal deaths, and support appropriate actions to be implemented to prevent them.
* Active citizens’ participation and feedback, independent verification and progress reviews will be used to track achievements of the investments made in RMNCAH.
* Strengthening Civil Registration and Vital Statistics (CRVS) will be an essential intervention to inform better planning and enhance accountability to results.

**Devolution in Achieving SDGs Related to RH**

* Kenya has introduced an ambitious devolution initiative, which could help to address the major demand and supply side challenges.
* National RMNCAH serve as a guide for the development and implementation of county RMNCAH implementation plans, which will be an integral part of County Integrated Development Plans and aligned with the County Health Strategic and Investment Plans.
* Devolution has the potential to address inequities and to enhance accountability.
* Increased government spending is necessary    to scale-up interventions but ensuring effective coverage with an equity focus is critical to improve health outcomes.

**Areas to improve on**

* Effective partnerships are critical for success as fragmented financing and governance cause high transaction costs, hindering effective harmonization at the country level.
* Integration can optimize the efficient use of resources and reduce duplication and wastage.
* Incentives are effective in changing and influencing behavior of providers and users to improve health outcomes.
* Political commitment has been key to improved RMNCAH outcomes in all countries that have made progress on MDGs 4 and 5.

**Innovation and Research**

The following research questions have been identified by MOH:

What are the factors that contribute to poor health seeking behaviors for RMNCAH services?

What are the most cost-effective models that can promote male involvement?

What are the factors contributing to non-adherence to standard operating procedures and guidelines by health care providers in both private and public sectors?

what is the impact of comprehensive RMNCAH training on the competency and skills of providers?

Does task sharing work; if not, what are key barriers?

What is the impact of current RMNCAH behavior change communication interventions?

How effective are the different service delivery models, social media and mobile technologies in delivering adolescent sexual and reproductive health?

What is the impact of maternal shelters?

What are the mechanisms to get real time feedback from adolescents on health services (satisfaction with services)?

How effective is the ongoing innovations such as cash-plus program and what are the implementation challenges?

What models work best to reach out of school adolescents?

## Unit Two Content..

### Topic 1: The Reproductive System

Female and Male Parts

•External

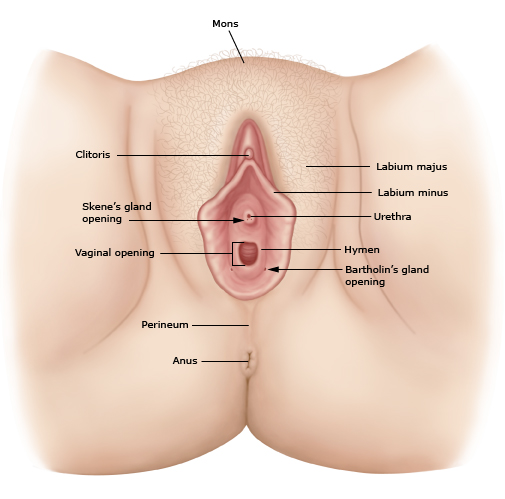
•Internal

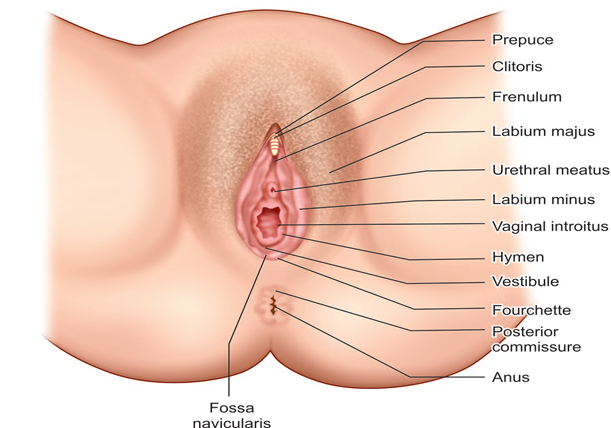
•Accessory reproductive organs -breasts

Male:

•External

•Internal

Female Reproductive system  
  
External Genitalia  




**EXTERNAL GENITALIA (SYN: VULVA, PUDENDUM)**

* The vulva or pudendum includes all the visible external genital organs in the perineum
* The vulva includes:

Ø  mons veneris,

Ø  labia majora,

Ø  labia minora,

Ø  clitoris,

Ø  vestibule and

Ø  conventionally the perineum.

* These are all visible on external examination.
* It is, therefore, bounded anteriorly by the mons veneris, and posteriorly by the rectum, laterally by the genitocrural fold.
* The vulvar area is covered by keratinized stratified squamous epithelium.

**MONS VENERIS (MONS PUBIS)**

* It is the pad of subcutaneous adipose connective tissue lying in front of the pubis and in the adult female is covered by hair.
* The hair pattern (escutcheon) of most women is triangular with the base directed upwards.

**LABIA MAJORA**

* The vulva is bounded on each side by the elevation of skin and subcutaneous tissue which form the labia majora.
* They are continuous where they join medially to form the **posterior commissure in front of the anus.**
* The skin on the outer convex surface is pigmented and covered with hair follicle.
* The thin skin on the inner surface has sebaceous glands but no hair follicle.
* The labia majora are covered with squamous epithelium and contain sweat glands.
* The adipose tissue is richly supplied with venous plexus which may produce hematoma, if injured during childbirth.
* The labia majora are homologous to the scrotum in the male. The round ligament terminates at its upper border.

**LABIA MINORA**

* They are two thin folds of skin, devoid of fat, on either side just within the labia majora.
* Except in the parous women, they are exposed only when the labia majora are separated.
* Anteriorly, they divide to enclose the clitoris and unite with each other in front and behind the clitoris to form the prepuce and frenulum respectively.
* The lower portion of the labia minora fuses across the midline to form a fold of skin known as **fourchette.**
* **It is usually lacerated during childbirth.**
* **Between the fourchette**and the vaginal orifice is the **fossa navicularis.**
* **The labia minora contain no hair follicles or sweat glands.**
* The folds contain connective tissues, numerous sebaceous glands, erectile muscle fibers and numerous vessels and nerve endings.
* The labia minora **are homologous to the penile urethra**and part of the skin of penis in males**.**

**CLITORIS**

* ·         It is a small cylindrical erectile body, measuring about 1.5–2 cm situated in the most anterior part of the vulva.
* ·         **It consists of a glans, a body and two crura.**
* ·         **The clitoris consists of two cylindrical**corpora cavernosa (erectile tissue).
* ·         is richly supplied with nerves.
* ·         Clitoris is **homologous to the penis**in the male**but it differs in being entirely separate**from the urethra.
* ·         It is attached to the under surface of the symphysis pubis by the suspensory ligament.

**VESTIBULE**

* ·         The vestibule is the cleft between the labia minora.
* ·         It is a triangular space bounded anteriorly by the clitoris, posteriorly by the fourchette and on either side by labia minora.
* ·         There are four openings into the vestibule.

o   *Urethral opening*

o   *Vaginal orifice and hymen*

o   *Opening of Bartholin’s ducts*

o   Skene’s glands

***a. Urethral opening***

* ·         The opening is situated in the midline just in front of the vaginal orifice about 1–1.5 cm below the pubic arch.
* ·         The paraurethral ducts open either on the posterior wall of the urethral orifice or directly into the vestibule.

***b. Vaginal orifice and hymen***

* ü  The vaginal orifice lies in the posterior end of the vestibule and is of varying size and shape.
* ü  In virgins and nulliparae, the opening is closed by the labia minora, but in parous, it may be exposed.
* ü  It is incompletely closed by a septum of mucous membrane, called hymen.
* ü  The membrane varies in shape but is usually circular or crescentic in virgins.
* ü  The hymen is usually ruptured at the consummation of marriage.
* ü  During childbirth, the hymen is extremely lacerated and is later represented by cicatrized nodules of varying size, called the carunculae myrtiformes.
* ü  On both sides it is lined by stratified squamous epithelium.

***c. Opening of Bartholin’s ducts:***

* ·         There are two Bartholin glands (greater vestibular gland), one on each side.
* ·         The vestibular bulbs are two oblong masses of erectile tissue that lie on either side of the vaginal entrance.
* ·         They contain a rich plexus of veins within the bulbospongiosus muscle.
* ·         They are **homologous to the bulb of the penis and corpus spongiosum in the male.**
* ·         Bartholin's glands, each about the size of a small pea, lie at the base of each bulb and open via a 2 cm duct into the vestibule between the hymen and the labia minora – outside the hymen at the junction of the anterior two-third and posterior one-third in the groove between the hymen and the labium minus.
* ·         They are pea-sized and yellowish white in color.
* ·         During sexual excitement, it secretes abundant alkaline mucus which helps in lubrication
* ·         Bartholin’s glands are **homologous to the bulb of the penis** in male.

**d. Skene’s glands**

* ·         are the largest **paraurethral glands**.
* ·         Skene’s glands are **homologous to the prostate**in the male**.**
* ·         The two Skene’s ducts may open in the vestibule on either side of the external urethral meatus.

**hymen**

* ·         is a thin fold of mucous membrane across the entrance to the vagina.
* ·         There are usually openings in it to allow menses to escape.
* ·         The hymen is partially ruptured during first coitus and is further disrupted during childbirth.
* ·         Any tags remaining after rupture are known as carunculae myrtiformes.

**blood supply to VULVA**

***Arteries—***

* ***Branches of internal pudendal artery—the chief being labial, transverse***perineal, artery to the vestibular bulb and deep and dorsal arteries to the clitoris.
* Branches of femoral artery—superficial and deep external pudendal.

***Veins—***

* ***The veins form plexuses and drain into:***
* ***Internal pudendal vein,***
* ***vesical or vaginal***venous plexus and
* ·         **Long saphenous vein**. Varicosities during pregnancy are not uncommon and may rupture spontaneously causing visible bleeding or hematoma formation.

**NERVE SUPPLY**

* ·         The supply is through bilateral spinal somatic nerves—
  + - * o   anterosuperior part is supplied by the cutaneous branches from the ilioinguinal and genital branch of genitofemoral nerve (L1 and L2) and the
      * o   posteroinferior part by the pudendal branches from the posterior cutaneous nerve of thigh (S1.2.3).
      * o   Between these two groups, the vulva is supplied by the labial and perineal branches of the pudendal nerve (S2.3.4).

**LYMPHATICS**

* ·         Vulval lymphatics have bilateral drainage. Lymphatics drain into—
  + - * Ø    Superficial inguinal nodes,
      * **Ø**intermediate groups of inguinal lymph nodes—**gland of Cloquet and**
      * Ø    **External**and internal iliac lymph nodes.

**Embryology of VULVA**

* ·         External genitalia is developed in the region of the cranial aspect of ectodermal cloacal fossa;
* ·         clitoris from the genital tubercle;
* ·         labia minora from the genital folds;
* ·         labia majora from the labioscrotal swelling and
* ·         the vestibule from the urogenital sinus.

***Age changes***

* ·         In infancy the vulva is devoid of hair and there is considerable adipose tissue in the labia majora and pubis that is lost during childhood but reappears during puberty, at which time hair grows.
* ·         After menopause the skin atrophies and becomes thinner.
* ·         The labia minora shrink, subcutaneous fat is lost and the vaginal orifice becomes smaller.

### INTERNAL GENITAL ORGANS

* ·         The internal genital organs in female include:
  + - * Ø     vagina,
      * Ø    uterus,
      * Ø    fallopian tubes and
      * Ø    the ovaries.
* ·         These organs are placed internally and require special instruments for inspection.

**VAGINA**

* The vagina is a fibromuscular canal lined with stratified squamous epithelium that leads from the uterus to the vulva.
* It constitutes the excretory channel for the uterine secretion and menstrual blood.
* It is the organ of copulation and forms the birth canal of parturition.
* The diameter of the canal is about 2.5 cm, being **widest in the upper part and narrowest at its introitus.**
* **It has got enough**power of distensibility.
* The canal is directed upwards and backwards forming an angle of 45° with the horizontal in erect posture.
* The long axis of the vagina almost lies parallel to the plane of the pelvic inlet and at right angles to that of the uterus.

**VAGINAL WALL**

* ·         Vagina has got an anterior, a posterior and two lateral walls.
* ·         The anterior and posterior walls are opposed together but the lateral walls are comparatively stiffer especially at its middle, as such, it looks “H” shaped on transverse section.
* ·         The length of the anterior wall is about 7 cm and that of the posterior wall is about 9 cm.
* ·         The vaginal walls are normally in apposition, except at the vault, where they are separated by the cervix.
* ·         The vault of the vagina is divided into four fornices: posterior, anterior and two lateral

**FORNICES**

* ·         The fornices are the clefts formed at the top of vagina (vault) due to the projection of the uterine cervix through the anterior vaginal wall where it is blended inseparably with its wall.
* ·         There are four fornices—

o   one anterior,

o   one posterior and

o   two lateral;

* ·         The posterior one being deeper and the anterior, most shallow one.

**VAGINA**

* ·         The vaginal walls are rugose, with transverse folds.
* ·         The vagina is kept moist by secretions from the uterine and cervical glands and by some transudation from its epithelial lining.
* ·         It has no glands.
* ·         The epithelium is thick and rich in glycogen, which increases in the postovulatory phase of the cycle.
* ·         However, before puberty and after the menopause, the vagina is devoid of glycogen because of oestrogen deficiency.
* ·         Doderlein's bacillus is a normal commensal of the vagina that breaks down the glycogen to form lactic acid, producing a pH of around 4.5.
* ·         This has a protective role for the vagina in decreasing the growth of pathogenic organisms.

**VAGINAL RELATIONS**

***Anterior—***

* *The upper one-third is related with base of the bladder and the lower two-thirds are with*the urethra, the lower half of which is firmly embedded with its wall.

***Posterior—***

* *The upper one-third is related with the pouch of Douglas,*
* *the middle-third with the*anterior rectal wall separated by rectovaginal septum and
* the lower-third is separated from the anal canal by the perineal body

***Lateral walls—***

* *The upper one-third is related with the pelvic cellular tissue at the base of broad*ligament in which the ureter and the uterine artery lie approximately 2 cm from the lateral fornices.
* The middle third is blended with the levator ani and
* the lower-third is related with the bulbocavernosus muscles, vestibular bulbs and Bartholin’s glands

**STRUCTURE OF VAGINA:**

* ·         Layers from within outwards are—
* ·                        mucous coat which is lined by stratified squamous epithelium without any secreting glands
* ·                        submucous layer of loose areolar vascular tissues
* ·                        muscular layer consisting of indistinct inner circular and outer longitudinal muscles and
* ·                        Fibrous coat derived from the endopelvic fascia and is highly vascular.

**VAGINAL SECRETION**

* ·         The vaginal pH, from puberty to menopause, is acidic because of the presence of Döderlein’s bacilli which produce lactic acid from the glycogen present in the exfoliated cells.
* ·         **The pH varies with the estrogenic activity and ranges between 4 and 5.**

**BLOOD SUPPLY TO VAGINA**

* ·         **The arteries involved are—**
  + - * + **Cervicovaginal branch of the uterine artery,**
        + **Vaginal**artery—a branch of anterior division of internal iliac or in common origin with the uterine,
        + Middle rectal and
        + internal pudendal. These anastomose with one another and form two azygos arteries— anterior and posterior.
* ·         ***Veins****drain into internal iliac veins and internal pudendal veins.*

**LYMPHATICS TO VAGINA:**

* ·         On each side, the lymphatics drain into—
  + - * o   Upper one-third—internal iliac group,
      * o   middle one-third up to hymen—internal iliac group,
      * o   below the hymen—superficial inguinal group.

**NERVE SUPPLY TO VAGINA**:

* ·         The vagina is supplied by sympathetic and parasympathetic from the pelvic plexus.
* ·         The lower part is supplied by the pudendal nerve.

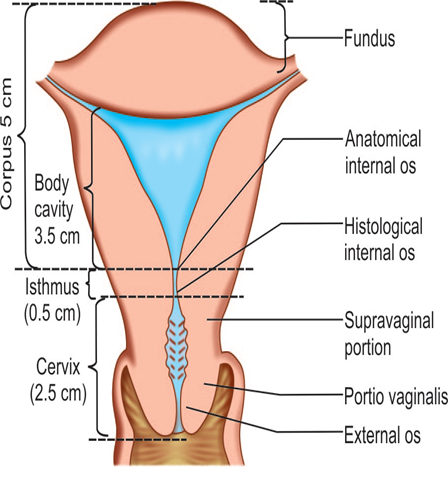
**DEVELOPMENT**

* ·         **The vagina is developed from the following sources:**
* ·                        **Upper 4/5th, above the**hymen—the mucous membrane is derived from endoderm of the canalized sinovaginal bulbs. The musculature is developed from the mesoderm of two fused Müllerian ducts.
* ·                        Lower 1/5th, below the hymen is developed from the endoderm of the urogenital sinus.
* ·                        External vaginal orifice is formed from the genital fold ectoderm after rupture of the urogenital membrane.

***Age changes***

* ·         At birth, the vagina is under the influence of maternal oestrogens, so the epithelium is well developed.
* ·         After a couple of weeks, the effects of the oestrogens disappear and the pH rises to 7 and the epithelium atrophies.
* ·         At puberty the reverse occurs, and finally, at the menopause, the vagina tends to shrink and the epithelium atrophies.

### THE UTERUS



**THE UTERUS**

* The uterus is a hollow pyriform muscular organ situated in the pelvis between the bladder in front and the rectum behind.
* The uterus is shaped like an inverted pear, tapering inferiorly to the cervix, and in the non-pregnant state is situated entirely within the pelvis.
* It is hollow and has thick muscular walls. Its maximum external dimensions are approximately 7.5cm long, 5cm wide and 3cm thick
* An adult uterus weighs about 70 g.

**POSITION OF UTERUS**

* ·         The longitudinal axis of the uterus is, approximately, at right-angles to the vagina and normally tilts forwards.
* ·         This is termed anteversion.
* ·         The uterus is usually also flexed forwards on itself at the isthmus - anteflexion.
* ·         Its normal position is one of the anteversion and anteflexion.
* ·         In around 20 per cent of women, this tilt is not forwards but backwards - retroversion and retroflexion.
* ·         This does not have a pathological significance.
* ·         The uterus usually inclines to the right (dextrorotation) so that the cervix is directed to the left (levorotation) and comes in close relation with the left ureter.

**MEASUREMENTS AND PARTS:**

* ·         The uterus measures about 8 cm long, 5 cm wide at the fundus and its walls are about 1.25 cm thick.
* ·         Its weight varies from 50 gm to 80 gm.
* ·         It has got the following parts:

o   **Body or corpus**

o   **Isthmus**

o   **Cervix**

**Body or corpus of the uterus**

* ·         The body is further divided into:
  + - * + o   fundus—the part which lies above the openings of the uterine tubes.
        + o   The body proper is triangular and lies between the openings of the tubes and the isthmus.
* ·         The superolateral angles of the body of the uterus project outwards from the junction of the fundus and body and is called the **cornua of the uterus.**
* ·         The uterine tube, round ligament and ligament of the ovary are attached to it.
* ·         The area of insertion of each Fallopian tube is termed the cornu and the part of the body above the cornu, the fundus

**Isthmus of the uterus**

* ·         is a constricted part measuring about 0.5 cm, situated between the body and the cervix.
* ·         **It is limited above by the anatomical internal os and below by the histological internal os (Aschoff).**
* ·         **Some**consider isthmus as a part of the lower portion of the body of the uterus

cervix

* ·         **Cervix**is cylindrical in shape and measures about 2.5cm.
* ·         It extends from the isthmus and ends at the external os which opens into the vagina after perforating its anterior wall.
* ·         The part lying above the vagina is called supravaginal and that which lies within the vagina is called the vaginal part

CAVITY:

* ·         The cavity of the uterine body is triangular on coronal section with the base above and the apex below.
* ·         It measures about 3.5 cm.
* ·         There is no cavity in the fundus.
* ·         The cervical canal is fusiform and measures about 2.5 cm.
* ·         **Thus, the normal length of the uterine cavity is usually 6.5–7 cm.**

**RELATIONS OF THE UTERUS**

***Anteriorly—***

* ·         *Above the internal os, the body forms the posterior wall of the uterovesical*pouch.
* ·         Below the internal os, it is separated from the base of the bladder by loose areolar tissue.

***Posteriorly—***

* ·         *It is covered with peritoneum*and forms the anterior wall of the pouch of Douglas containing coils of intestine.

***Laterally—***

* ·         ***The double fold of peritoneum***of the broad ligament are attached between which the uterine artery ascends up.

**STRUCTURES - BODY**

The wall consists of three layers from outside inwards:

* ü  ***Parametrium****: It is the serous coat which invests the entire organ except on the lateral borders.*The peritoneum is intimately adherent to the underlying muscles.
* ü  ***Myometrium****: It consists of thick bundles of smooth muscle fibers held by connective tissues*and are arranged in various directions. During pregnancy, however, three distinct layers can be identified—outer longitudinal, middle interlacing and the inner circular.
* ü  ***Endometrium:****The mucous lining of the cavity. As there is no submucous*layer, the endometrium is directly opposed to the muscle coat. It consists of:
  + - * + o   lamina propria and
        + o   surface epithelium.
* The surface epithelium is a single layer of ciliated columnar epithelium.
* The lamina propria contains stromal cells, endometrial glands, vessels and nerves. The glands penetrate the stroma and sometimes even enter the muscle coat.
* The endometrium is changed to **decidua during pregnancy**.

**STRUCTURE-CERVIX**

* ·         The cervix is composed mainly of fibrous connective tissues.
* ·         The smooth muscle fibers average 10–15%.
* ·         Only the posterior surface has got peritoneal coat.
* ·         Mucous coat lining the endocervix is simple non-ciliated columnar.
* ·         The vaginal part of the cervix is lined by stratified squamous epithelium.
* ·         **The squamocolumnar junction is situated at the external os.**

**Uterine secretion**

* ·         The endometrial secretion is scanty and watery.
* ·         Secretion of the cervical glands is alkaline and thick, rich in mucoprotein, fructose and sodium chloride.

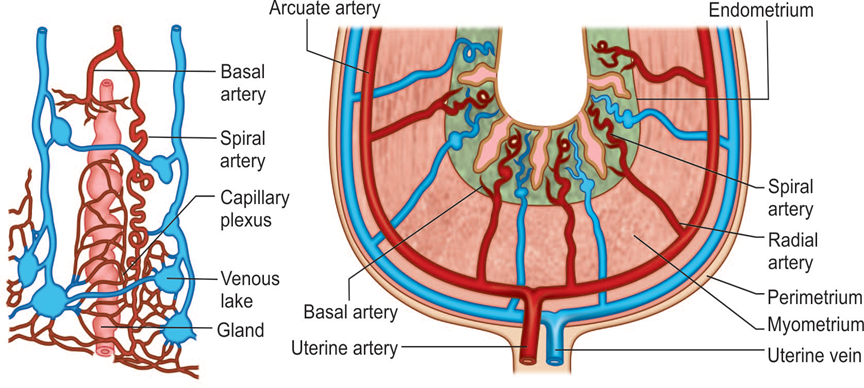
 BLOOD SUPPLY

·         ***Arterial supply —***

* o   ***uterine arteries one on each side (***anterior division of the internal iliac or in common with superior vesical artery.)
* o   ovarian and vaginal arteries with which the uterine arteries anastomose.
* ·         **The cervix is insensitive to touch, heat and also when it is grasped by any instrument. The uterus, too, is insensitive to handling and even to incision over its wall.**

*A) Showing pattern of basal and spiral arteries in the endometrium;*

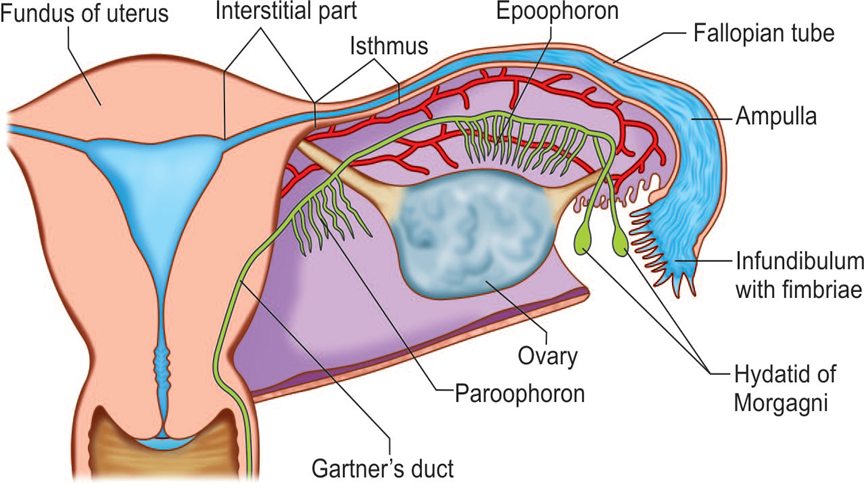
*(B) Internal blood supply of uterus*



***Age changes***

* ·         The disappearance of maternal oestrogens after birth causes the uterus to decrease in length by around one-third and in weight by about one-half.
* ·         The cervix is then twice the length of the uterus.
* ·         At puberty, however, the corpus grows much faster and the size ratio reverses.
* ·         After the menopause, the uterus atrophies, the mucosa becomes very thin, the glands almost disappear and the wall becomes relatively less muscular.
* ·         These changes affect the cervix more than the corpus; cervical loops disappear and the external os becomes more or less flush with the vault.

FALLOPIAN TUBE (***Synonyms: Uterine tube, oviduct)***



* ·         The uterine tubes are paired structures, measuring about 10 cm and are situated in the medial three-fourth of the upper free margin of the broad ligament.
* ·         Each tube has got two openings:
  + - o   one communicating with the lateral angle of the uterine cavity called uterine opening and measures 1 mm in diameter,
    - o   the other is on the lateral end of the tube, called pelvic opening or abdominal ostium and measures about 2 mm in diameter.

**Parts of oviduct**

·         From medial to lateral are—

* + intramural or interstitial lying in the uterine wall and measures 1.25 cm in length and 1 mm in diameter,
  + isthmus—almost straight and measures about 3–4 cm in length and 2 mm in diameter,
  + ampulla—tortuous part and measures about 5 cm in length which ends in,
  + wide infundibulum measuring about 1.25 cm long with a maximum diameter of 6 mm.
    - o   The abdominal ostium is surrounded by a number of radiating fimbriae (20–25), one of these is longer than the rest and is attached to the outer pole of the ovary called **ovarian fimbria**

**The important functions of the tubes**

·         Transport of the gametes,

·         To facilitate fertilization and survival of zygote through its secretion.

**Blood Supply**

·         **Arterial supply**is from the uterine and ovarian.

·         Venous drainage is through the pampiniform plexus into the ovarian veins.

**THE OVARY**

* ·         The ovaries are paired sex glands or gonads in female which **are concerned for:**
  + - * •                  **germ cell maturation,**storage and its release and
      * •                  steroidogenesis.
* •Each gland is oval in shape and pinkish gray in color and the surface is scarred during reproductive period.
* •It measures about 3 cm in length, 2 cm in breadth and 1 cm in thickness.
* •Each ovary presents:
* •two ends—tubal and uterine,
* •two borders—mesovarium and free posterior and
* •two surfaces—medial and lateral.
* ·         The ovary is the only intra-abdominal structure not to be covered *by peritoneum*
* ·         it lies in the ovarian fossa on the lateral pelvic wall.
* ·         it is attached to the posterior layer of the broad ligament by the mesovarium, to the lateral pelvic wall by the infundibulopelvic ligament and to the uterus by the ovarian ligament.

 RELATIONS

·         ***Mesovarium or anterior border***

* + o   *A fold of peritoneum from the posterior leaf of the*broad ligament is attached to the anterior border through which the ovarian vessels and nerves enter the hilum of the gland.

·         ***Posterior border***

o   *is free and is related to the tubal ampulla. It is separated by the peritoneum from*the ureter and the internal iliac artery.

·         ***Medial surface***

* + o   *is related to fimbrial part of the tube****.***

·         ***Lateral surface***

* + o   *is in contact with the ovarian fossa on the lateral pelvic wall.*

**STRUCTURES:**

•The ovary is covered by a single layer of cubical cell known as germinal epithelium.

•The substance of the gland consists of:

* + a.    outer cortex and
  + b.    inner medulla.

***Cortex of the ovary***

* ·         It consists of stromal cells which are thickened beneath the germinal epithelium to form **tunica albuginea.**
* ·         During reproductive period the cortex is studded with numerous follicular structures, called the **functional units**of the ovary, in various phases of their development which include:
  + primordial follicles,
  + maturing follicles,
  + Graafian follicles and
  + corpus luteum.

·         Atresia of the structures results in formation of atretic follicles or corpus albicans.

·         These are related to sex hormone production and ovulation

***Medulla of the ovary***

·         It consists of loose connective tissues, few unstriped muscles, blood vessels and nerves.

·         There is a small collection of cells called “**hilus cells” which are homologous to the interstitial cells of the testes.**

**Blood supply**

·         *Arterial supply – ovarian artery, a branch of the abdominal aorta.*

·         *Venous drainage – pampiniform plexus.*

·         NERVE SUPPLY: Sympathetic supply comes down along the ovarian artery from T10 segment. **Ovaries are sensitive to manual squeezing.**

**DEVELOPMENT:**

·         The ovary is developed from the cortex of the undifferentiated genital ridges by about 9th week; the primary germ cells reaching the site migrating from the dorsal end of yolk sac.

### PELVIC FLOOR (Synonym: Pelvic diaphragm)

·         **Pelvic floor is a muscular partition which separates the pelvic cavity from the anatomical perineum.**

·         It consists of three sets of muscles on either side—

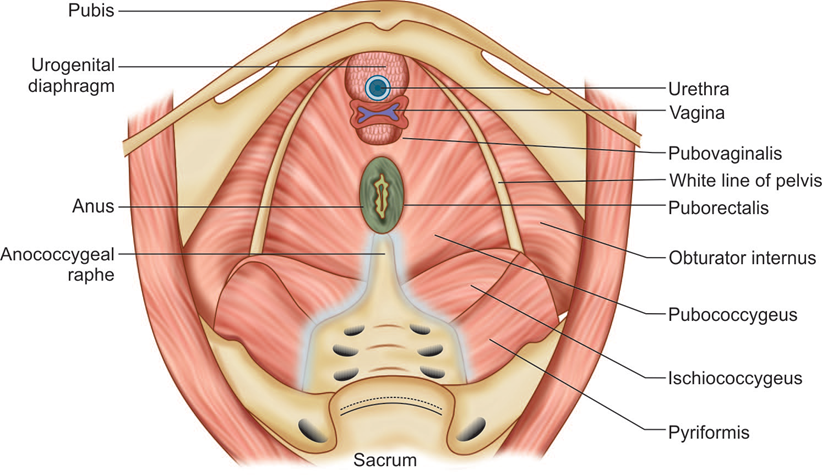
o   pubococcygeus,

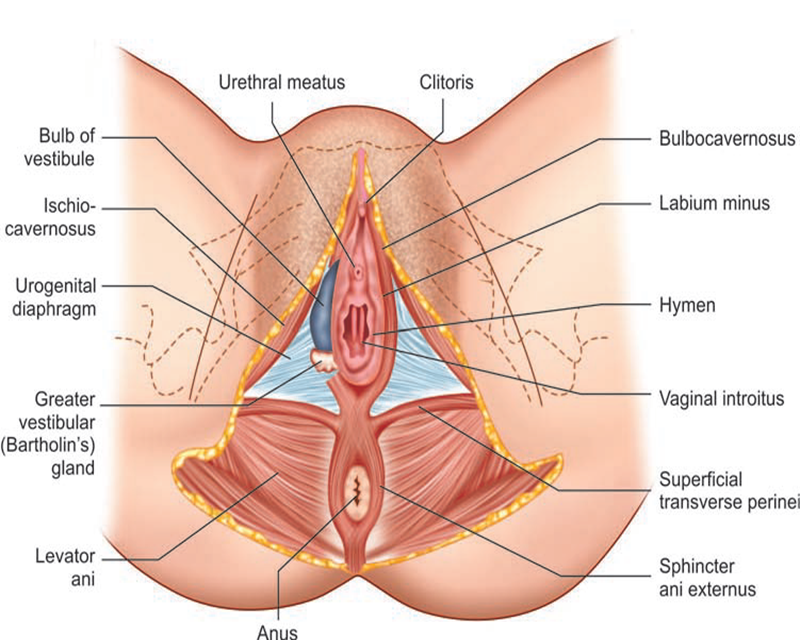
o   iliococcygeus and

o   Ischiococcygeus

* ·         these are collectively called levator ani.
* ·         Its upper surface is concave and slopes downwards, backwards and medially and is covered by parietal layer of pelvic fascia.
* ·         The inferior surface is convex and is covered by anal fascia.
* ·         **The muscle with the covering fascia is called the pelvic diaphragm.**

Levator ani muscles viewed from above





**Levator ani muscle**

·         ORIGIN:

o   Each levator ani arises from the back of the pubic rami, from the condensed fascia covering the obturator internus (white line) and from the inner surface of the ischial spine.

·         INSERTION:

o   From this extensive origin, **the fibers pass, backwards and medially to be inserted**in the midline from before backwards to the vagina (lateral and posterior walls), perineal body and anococcygeal raphe, lateral borders of the coccyx and lower part of the sacrum

**GAPS ON PELVIC FLOOR**

·         There are **two gaps in the midline—**

* + - The anterior one is called hiatus urogenitalis which is bridged by the muscles and fascia of urogenital triangle and pierced by the urethra and vagina.
    - The posterior one is called hiatus rectalis, transmitting the rectum.

**STRUCTURES IN RELATION TO PELVIC FLOOR**

The superior surface is related with the following:

* Pelvic organs from anterior to posterior are bladder, vagina, uterus and rectum.
* Pelvic cellular tissues between the pelvic peritoneum and upper surface of the levator ani which fill all the available spaces.
* Ureter lies on the floor in relation to the lateral vaginal fornix. The uterine artery lies above and the vaginal artery lies below it.
* Pelvic nerves.
* *The inferior surface is related to the anatomical perineum.*

**FUNCTIONS OF THE PELVIC FLOOR**

* To support the pelvic organs—The pubovaginalis which forms a “U” shaped sling, supports the vagina which in turn supports the other pelvic organs— bladder and uterus. Weakness or tear of this sling during parturition is responsible for prolapse of the organs concerned.
* To maintain intra-abdominal pressure by reflexly responding to its changes.
* Facilitates anterior internal rotation of the presenting part when it presses on the pelvic floor.
* Puborectalis plays an ancillary role to the action of the external anal sphincter.
* Ischiococcygeus helps to stabilize the sacroiliac and sacrococcygeal joints.
* To steady the perineal body.

**PELVIC FLOOR DURING PREGNANCY AND PARTURITION**

·         **During pregnancy**

* **levator muscles**undergo hypertrophy, become less rigid and more distensible. Due to water retention, it swells up and sags down.

·         **In the second stage,**

* **the pubovaginalis and puborectalis**relax and the levator ani **is**drawn up over the advancing presenting part in the second stage.
* Failure of the levator ani to relax at the crucial moment may lead to extensive damage of the pelvic structures

### PERINEUM

**ANATOMICAL PERINEUM**

·         It is bounded above by:

o   inferior surface of the pelvic floor,

o   below by the skin between the buttocks and thighs.

o   Laterally by the ischiopubic ramus, ischial tuberosities and sacrotuberous ligaments and  
o   posteriorly, by the coccyx.

·         The diamond shaped space of the bony pelvic outlet is divided into two triangular spaces with the common base formed by the free border of the urogenital diaphragm.

o   The anterior triangle is called the urogenital triangle which fills up the gap of the hiatus urogenitalis and is important from the obstetric point of view.

* + o   The posterior one is called the anal triangle

**Urogenital Triangle**

* ·         It is pierced by the terminal part of the vagina and the urethra.
* ·         Has superficial and deep perineal pouch compartments.
* ·         **The superficial pouch is formed**by the deep layer of the superficial perineal fascia (Colles fascia) and inferior layer of the urogenital diaphragm (perineal membrane
* ·         The **deep perineal pouch is formed by the inferior and**superior layer of the urogenital diaphragm—together called urogenital diaphragm or triangular ligament

**Anal Triangle**

* ·         The triangle has got no obstetric importance.
* ·         It contains the terminal part of the anal canal with sphincter ani externus, anococcygeal body, ischiorectal fossa, blood vessels, nerves, and lymphatics.

**OBSTETRICAL PERINEUM:*(Synonyms: Perineal body, central point of the perineum)***

* ·         The pyramidal shaped tissue where the pelvic floor and the perineal muscles and fascia meet in between the vagina and the anal canal is called the obstetrical perineum.
* ·         **It measures about 4 cm × 4 cm with the**base covered by the perineal skin and the apex is pointed and is continuous with the rectovaginal septum.

***Importance of obstetric perineum***

•      *It helps to support the levator ani which is placed above it.*

•      *By supporting the*posterior vaginal wall, it indirectly supports the anterior vaginal wall, bladder and the uterus.

•      It is vulnerable to injury during childbirth.

•      Deliberate cutting of the structures during delivery is called episiotomy.

**PELVIC CELLULAR TISSUE**

•      It lies between the pelvic peritoneum and the pelvic floor and fills up all the available empty spaces.

•      It contains fatty and connective tissues and unstriated muscle fibers.

•      Its distribution around the vaginal vault, supravaginal part of the cervix and into the layers of the broad ligament is called **parametrium.**

***Importance of the pelvic cellular tissue***

•      To support the pelvic organs.

•      To form protective sheath for the blood vessels and the terminal part of the ureter.

•      infection spreads along the track, so formed, outside the pelvis to the perinephric region along the ureter, to the buttock along the gluteal vessels, to the thigh along the external iliac vessels and to the groin along the round ligament.

•      Marked hypertrophy occurs during pregnancy to widen up the spaces.

**POUCH OF DOUGLAS**

•      This is a narrow peritoneal **cul-de-sac** in the pelvis situated in the rectouterine space.

•      It is continuous with the pararectal fossa of either side.

**Contents of Pouch of Douglas:**

o   **It may remain empty but may contain coils**of intestine or omentum.

**Relations of POUCH OF DOUGLAS**

•      **Anteriorly,**

o   **it is bounded by the peritoneal covering**of the cervix, posterior vaginal fornix and upper-third of the posterior vaginal wall.

•      **Posteriorly,**

o   **it is bounded by the peritoneal covering**on the anterior surface of the rectum.

•      **Laterally,**

o   **it is limited by the uterosacral folds of**peritoneum covering the uterosacral ligaments.

•      **The floor**

o   **is formed by the reflection of the anterior**peritoneum onto the anterior surface of the rectum. It is about 6–7 cm above the anal orifice. Below the floor, there is a thin fibrous tissue septum (rectovaginal).

**Surgical Importance of Pouch of Douglas**

•      As it is the most dependent part of the peritoneal cavity, intraperitoneal blood or pus usually settles down to the pouch to produce either pelvic hematocele or pelvic abscess.

•      Herniation of the pouch through the posterior fornix may occur producing the clinical entity of enterocele.

•      Vaginal ligation is done through opening the pouch.

•      Culdoscopy, culdocentesis or at time pneumoperitoneum may be done through the pouch.

•      Nodules deposited in the pouch can help in the clinical diagnosis of pelvic malignancy, endometriosis or genital tuberculosis.

### Uterine Support

**BROAD LIGAMENT**

•      The double fold of peritoneum which extends from the lateral border of the uterus to the lateral pelvic wall of pelvis is called broad ligament.

•      These, truly are not ligaments

**BROAD LIGAMENT**

•      consists of two layers, anterior and posterior.

•      The layers are continuous at its upper free border embracing the Fallopian tube.

•      The lower part of the broad ligament is wider from before backwards and the layers are reflected above the pelvic diaphragm.

**PARTS OF BROAD LIGAMENT**

**1. Infundibulopelvic Ligament (Syn: Suspensory ligament of the ovary):**

•      It includes the portion of the broad ligament which extends from the infundibulum to the lateral pelvic wall.

•      It contains ovarian vessels and nerves and lymphatics from the ovary, Fallopian tube and body of the uterus.

2. **Mesovarium:**

•      **The ovary is attached to the posterior**layer of the broad ligament by a fold of peritoneum called mesovarium (ovarian mesentery).

•      Through this fold, ovarian vessels, nerves and lymphatics enter and leave the hilum.

•      The ovary is not enclosed within the broad ligament

**3. Mesosalpinx:**

•      The part of the broad ligament between the fallopian tube and the level of attachment of the ovary is the mesosalpinx.

•      It contains utero-ovarian anastomotic vessels and vestigial remnants.

**4. Mesometrium:**

* + The part of the broad ligament below the mesosalpinx is called mesometrium.
  + It is the longest portion which is related with the lateral border of the uterus.

**contents of the broad ligament**

Fallopian tube.

Uterine and ovarian arteries with their branches, including the anastomotic branches between them and corresponding veins.

Nerves and lymphatics from the uterus, Fallopian tube and ovary.

Proximal part of the round ligament which raises a peritoneal fold on the anterior leaf.

Ovarian ligament which raises a peritoneal fold on the posterior leaf.

Parametrium containing loose areolar tissue and fat. The terminal part of the ureter, uterine artery, paracervical nerve and lymphatic plexus are lying at the base of the broad ligament.

Vestigial structures, such as duct of Gartner, epoophoron, and paroophoron.

**MACKENRODT’S LIGAMENTS (SYN: CARDINAL LIGAMENT, TRANSVERSE CERVICAL)**

•      **Origin:**

o   **Condensation of parietal fascia covering the**obturator internus.

•      **Insertion:**

o   **Lateral supravaginal cervix and upper**part of lateral vaginal wall in a fan-shaped manner. This insertion is continuous with the endopelvic and paricervical fascial ring

**Function:**

•      **Lateral stabilization to the cervix at the**level of ischial spine.

o   Primary vascular conduits of the uterus and vagina

**UTEROSACRAL LIGAMENTS**

•      **Origin:**

o   Periosteum of sacral vertebra 2, 3 and 4.

•      **Insertion:**

o   Posterolateral surface of the cervix at the level of internal os

•      **Content:**

o   Uterosacral plexus of autonomic nerves. Smooth muscle and minimal vessels.

•      **Function:**

o   These are the primary proximal suspensory ligaments of the uterovaginal complex.

o   They hold the cervix posteriorly at the level of the ischial spines.

o   Uterus is thus maintained anteflexed and the vagina is suspended over the levator plate.

**ROUND LIGAMENTS**

* Each measures about 10–12 cm.
* It is attached at the cornu of the uterus below and in front of the fallopian tube.
* It courses beneath the anterior leaf of the broad ligament to reach the internal abdominal ring.
* After traversing through the inguinal canal, it fuses with the subcutaneous tissue of the anterior third of the labium majus.
* It contains plain muscles and connective tissue.

**OVARIAN LIGAMENTS**

* These are paired, one on each side.
* Each one is a fibromuscular cord-like structure which attaches to the inner pole of the ovary and to the cornu of the uterus posteriorly below the level of the attachment of the fallopian tube
* Morphologically, it is continuous with the round ligament.

### Urinary System and breast

**FEMALE URETHRA**

•      The female urethra extends from the neck of the bladder to the external urethral meatus which opens into the vestibule about 2.5 cm below the clitoris.

•      **It measures about 4 cm and has a diameter of 6 mm.**

•      Its upper half is separated from the anterior vaginal wall by loose areolar tissue and the lower half is firmly embedded in its wall.

•      Numerous tubular glands called paraurethral glands open into the lumen through ducts eg Skene’s ducts which open either on the posterior wall or into the vestibule.

•      These glands are the sites for harboring infection and occasional development of benign adenoma or malignant changes.

**THE URINARY BLADDER**

•      The bladder is a hollow muscular organ with considerable power of distension.

•      Its capacity is about 450mL but can retain as much as 3–4 liters of urine.

•      When distended it is ovoid in shape. It has got:

* o   an apex
* o   superior surface
* o   Base
* o   two inferolateral surfaces and
* o   neck, which is continuous with the urethra.

•      **The base and the neck remain fixed even when the bladder is distended.**

**THE BREAST**

•      The breasts are large, modified sebaceous glands.

•      The breasts are bilateral and in female constitute accessory reproductive organs as the glands are concerned with lactation following childbirth.

•      The shape of the breast varies in women and also in different periods of life.

•      But the size of the base of the breast is fairly constant.

•      It usually extends from the second to sixth rib in the midclavicular line.

•      It lies in the subcutaneous tissue over the fascia covering the pectoralis major or even beyond that to lie over the serratus anterior and external oblique.

•      A lateral projection of the breast towards the axilla is known as axillary tail of Spence.

•      It lies in the axillary fossa, sometimes deep to the deep fascia.

•      The breast weighs 200–300 gm during the childbearing age.

NB

**G-spot**

•      The **G-spot**, also called the **Gräfenberg spot** (for German gynecologist Ernst Gräfenberg, is characterized as an erogenous area of the vagina that, when stimulated, may lead to strong sexual arousal, powerful orgasms and potential female ejaculation.

•      It is typically reported to be located 5–8 cm up the front (anterior) vaginal wall between the vaginal opening and the urethra and is a sensitive area that may be part of the female prostate.

•      The existence of the G-spot has not been proven, nor has the source of female ejaculation.

•      Although the G-spot has been studied since the 1940s,disagreement persists over its existence as a distinct structure, definition and location.

•      A 2009 British study concluded that its existence is unproven and subjective, based on questionnaires and personal experience.

•      Other studies, using ultrasound, have found physiological evidence of the G-spot in women who report having orgasms during vaginal intercourse

It is also hypothesized that the G-spot is an extension of the clitoris and that this is the cause of orgasms experienced vaginally.

### The Male reproductive system

•      The reproductive system in men has components in the abdomen, pelvis, and perineum

•      The major components are a ***testis***, ***epididymis***, ductus***deferens***, and ejaculatory***duct***on each side, and the ***urethra***and penisin the midline.

•      Three types of accessory glands are associated with the system:

o   A single prostate;

o   A pair of seminal vesicles;

o   A pair of bulbourethral

o   glands.

**The scrotum**

•      The scrotum is an outpouching of the lower part of the anterior abdominal wall.

•      It contains the testes, the epididymis’s, and the lower ends of the spermatic cords.

•      It is divided on its surface into two compartments by a *raphé*, which is continued forward to the under surface of the penis, and

•      backward, along the middle line of the perineum to the anus.

•      Each compartment contains one of the two testes, and one of the epididymides.

•      The wall of the scrotum has the following layers:

o   Skin

o   Superficial fascia

o   Spermatic fasciae

o   Tunica vaginalis

Skin

* •      The skin of the scrotum is thin, wrinkled, and pigmented and forms a single pouch. A slightly raised ridge in the midline indicates the line of fusion of the two lateral labioscrotal swellings.

Superficial fascia

•      This is continuous with the fatty and membranous layers of the anterior abdominal wall.

•      The fat is replaced by smooth muscle called the dartos muscle.

•      This is innervated by sympathetic nerve fibers and is responsible for the wrinkling of the overlying skin.

Spermatic fasciae

•      It has three layers which lie beneath the superficial fascia and are derived from the three layers of the anterior abdominal wall on each side.

•      The *external spermatic fascia*is derived from the aponeurosis of the external oblique muscle; the cremasteric*fascia*is derived from the internal oblique muscle; and, finally, the *internal spermatic fascia*is derived from the fascia transversalis**.**

Tunica vaginalis

•      This lies within the spermatic fasciae and covers the anterior, medial, and lateral surfaces of each testis.

Lymph from the skin and fascia, including the tunica vaginalis, drains into the superficial inguinal lymph nodes

### Testes

* Testis has ellipsoid- shaped.
* Testes develop in the abdomen and move before birth into the scrotum.
* The left testis usually lies at a lower level than the right.
* The testis are covered by:
  + - A closed sac of peritoneum (the **tunica vaginalis)**, which originally connected to the abdominal cavity.  Normally after testicular descent, the connection closes, leaving a fibrous remnant.
    - It is covered by a fibrous capsule called the ***tunica albuginea.***
* In the inner surface of the capsule is a series of fibrous septa that divide the interior of the organ into lobules.
* Lying within each lobule are 1 to 3 coiled ***seminiferous tubules***.
* The tubules open into a network of channels called the rete***testis***.
* Small efferent ductules connect the rete testis to the upper end of the epididymis.

**Epididymis**

•      The **epididymis**is a single, long coiled duct that courses along the posterolateral side of the testis.

•      The tunica vaginalis covers the epididymis with the exception of the posterior border.

•      Structurally, epididymis divided into:

o   Head

o   Body

o   tail

**Epididymis**

It has two distinct components:

•      The efferent ductules, which form an enlarged coiled mass that sits on the posterior superior pole of the testis and forms the head of the epididymis;

•      The true epididymis, which is a single, long coiled duct into which the efferent ductules all drain, and which continues inferiorly along the posterolateral margin of the testis as the body of epididymis and enlarges to form the tail of epididymis at the inferior pole of the testis.

•      The testicular artery is a branch of the abdominal aorta.

**Venous drainage of the Testis and Epididymis**

•      The testicular veins emerge from the testis and the epididymis as a venous network, the pampiniform plexus.

•      This becomes reduced to a single vein as it ascends through the inguinal canal.

•      The right testicular vein drains into the inferior vena cava, and the left vein joins the left renal vein**.**

•      Lymphatic drainage of the testes is to the para- aortic lymph nodes

**Ductus deferens**

•      (Latin: "carrying-away  vessel"), also called *vas  deferens*.

•      The ductus deferens is a  long muscular duct that  transports spermatozoa  from the tail of the  epididymis to the  ejaculatory duct

•      The vas arises from the tail of  the epididymis and traverses  the inguinal canal to the deep  ring, passes downwards on the  lateral wall of the pelvis  almost to the ischial tuberosity  and turns medially to cross the  ureter posterior to the  bladder.

•      It continues inferomedially along the base of the bladder, anterior to the rectum, almost to the midline, where it is joined by the duct of the seminal*vesicle*to form the ejaculatory*duct***.**

•      The terminal part of the vas deferens is dilated to form the *ampulla*of the vas deferens.

•      The *ejaculatory duct penetrates* through the prostate*gland*to connect with the prostatic urethra.

**Seminal vesicle**

•      The seminal vesicles are an accessory gland of the male reproductive system.

•      The seminal vesicles are two lobulated organs about 2 in. (5 cm) long lying on the posterior surface of the bladder

•      On the medial side of each vesicle lies the terminal part of the vas deferens.

•      Posteriorly, the seminal vesicles are related to the rectum.

•      Inferiorly, each seminal vesicle narrows and joins the vas deferens of the same side to form the ejaculatory duct.

•      **Arteries**

o   The arterial blood supply from, the inferior vesicle and middle rectal arteries

•      **Veins**

o   The veins drain into the internal iliac veins.

**Ejaculatory Ducts**

•      The two ejaculatory ducts are each less than 1 in. (2.5 cm) long and are formed by the union of the vas deferens and the duct of the seminal vesicle.

•      The ejaculatory ducts pierce the posterior surface of the prostate and open into the prostatic part of the urethra, close to the margins of the prostatic utricle; their function is to drain the seminal fluid into the prostatic urethra.

**Prostate**

•      The prostate is an unpaired accessory structure of the male reproductive system that surrounds the urethra in the pelvic cavity.

•      It lies immediately inferior to the bladder, above the urogenital diaphragm, posterior to the pubic symphysis, and anterior to the rectum.

•      The prostate is shaped like an inverted rounded cone with a larger base, which is continuous above with the neck of the bladder, and a narrower apex, which rests below on the pelvic floor.

•      The inferolateral surfaces of the prostate are in contact with the levator ani muscles that together cradle the prostate between them.

•      The two ejaculatory ducts pierce the upper part of the posterior surface of the prostate to open into the prostatic urethra at the lateral margins of the prostatic utricle.

**Relations of Prostate**

•      Superiorly

o   The base of the prostate is continuous with the neck of the bladder.

o   The urethra enters the center of the base of the prostate.

•      Inferiorly

o   The apex of the prostate lies on the upper surface of the urogenital diaphragm.

o   The urethra leaves the prostate just above the apex on the anterior surface

•      Anteriorly

o   The prostate is related to the symphysis pubis.

o   The prostate is connected to the posterior aspect of the pubic bones by the puboprostatic ligaments.

•      Laterally

o   The prostate **is**embraced by the anterior fibers of the levator ani.

•      Posteriorly

o   The prostate is closely related to the anterior surface of the rectal ampulla and is    separated from it by the rectovesical septum (*fascia of Denonvilliers).*

**Structure of the Prostate**

•      Enclosed within thin dense fibrous capsule

•      Inner loose sheath derived from pelvic fascia – “prostatic sheath”

–     Continuous inferiorly with superior fascia of urogenital diaphragm

–     Posteriorly it is part of rectovesical septum

–     Separates bladder, seminal vesicles and prostate from rectum

•      Prostatic venous plexus lies between fibrous capsule and prostatic sheath.

**Prostate divided into:**

–     Two lateral lobes

–     One median lobe

–     Anterior and posterior lobes

**Structure of the Prostate**

•      Anterior

–     Tissue lying anterior to urethra

–     No glands; fibromuscular tissue only

•      Median

–     Cone-shaped region between ejaculatory ducts and urethra

•      Lateral (left & right)

–     Main mass of gland, continuous posteriorly

–     Separated by prostatic urethra

•      Posterior

–     Describes postero-medial part of lateral lobes palpable through rectum on DRE.

**Blood Supply of The Prostate**

•      **Arterial supply**

–     Arteries derived from *internal pudenal, inferior vesical and* *middle rectal arteries (branches of internal iliac)*

•      **Venous drainage**

–     Veins form prostatic venous plexus around sides and base of prostate – located between capsule and sheath

–     Drains into *internal iliac veins*

–     Also communicates with vesical venous plexus and  vertebral venous plexuses.

**Lymphatics and innervation of The Prostate**

o   Lymphatic drainage

§  Lymph vessels terminate in internal iliac and sacral lymph nodes

§  Some vessels from posterior surface pass with lymph vessels from bladder to external iliac LN’s

o   Innervation

§  Parasympathetic fibres arise from pelvic splanchnic nerves

§  Sympathetic fibres from inferior hypogastric plexuses

 Penis

•      The penis is a pendulous organ suspended from the front and sides of the pubic arch and containing the greater part of the urethra.

•      It consists of internal root, external shaft, & glans.

•      **Root**: the portion of the penis that extends internally into the pelvic cavity.

•      **Shaft**: the length of the penis between the glans and the body.

•      **Glans**: the head of the penis; has many nerve endings.

•      **Foreskin**: a covering of skin over the penile glans.

•      The **root of penis**consists of the two crura, which are proximal parts of the corpora*cavernosa attached* to the pubic arch, and the **bulb of penis**, which is the proximal part of *the corpus spongiosum anchored* to the perineal membrane.

•      The body of the penis is essentially composed of three cylinders of erectile tissue enclosed in a tubular sheath of fascia (Buck's fascia).

•      The erectile tissue is made up of two dorsally placed corpora cavernosa and a single corpus spongiosum applied to their ventral surface.

•                  At its distal extremity, the corpus spongiosum expands to form the glans penis, which covers the distal ends of the corpora cavernosa.

•      On the tip of the glans penis is the slit like orifice of the urethra, called the external urethral meatus.

**External penile structures**

•      **Corona**: the rim of the penile glans.

•      **Frenulum**: thin strip of skin connecting the glans to the shaft on the underside of the penis.

•      Both are highly sensitive areas to the touch

**Blood Supply of The Penis**

**Arteries**

•      The corpora cavernosa is supplied by the deep arteries of the penis; the corpus spongiosum is supplied by the artery of the bulb.

•      In addition, there is the dorsal artery of the penis.

•      ***All the above arteries are  branches of the internal  pudendal artery.***

**Veins**

•      The veins drain into the internal pudendal veins.

**Lymphatics and innervation of The Penis**

**Lymph Drainage**

•      The skin of the penis is drained into the medial group of superficial inguinal nodes.

•      The deep structures of the penis are drained into the internal iliac nodes

**Nerve Supply**

•      Sensation

•      The nerve supply is from  the pudendal nerve and the  pelvic plexuses.

•      Erectile function

•      Parasympathetic(excitatory)

Sympathetic**(inhibitory)**

### Summary

**Penis Nerve Supply**

**•      Sensation**

**•      The nerve supply is from  the pudendal nerve and the  pelvic plexuses.**

**•      Erectile function**

**•      Parasympathetic (excitatory)**

* + **Sympathetic (inhibitory)**

## Unit Three Content..

### Topic 1: Obstetrics History Taking

**CONFIDENTIALITY:**

During history taking, the medical student should at all times show the patient the respect that is due to her;  while  full  confidentiality  must  be  maintained  at  all  times  bearing  in  mind  that  the  relationship between the professional and his client is based on mutual trust and respect.

**Classical Hippocratic Oath state**: “All that may come to my knowledge in the exercise of my profession or in daily commerce with men, which ought not to  be  spread  abroad,  I  will  keep  secret  and  will  never  reveal.”

Criminal Code of Malta [Ch.9:257]. The law reads as follows: “If any physician, surgeon, obstetrician or apothecary or, in general, any other person who, by reason of his calling or profession, becomes the

depository of any secret confided to him, shall, except when compelled by law to give information to the public authority, disclose such secret, he shall, on conviction be liable to a fine.”

**Summary of code of ethics**

• Informed consent; rapport

• Confidentiality; Privacy; Dignity: chaperon

• Woman’s views

• Informed consent for clinical evaluation and management

• Professional etiquette

• Ultimate primacy of the patient in making treatment decisions

**HISTORY TAKING IN OBSTETRICS**

**1.       Introduce yourself and obtain consent to take history:**

Ø  **“**Hello. I am Mr/Ms \*\*\*\*, a medical student. Do you mind if I ask you some questions about your medical condition?”

**2.       PARTICULARS:**

Ø  Name, age, address, marital status, occupation, religion, sex.

Ø  LMP, parity, gravity, EDD - Naegele’s rule

o   Gravidity is no. of pregnancies including current pregnancy (regardless of the outcome Normal or abortion)

o   Parity is no. of births beyond 24 wk gestation

**3.** **PRESENTING COMPLAINT:**

Ø  “What is the problem that brought you to the hospital/clinic?”

Ø   Best to record this in the patient’s own words.

Ø    “Were  you  referred  by  your  doctor  or  did  you  self‐refer  yourself  to  the hospital/clinic?”

Ø  Duration of the presenting complain.

**4.       HISTORY OF PRESENT ILLNESS (HPI)**

Ø  In the obstetric patient, its may be best to consider the “presenting complaint” in two

parts:

a.       The history of present illness or complaint; and

b.       The history of the current pregnancy.

Patient may not furnish sufficient details, in which case it will be necessary to amplify with specific

Directed questions. E.g**. SOCRATES** relating to pain: ‐

         i.            **Site**: where, local/diffuse

       ii.            **Onset**: rapid/gradual, pattern, worse/better since onset

     iii.            **Character:** sharp/dull/stabbing, burning/cramp/crushing

     iv.            **Radiation**: “Does the pain affect you anywhere else?” [to thigh/loin/elsewhere]

       v.            **Alleviating factors**: “What do you do to make yourself comfortable?”  “Is the pain better after menstruation?”

     vi.           **Time course**: “When did the pain start?”; if pain is chronic “What made you seek attention now” “Is the pain worse at any particular time of the cycle?”

    vii.            **Exacerbating factors:** “Is there anything that brings on the pain or makes it worse?”

  viii. **Severity & Impact on life**: “On a scale of 1 to 10, at what level would you classify the pain?”

"Does it interrupt your life?"

**5.        SYSTEMIC ENQUIRY OF ASSOCIATED SYMPTOMS**

v  GIT system:

v  Respiratory system

v  Cardiovascular system

v   Cardiovascular system

v  Urinary system

v  CNS

v  Musculoskeletal system

**6.       OBSTETRIC HISTORY**

**A.      HISTORY OF PRESENT PREGNANCY**

**a.       Menstrual history**

ü  The date of the first day of the last menstrual period (or LMP).

ü The length of the menstrual cycle refers to the time interval between the first day of the period and the first day of the subsequent period. This may vary from 21 to 35 days in normal women, but menstruation usually occurs every 28 days

**b.      The estimated date of delivery (EDD)**

ü  Can be calculated from the first day of the last menstrual period (LMP) by  adding  9months  and  7days  to this date.

ü  However, to apply **this Naegele's rule**, LMP should be  accurate and  the woman should have had  regular 28­day menstrual cycles.

ü  The average duration of human gestation is 269 days from the date of conception

ü  Therefore, in a woman with a 28­day cycle, this is 283 days from the first day of the last menstrual  period (14 days are added for the period between menstruation and conception)

ü   In a 28 day cycle, the estimated date of delivery can be calculated by subtracting 3 months from the first day of the LMP and adding on 7 days (or alternatively, adding 9 months and 7 days).

ü  It is important to appreciate that only 40% of women will deliver within 5days of the EDD and about two­thirds of women deliver within 10 days of EDD.

ü  The calculation of EDD based on a woman's LMP is therefore, at best, a guide to a woman as to the date around which her delivery is likely to occur.

ü  If a woman's normal menstrual cycle is less than 28 days or is greater than 28 days, then an appropriate number of days should be subtracted from or added to the estimated date of delivery.

ü For example, if the normal cycle is 35 days, 7 days should be added to the estimated date of delivery

a.       GRAVITY

üThe term**‘gravidity’** refers to the number of times a woman has been pregnant, irrespective of the outcome of the pregnancy, i.e. termination, miscarriage or ectopic pregnancy.

ü   A primigravida is a woman who is pregnant for the first time and a multigravida is a woman  who  has been pregnant on two or more occasions.

ü This term ‘gravidity’ must be distinguished from the term ‘parity’, which describes the number  of live­born  children and stillbirths a woman has delivered after 24 weeks  or with a birth weight  of  500g.

ü  Thus, a primipara is a woman who has given birth to one infant after 24 weeks.

ü A multiparous woman is one who has given birth to two or more infants, whereas, a nulliparous woman has not given birth after 24 weeks.

ü  The term ‘grand multipara’ has been used to describe a woman who has given birth to five or more  infants

ü A parturient is a woman in labour and a puerpera is a woman who has given birth to a child during the preceding 42 days.

b.       Gestation by dates

c.       FOCUSED ANTENATAL CLINIC

üWhen started, number of visits so far

üantenatal profile

o   haemoglobin

o   blood groups & rhesus factor

o   syphilis test – VDRL / RPR

o   HIV test

o   Urinalysis

o   Optional : blood slide for MPS, Stool for ova & cyst

üMedication presently and previously

üRadiological examinations during pregnancy

d.       Assess about Symptoms of pregnancy and their severity

ü  Nausea and vomiting

o   commonly occur within 2 weeks of missing the first period and it is  believed to be secondary to human chorionic gonadotrophin (hCG.

o    it is described as morning sickness, vomiting may occur at any  time of the day and is  often precipitated by the smell or sight of food.

o    Morning sickness commonly occurs in the first 3 months but, in some women, it may  persist throughout pregnancy

o    Severe and persistent vomiting leading to maternal dehydration, ketonuria and  electrolyte  imbalance is termed hyperemesis gravidarum

ü  Increased frequency of micturition

o   due to the pressure on the bladder exerted by the gravid uterus.

o   It tends to diminish after the first 12 weeks of pregnancy as the uterus rises above  the symphysis pubis,  i.e. into the larger abdominal cavity.

ü  Excessive lassitude or lethargy

o   is a common symptom of early pregnancy and may become apparent even before the first period is missed.

o   Often, it disappears after 12 weeks of gestation.

ü  Breast tenderness and heaviness,

o   which are really an extension of those experienced by many women in the premenstrual  phase of the cycle, are common during early pregnancy.

o   It is due to the effect of increasing serum progesterone as well as an increased retention of water.

ü  First maternal perception of fetal movements , also called ‘quickening’

o   is not usually noticed until 20 weeks gestation during first pregnancy  and 18 weeks  in the second or subsequent  pregnancies.

o   However, many women may experience fetal movements earlier than 18 weeks and others may progress beyond 20 weeks of gestation without being aware of fetal  movements at all.

ü  Some women may experience an abnormal desire for a particular food and this is termed pica

ü  Pseudocyesis

o   Development of symptoms and many of the signs of pregnancy in a woman who is not pregnant.

o   This is often due to an intense desire for or fears of pregnancy that may result in hypothalamic  amenorrhoea

ü  Leg cramps

ü  Limb swelling

e.       Enquire about symptoms which could indicate complications of pregnancy

üPer vaginal discharge, bleeding

üLAPs

üHeadache

üVisual disturbance

üPain on micturation

**f.        PREVIOUS OBSTETRIC HISTORY(Previous deliveries/miscarriages)**

üDetail each previous pregnancy - dates of deliveries, where birth took place, delivered at what gestation, how long did labour take, mode of delivery, condition of baby at birth,  sex, birth weight, other complications at delivery and postpartum, any blood transufusion,  did she breast fed if not why,  Length of labour & complications,  outcome, Previous miscarriages

**I.              GYNAECOLOGY  HISTORY**

üUse of family planning

 past medical surgical history

personal  social , economic history

family history

summary of the history

### Gynaecological History Taking

1.       Personal identification

2.       Chief complain

3.       History of presenting illness

4.       Review of systems

5.       Gynaecology history

ü  LMP, parity, last date of delivery

ü  Menstrual history

* + Age of menarche (10-16yrs)
  + Duration of flow
  + Regularity  and duration of cycle
  + Amount of bleeding (number of pads used)
  + History of dysmenorrheal

ü  Irregular bleeding – intermenestrual, post coital – so amount, timing, colour, pain associated

ü  Per vaginal discharge=colour, timing, amount, smell

ü  Per vaginal bleeding – colour. Amount, Smell, presence of clots

ü  Fertility / infertility

* + Use of family planning methods-duration & type
  + Any problems on fertility
  + Previous pregnancies & last date of delivery if applicable.

ü  Sexual history

* + Sexual partners and for how long
  + Any problems – dyspareunia, electile dysfunction
  + Frequency & timing of coitus (when applicable)

ü  History of STIs

* + Any previous STIs
  + Treatment for STIs
  + Use of protective measures

ü  Past gynaecological problems and operations

6.       Past medical surgical history

7.       Personal social economic history

8.       Family history

9.       Summary of the history

10.   Examination.

General Examination Techniques

**Prerequisites when examining a patient**

·         Use your senses well – listen, look, touch, smell, when examining

·         Knowledge of anatomical land marks

·         Ensure adequate lighting in the room & Secondary tangential lighting from a lamp

·         Ensure the place is quiet to allow proper percussion and auscultation

·         Ensure presence of necessary instruments

·         Ensure privacy.

·         Explain the procedure to the patient and ask permission to examine

·         Be thorough without wasting time, systemic without being rigid, gentle yet not afraid to cause discomfort

·         Try to look calm, organized and competent even if you do not exactly feel that way

·         Avoid expressions of disgust, alarm, distaste, or other negative reaction

·         Sequence the comprehensive examination in a manner designed to minimize the patient’s need to change positions and maximize your efficiency. Variations are possible and you may wish to develop a method of your own. In general it is helpful to move from head to toe.

·         Examine patient from the right side. Working from one side helps you master skills more quickly and promotes efficiency. Left-handed students find it awkward but are encouraged to practice it for convenience of themselves and others

**GENERAL TECHNIQUES USED IN PHYSICAL EXAMINATION**

**1.       Inspection:**

·         Definition: it’s the process of observing signs indicative of a healthy or a pathological state of a certain body part or system within a patient

·         Inspection should start as the patient enter consultation room (posture, gait, appropriate clothing, colour and moisture of skin, unusual odours), during history taking and during physical examination where you expose the areas of inspection as you validate the inspection findings with your patient (“I see a black spot here, have you noted it?)

**2.       Palpation**

·         Definition: it involves the use of your hands and fingers to gather information through sense of touch.

·         Certain parts of the hands are better than others for specific types of palpation eg:

o   Palmar surface & finger pads are most suitable to assess position, texture, size, form, consistency and presence of fluid or crepitus of a mass or structure

o   Ulnar surface of hands is suitable to assess vibration

o   Dorsal surface of hands is suitable to assess temperature.

·         Palpation may be light or deep controlled by amount of pressure applied. Light palpation always precedes deep palpation.

·         On the abdomen: always begin the palpation process with light systemic palpation of four quadrants initially avoiding areas of tenderness or problem spot

·         Light palpation is important in eliciting areas of muscle resistance and tenderness.

·         Deep palpation is normally only done in the abdomen

·         Palpation can be made with one hand or by two hands on top of each other with the upper one exerting the pressure (reinforced palpation)

·         Bimanual palpation is a technique whereby an organ is palpated using both hands.

·         Preparation:

o   Short nails

o   Patient to lie supine to relax abdominal muscles

o   Warm your hands to avoid producing muscle contractions

o   Stand on right of the patient (if the patient is in a low bed – sit on or kneel besides the patient’s right)

o   Ensure patient is comfortable

o   Ask patient to show areas of pain before you start palpation.

**3.       Percussion**

·         Definition: it involves striking one object against another, thus providing vibration and subsequent sound waves.

·         Your middle finger functions as the hammer and the vibration is produced by the impact of the finger against the underlying tissue.

·         Sound waves are heard as percussion tones (notes) that arise from vibrations in the body tissue

·         The degree of percussion tone is determined by the intensity of medium through which the sound waves travel eg air, fluid or solid:

·         Example

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Tone | Intensity | Pitch | Duration | Quality | Example |
| Tympanic | Loud | High | Moderate | Drum like | Gastric bubble |
| Hyper-resonant | Very loud | Low | Long | Boom like | Emphysematous lung |
| Resonant | Loud | Low | Long | Hollow | Health lung |
| Dull | Soft to moderate | Moderate to high | Moderate | Thud like | Over liver |
| Flat | Soft | High | Short | Very dull | Over muscle |

Percusion Technique

I.            Your (dormant) middle finger acts as a hammer (plexor) and fingers on the other hand (non-dorminant) acts as the striking surface (pleximeter) spread over surface of the body.  The middle finger should be slightly flexed, relaxed and posed to strike. Plexor should be at right angles with pleximeter. Snap the wrist of the tapping hand downwards, and with the tip of the plexor sharply tap the inter-phalangeal joint or second phalanx of pleximeter

**NB. The downward snap of the striking finger originates from the wrist and not from movement in the forearm or shoulder. It should be quick, sharp but relaxed wrist motion**

 Once your finger has struck, snap the wrist back quickly lifting the finger to prevent dampening the sound.

Use the tip and not the pad of the flexor finger

You can percuss one location several times for ease interpretation of the tone

II.            Alternative technique:

ü  Strike your finger or hand directly against the body eg clavicle and skull

(hydrocephalus)

**4.       AUSCULTATION**

·         Definition: means listening for sounds produced by the body.

·         It should be carried out last after the other techniques have provided information that will assist in interpreting what you hear

·         NB. Only abdomen you auscultate before palpation and percussion as the latter two techniques sometimes influence the bowel sounds.

·         Technique:

o   Ensure a quiet environment

o   Place bell or diaphragm of the stethoscope on naked skin as clothing obscures sound

o   Stabilize the stethoscope by holding the chest piece between the second and third finger.

o   When using the diaphragm press it firmly against the skin.

o   Avoid touching the tubing with your hands or allowing it rub against any surface as it creates extraneous noise.

o   Listen to sound not only for its presence or absence but also for its characteristics – intensity, pitch, duration and quality

o   Closing your eyes may help you focus on the sound

o   Target and isolate each sound, concentrating on one sound at a time.

### General Examination and Vital Signs

**General examination**

·         Observe the patients general state of health, height, built and sexual development

·         Note posture, motor activity, gait, dresss, grooming and personal hygiene – any odours of the body and breath

·         Watch patient’s facial expressions and notes manner and affect and reactions to things in the environment

·         Listen to manner of speaking and note state of awareness

**CLINICAL PARAMETERS**

**1.       PALLOR:**

ü  Sites in the body to examine pallor

v  Conjunctiva

v  Tongue

v  Palms

v  Nail bed: capillary refill

v  Sole of foot

v  Anus/ perineum

**Conjunctiva**: ask patient to be seated or lie supine, facing you. Place both thumbs on the margins of the lower eyelids and gently pull the skin downwards to evert lower lids (palpebral conjunctiva) and examine colour.

**Tongue /lips**: ask patient to open mouth and protrude the tongue. Observe colour of the tongue. Pull and evert the upper and lower lips gently to observe colour of inner parts.

**Palms**: ask the patient to supinate the two palms. Observe colour while you compare with your own palms

**Capillary refill test**: blanch the nail bed with the thumb and sustain the pressure for several seconds on fingernail or toenail. Release the pressure, observe the time elapsed before the nail regains its full colour. Normally this should occur almost instantly – in less than 2seconds.

**Soles**: with the patient lying supine or seated: look at the soles and observe colour

**Anus / perineum**: patient in lithotomy position (supine, hips and knees flexed) or lying on side, assess the perineum / anus for colour if applicable. Use gloves

2.       **JAUNDICE:**

Ø  Means having a yellowish discoloration.

Ø  Can be observed at: **sclera, mucous membranes and skin**

Ø  With patient seated or supine, gently elevate upper eyelids using both thumbs with patient facing a light source, ask him/her to look downwards to expose sclera and assess colour.

Ø  Ask patient to open mouth assess colour of mucous membrane

Ø  Examine skin and assess colour – light skinned people and infants.

Ø  Look at palms and soles for yellowish discoloration

**3.       HYPOXAEMIA : CYANOSIS**

·         A bluish discolouration these sites

·         TYPES:

o   Central cyanosis – lips & frenulum and

o   peripheral cyanosis – extremities at hands and feets

·         examine palms, sole, lips and frenulum for colour

**4.       HYPOXAEMIA: FINGER CLUBBING**

**I.**look at the shape of nails as compare with yours

**a.**Nail base angle should measure about 160 degrees.

**b.**Observe this by placing a ruler or a sheet paper across the nail and dorsal surface of the finger and examine the angle formed by the proximal nail fold and nail plate.

**II.**In clubbing the angle increases and approaches or exceeds 180 degrees

**a.**Ask the patient to place together the nail (dorsal) surfaces of the fingertips from the right and left hands

**b.**When nails are clubbed, the diamond – shaped window at the base of the nails disappears and the angle between the distal tips increases (shamroth technique)

**III.**Gently squeeze nail between your thumb and the pad of your finger to test for adherence of the nail to the nail bed.

**a.**The nail bed should feel firm

**b.**A boggy nail base accompanies clubbing.

**5.       DEHYDRATION**

·         Sites: **fontanels, eyes, mucous membranes, skin (abdomen or chest)**

·         **Fontanels –** below 18months – sunken in dehydration

·         **Eyes –** sunken in dehydration. Absent tears in dehydration

·         **Mucous membranes –** dry in dehydration

·         **Skin turgor –** pinch skin over chest or abdomen using thumb and index finger. Observe duration skin takes to go back.

**6.       OEDEMA**

·         **Sites:**face, sacrum, abdomen. Extremities.

·         **Face:** observe peri-orbital oedema

·         **Sacrum:** press sacral area with thumb for 30seconds

·         **Lower limbs:** use both thumbs to apply pressure on the lower limbs 1cm above medial malleolus for 30seconds as you look patient face.assess for pitting by running you finger over the site

·         **Report oedema as –** bi- or unilateral, tender or non-tender, pitting or non-pitting.

**7.       LYMPH NODES.**

ü  **S**mall, mobile and painless lymph nodes are often palpable in healthy individuals

ü  A painful lymph node is suggestive for inflammatory process

ü  A firm or fixed non-motile LN is very suggestive for a malignant process

Technique:

Inspect area of LN for apparent LN, oedema, erythema, red streaks and skin lesions

Using pads of 2rd 3rd & 4th fingers gently palpatefor superficial nodes

Note location, consistency, mobility, tenderness, size, shape, discreteness & warmth. Move skin over area.

Head & neck LN: Palpate the anterior LN from behind the patient and vice varsa. Sternocleidomastoid muscle divide into anterior & posterior

Axillary LN:

Other LN.

**8.       ORAL THRUSH**

**VITAL SIGNS**

**The word vital signs means essential to life.**

**T**hey include:

·         Pulse rate

·         Respiratory rate

·         Blood pressure

·         Temperature

**1.       PULSE RATE**

Radial pulse is mostly used to assess:

·         Heart rate

·         Cardiac cycles per minute

·         Collapsing pulse

·         Arrhythmias

·         Condition of the blood vessels

·         Assessing the blood pressure

Sites for taking pulse rate:

·         Radial: on the thumb side of the wrist

·         Temporal: lateral to eye brow on the temporal bone

·         Brachial: medial aspect of cubital fossa

·         Femoral: upper inner aspect of thigh

·         Popliteal: behind the knee

·         Dorsalis pedis: upper surface of the foot

·         Carotid pulse: side of the neck

**Characteristics of pulse**

ü  Rhythm:

o   Regular

o   Irregular

§  Regular irregular

§  Irregular irregular

ü  Character:

o   Feeble (small volume – eg dehydration)

o   Pounding eg anxiety

o   Collapsing

ü  Pulse volume.

**2.       BLOOD PRESSURE**

Requirements:

·         Mercury sphygmomanometer

·         Aneroid sphygmomanometer

·         Electronic sphygmomanometer

·         Stethoscope

False reading may occur in:

·         Bp machine defect

·         Dehydration

·         Anxiety

·         Exercise

·         Individuals with small and large biceps

·         Differences between supine and erect Bp especially in elderly.

**Classification of hypertension**

**Pre-hypertension**: Systolic BP 120-140 or diastolic BP 80-90.

**Stage I hypertension**: Systolic BP > 140-160 or diastolic BP >90-100.

**Stage II:** Systolic BP > 160 or diastolic BP > 100.

**3.       TEMPERATURE**

Sites:

·         Armpit (most used)

·         Oral – adults & children who can follow instructions

·         Groin – in children

·         Rectal – for children and unconscious. Most accurate but uncomfortable.

Requirements:

·         Thermometer:

o   Mercury

o   Electronic

o   Inflated auxiliary thermometers

o   Low reading thermometer

·         A container containing cotton soaked in disinfectant. Change disinfectant every 24hours

·         A quick assessment of temperature can be done by use of back of the hand.

**4.       RESPIRATORY RATE**

Note character and rhythm.

**1.     OBSTETRIC ABDOMINAL EXAMINATION**

Regular abdominal examination is an important component of both antenatal care and monitoring labour

By abdominal examination you can ascertain:

·         The size of the uterus and note whether it corresponds to the period of amenorrhea

·         The size of the foetus

·         The lie presentation and attitude of the foetus

·         The relative sizes of the brim of the pelvis and the presenting part

·         Whether the foetus is alive

·         The presence of the abnormal condition eg excess liquor amnii, twin pregnancy, abdominal tumours

**Requirements**

·         Foetoscope

·         Watch with second hand

·         A bowl with dry gauze and cotton swabs

·         Tape measure

·         Receiver for the dirty swabs

·         Couch

·         Bed sheet

**Examination**

Explain the procedure to the patient

Ensure privacy

Wash and dry your hands to ensure they are warm

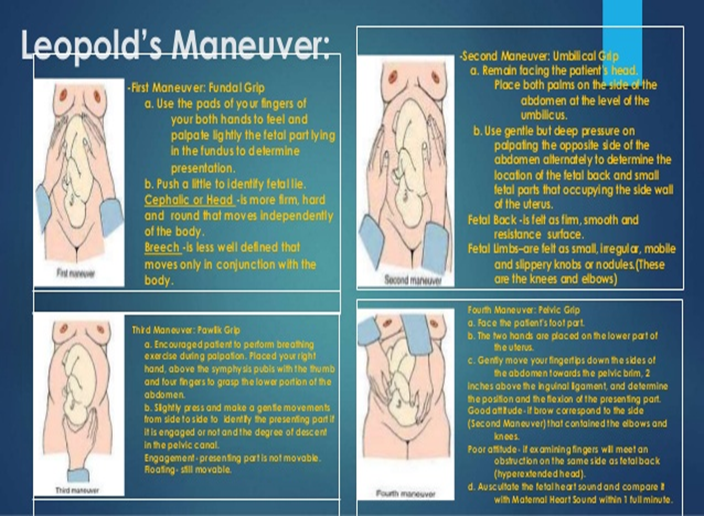
Expose the abdomen

**Inspection**

Inspect for shape, size, skin, linear nigra, striae gravidarum, scars, foetal movements, umbilicus

**Palpation**

**LEOPOLD’S MANEUVER**



* First maneuver: Fundal Grip
* Second maneuver: Umbilical Grip
* Third maneuver: (1st pelvic grip) = The Pawlick's Grip,
* Fourth maneuver: (2nd Pelvic Grip) = pelvic grip

**First Leopold’s maneuver: Fundal Grip**

Facing the mother, palpate the fundus with both hands

–      Assess for shape, size, consistency and mobility

Fetal head: firm, hard, and round

–      Moves independently of the rest

–      Detectable by ballottement

Breech/buttocks: softer and has bony prominences

–      Moves with the rest of the form

**Fundal height**

·         Using your left hand, place either the ulnar/radial border of the index finger on the abdomen from xiphisternum

·         Apply pressure as you move downwards in steps to locate the underlying mass

·         Once the mass is encountered maintain the hand or finger at that level

·         Using the right hand, determine the number of fingerbreaths from the superior border of the umbilicus up to the level of fundus

·         Each finger breath multiplied by 2. Fundal height at umbilicus is 22/40, at sympysis pubis is 12/40

·         Alternatively you can use tape measure to check fundal height from symphysis pubis through umbilicus to xiphoid sternum. Note the fundal height

**Second Leopold’s maneuver - Determine position of the back (lie)**

·         Lie is the relation of the baby’s long axis to the mother’s uterus long axis

·         Still facing the mother, place both palms on the abdomen

·         Hold right hand still and with deep but gentle pressure, use left hand to feel for the firm, smooth back. Repeat using opposite hands

·         Feel for:

·         The foetal poles

·         Regularity or irregularity and convexity

·         Absence of foetal poles in the flanks = **longitudinal lie**

·         Presence of foetal poles on the flanks = **transverse lie**(long axis of the fetus is perpendicular to that of the mother’s)

·         **Oblique lie =**long axis of the fetus is 0-90 degrees (or 90-180 degrees) to that of the mother’s

·         Uniform, firm convexity of contour = side of the foetal back

·         Determine whether convex contour is nearer the front or far out in the flank – gives idea about **position**

**Third Leopold’s Maneuver - presentation**

·         Determine what part is lying above the inlet.

·         Using the right hand apply the pawlik’s grip (single handed palpation) to fell the part of foetus overlying the pelvic inlet

·         If transverse, any mass felt is most likely the shoulder = **shoulder presentation**

·         If longitudinal lie differentiate between **cephalic and breech presentation.** A hard rounded mass indicates cephalic presentation

·         Pawlick’s grip gives precise findings when head is not engaged

**Fourth leopold’s maneuver - engagement**

·         **Engagement i**s checked while facing the mothers lower limbs and with both hands placed on the lower abdomen

·         If the fingers seem to meet below the presenting part then engagement has not occurred. When engagement occurs, the head will be fixed on the pelvis

·         Before deep engagement, as the fingers are passed down the presenting part, the area that is most prominent on the head is noted.

·         If the cephalic prominence is on the side of the small parts (limbs), it implies the head is well flexed hence **Vertex presentation**

·         If prominence is felt equally on both sides – deflexed hence **brow presentation**

·         **If**prominence is on the side opposite that with the small parts and indistinct back curvature, the findings suggest **face presentation.**

**Attitude**

·         Refers to the position of the foetal head to its trunk.

·         The normal position is **complete flexion giving vertex presentation**

·         When deflexion = **brow presentation**

·         When extension = **face presentation**

**DENOMINATOR**

·         Denominator is the arbitrary point on the presenting part used as a point of reference in denoting the position of the presenting part in relation to the pelvis.

o   Vertex presentation - the denominator is **OCCIPUT**

o   Brow presentation – the denominator is **sinciput**

o   Face presentation – the denominator is **mentum or chin**

o   Breech presentation – the denominator is **sacrum**

**Position**

·         Relation of denominator (occiput/ sacrum) of presenting part to the quadrants of pelvis.

·         Eight positions are described

o   When the denominator is directed towards symphysis pubis it gives a **direct anterior position** and when directed towards sacrum it gives **direct posterior position**

o   When denominator is directed towards the ileopectineal eminences, the position is **left or** **right anterior position**

o   If the denominator is directed towards  the mid points of the ileopectineal line, it gives **left or right anterior position**

o   If the denominator is directed towards the left or light sacro-iliac joint then it is left or right posterior position

·         Normal presentation is anterior position with commonest being left occipital anterior.

·         Left Occipito Posterior (LOP) – 3%

·         Left occipito lateral (LOL) – 40%

·         Left occipito anterior (LOA) – 15%

·         Right occipito anterior (ROA) – 10 %

·         Right occipito lateral (ROL) – 24%

·         Right occipito posterior (ROP) –

DESCENT AND ENGAGEMENT

·         Refers to entry of the presenting part into the pelvis

·         Head is the presenting part in 99% of all labours

·         Descend is recorded in terms of the span of the foetal head still palpable in the abdomen above the level of symphysis pubis

·         The whole span of the head is subdivided into 5 fifths and each finger breaths spans 1 fith (1/5)

·         When is completely free, the descend is 5/5 then to 4/5, 3/5, 2/5, 1/5, 0/5.

·         Once the widest diameter of foetal head has entered, **only 2/5 is palpable** and strictly speaking, this is **when engagement has occurred**

·          If a portion has sunk in the pelvis, pawlik’s grip may not determine the identity of the presenting part.

**AUSCULTATION**

v  Place the foetoscope on the back of the foetus, apply sufficient pressure to exclude external sounds

v  Take note of foetal heart rate count and regularity.

### OBSTETRIC VAGINAL EXAMINATION

**Indications:**

ü  To confirm labour

ü  Pelvic assessment

ü  Assess progress of labour

ü  Assessment of cervical ripening before induction of labour

ü  Diagnose or rule out complications of labour

**Contraindications**

ü  Suspected PROM

ü  Suspected placenta praevia.

**Procedure**

ü  Explain the procedure to the patient

ü  Let patient empty bladder

ü  Provide privacy

ü  Ensure adequate working space

ü  Aseptic technique

**Inspection of the external genitalia**

ü  Inspect for: scars, warts, scratch marks, varicose veins, hair distribution,

ü  Any discharge – colour, consistency, smell, quantity,

ü  State of urethral meatus

**VULVA TOILET**

ü  Aseptic technique – sterile gloving

ü  Roll the sterile cotton wool swabs into five small balls and Soak them in a galipot containing antiseptic solution

ü  Take them on the right hand, then drop one the left hand – swab with left hand the furthest labia majora from the top to the perineum. Similarly, swab the other side of labia majora.

ü  Similarly, swab labia minora on both sides with the next two swabs using left hand.

ü  Separate labia minora using your left hand then swab vestibule with the swab in the right hand from clitoris to fourchette using the right hand.

ü  Drape the mother by placing the sterile towel under her buttocks and another one on the abdomen up to the hairline of the pubic area.

**SPECULUM EXAMINATION IN OBSTETRIC**

 Read section on speculum examination below

**DIGITAL VAGINAL EXAMINATION**

ü  Lubricate index and middle fingers of the right hand

ü  Separate the labia with two fingers of the left hand

ü  Insert index and middle finger of the right hand into the vaginal canal

o   The terminal phalange of the middle finger is inserted first and pressed against the perineal body in order to relax introitus

o   The index finger is then slipped in and the two fingers are directed toward the cervix

ü  Assess features at the cervis:

o   Dilatation of the cervix

o   Degree of thinning and softness (effacement)

o   Presence or absence of membranes

o   Confirm presenting part and its state eg moulding, caput

o     Direction of sutures and position of fontanels

o   Level of presenting part compared to the ischial spines

ü  **pelvic assessment**

o   Feel for sacral promontory

o   Assess curvatures of the sacrum by sweeping your fingers downwards towards the outlet

o   Assess prominences of the ischial spines and tip of coccyx by sliding the examining fingers along the iscial spines and coccyx

o   Measure the sub-pubic angle by fitting the two examining fingers in the sub-pubic angle

o   Measure the inter-tuberous diameter by fitting the four knuckles of the examining hand between the ischial tuberosity.

**GYNAECOLOGICAL PELVIC EXAMINATION**

**Indication**

v  Suspicion of pathology of female genital system

v  Exclusion of pathology

**Contraindication**

v  Intact hymen

**Requirements**

v  Couch with stirrups

v  Sterile speculum: cusco’s, sims’, ferguson’s

o   **Assignment: types of speculums and different sizes. How to examine patient using univalve speculums.**

v  Sterile gloves

v  Non-sterile gloves

v  Receiver with decontamination solution.

v  chaperone, consent, good lighting

**Procedure**

Preparation

v  Prepare environment for pelvic examination – privacy, equipment & supplies

v  Explain the procedure to the patient

v  Patient empty bladder, remove underclothing, position in lithotomy

v  Cover her to avoid unnecessary exposure

**Method: speculum examination**

1.       Maintain infection prevention throughout the examination – wash hands, put on sterile gloves, use high-level disinfected / sterile instruments

2.       Inspect external genitalia to screen for STI / FGM

v  Warts, abnormal discharge, ulcers, bleeding from vagina, sores, swelling, presence and distribution of hair, obvious anatomical anomalies, female genital mutilation, cosmetics (eg rings)

3.       Reassure the patient throughout the procedure

4.       Choose speculum size for the patient

5.       Warm blades under a stream of tepid  water

6.       Hold speculum in the right hand while the index finger of the left hand presses downwards on the fourchette to expose the introitus

7.       Slide the closed blades obliquely (away from the urethral area and clitoris) over the fingers into the introitus , introduce the instrument into the vagina

8.       While inserting the instrument rotate it to a clockwise direction until the anterior and posterior blades run along the anterior and posterior vaginal walls with the handles pointing towards the anus.

9.       Open the blades to expose the cervix

10.   Inspect cervix- erosion, colour, growths, friability, discharge,

11.   Take lab specimens if necessary for investigation for PAP smear for cytology, vaginal secretions for microscopy.

12.   Rotate the speculum and inspect vaginal wall for: warts, abnormal discharge, sores, bleeding

13.   Remove the speculum gently in horizontal position and place it in decontamination solution.

DIGITAL VAGINAL EXAMINATION IN GYNAECOLOGY

v  Put on sterile gloves

v  Clean the vulva

v  Insert the index and middle fingers of right hand gently into the vaginal canal while you avoid touching clitoris with your thumb

v  Examine the cervix:

ü  Ostium- open or closed

ü  Irregularity

ü  Growth

ü  Consistency

ü  Mobility

ü  Tenderness

v  Examination of uterus – bimanual examination

ü  Place your left hand on the abdomen, just above sympysis pubis

ü  Locate uterus by feeling it between your left hand over abdomen and the finger tips of your right index and middle fingers placed on the cervix

ü  Palpate for:

o   size, shape and consistency, mobility,

o   position : anteverted or retroverted

v  **Examination of the adnexia**

ü  locate the left fornix and place the finger tips in this fornix

ü  together with left hand placed over the left side of lower abdomen, try to palpate for any mass or tenderness

ü  repeat same on the right  side

v  Excitation of the cervix for endometritis

ü  use your right index and middle fingers to gently move the cervix from left to right and check for any pain

v  palpate the anterior, left and lateral walls of the vagina walls for: ruggae, growth, tenderness

v  check vaginal muscle tone by asking the client to tighten vaginal muscle

v  check for cystocele and rectocele

ü  press the left index and middle finger downwards and ask patient to cough = rectocele

ü  press the left index and middle finger upwards and ask patient to cough = cystocele

* palpate bartholins glands on both sides of the labia majora
* press on the trigone muscle to exclude cystitis
* Milk the skenes glands with left hand.
* Palpate the inguinal LN.

### EXAMINATION OF THE MALE GENITALIA

**Requirements:**

ü  Gloves

ü  Penlight

Inspection:

ü  The pubic hair characteristics and distribution

ü  The glans penis (retract the foreskin if patient uncircumcised):

o   Colour

o   Smegma

o   External meatus of urethra

o   Urethra discharge

Palpation:

ü  The penis:

o   Tenderness

o   Induction

ü  Strip the urethra for any discharge (you can ask the patient to perform this part of procedure for you:

o   Firmly compress the base of the penis with your thumb and forefinger and move them towards the glans

o   Press the glans penis between the thumb and forefinger

o   Collect discharge

Scrotum and ventral surface of the penis:

Inspection:

ü  Colour

ü  Texture

ü  Asymmetry

ü  Unusual thickening

ü  Presence of hernia

ü  Trans – illuminate any masses in the scrotum

o   When any mass is felt other than the testicle or spermatic cord determines whether it is filled with gas, fluid or solid material using penlight

**Palpation:**

a.       Inguinal canal for direct and indirect hernia

ü  With patient standing, ask him to bear down as if having bowel movement

ü  While he is straining inspect the areas of inguinal canal and the region of fossa ovalis

ü  Ask patient to relax, insert the pulp of your examining finger into the lower part of the scrotum and carry it upward along vas deferens into the inguinal canal. (the finger depends on the size of the external ring which is normally palpable)

ü  With your finger placed at the external ring ask the patient to cough and feel for a viscous mass against your finger (present if a hernia is present)

b.       The testis, epididymitis, vasa deferentia

ü  Use the thumb and first two fingers to assess:

                                                   i.      Consistency

                                                 ii.      Size

                                               iii.      Tenderness

                                               iv.      Fluid

                                                 v.      Lumps or nodules

c.       The inguinal lymph nodes

d.       Cremasteric reflex

ü  Elicit the cremasteric reflex bilaterally

ü  Stroke the inner thigh with a blunt instrument such as the handle of the reflex hammer or with your finger

ü  Normally the testicle and scrotum rise on the stroked side

### EXAMINATION OF THE FEMALE BREAST AND AXILLA

Breast is an appendix of the skin in the milk-lines from axilla to groin.

The development has different stages – tanner’s classification

**Preparation**

Undress patient as far as waist, sits upright

It is necessary after examining the patient sitting upright to ask the patient to lie flat and reexamine the breasts

Both breasts must be exposed completely

**Method**

**Inspection**

Ø  While patient in the following positions:

o   Arms hanging loosely at the sides (arms aside)

o   Arms held over head

o   Arms held at hips

o   Leading (bending) forward

Ø  Inspect each breast and compare them for:

o   Size

o   Symmetry

o   Contour

o   Skin colour

o   Venous pattern

o   Lesions

Ø  Inspect nipple for:

o   Size (compare both breast)

o   Retraction

o   Discharge

o   Ulceration

Ø  Inspect the areola for pigmentation

Palpation

Ø  Systematically palpate the breast, axilla and supra-clavicular regions

Ø  Ask the patient to find first herself the lump and to point the lesion she has detected, before you attempt to do so and before you start palpation

Ø  Start with the normal breast to have her impression of the normal breast

Ø  Palpate the areola and nipple and finish with axilla

**Techniques**

Ø  Two techniques:

o   Palpation “with the flat of the hand”

o   Palpation “between the pulps of the fingers and the thumb”

Ø  **Flat-hand- technique:**

o   Exert a slight pressure on skin with the pulps of the middle and end phalanges of the fingers 2-5 and perform a small circular movement

o   Normal breast gives a firm lobulated impression with fine nodularity a feature particularly before the periods. In fat and after menopause expect to feel both lobulation and nodularity less easily

o   With flat-hand-technique accepted techiniques include:

§  Vertical zigzag palpation

§  Concentric circular palpation

§  Quadrant palpation

o   Most used technique is quadrant for clinician and self breast examination.

Ø  Perform palpation calmly, with patience, without skipping any part including axilla.

Ø  Palpation of areola:

o   Gently squeeze the areola skin between thumb and index together with a rolling movement (small retention cysts and glands of Montgomery can be noted as well as small centrally located tumours)

o   Indirectly by pressing with the top of index on different places of the areola, elicit nipple secretion, if present.

Ø  Palpation of nipple

o   Inform patient the procedure may hurt

o   Perform a short but firm squeeze of nipple between thumb and index finger

o   Notice secretion

Ø  Palpation of the tail of Spence and axillae

o   Palpate the tail as it enters axillae by gently compressing the tissue between the thumb and fingers

Ø  Palpation of the LN

o   With patient seated with arms flexed at elbow

o   Support the patient’s **right lower arm** with **your right hand** to examine the **right axilla & vice varsa**

o   Examine-apex, medial, lateral, anterior & posterior.

NB: Make always a well documented record of your examination

v  Localization : name the quadrant or describe the location using “hours”

v  Size: in mm or cm

v  Shape: round –discoid – regular – irregular

v  Consistency: very soft, soft, firm, hard, stony or bony hard

v  Contour: in relation to surrounding tissue

v  Tenderness: yes or no, spontaneous

v  Mobility: in relation to the skin, breast tissue, fascia, thorax wall.

### COMMON INVESTIGATIONS IN REPRODUCTIVE HEALTH PRACTICE

* ***Learning objectives***
* •       *At the end of the lecture, the student is expected to be able to:-*

–   *Know the scope of the common investigations in RHC*

–   *Acquire insight into the rational & objective request for relevant investigations*

–   *Apply the same or similar investigations in different clinical situations*

–   *Interpret results of investigations for the benefit of patients*

***Introduction: Investigations***

•        *Important aspect of patient management*

•        *Should always be rational/objective*

•        *Normal parameters should be known*

•        *Can be expensive*

•        *May be invasive*

•        *Supplement but not replace clinical acumen*

***“Good, comprehensive medical history & physical examination remain key to paient management”***

***PURPOSE FOR INVESTIGATIONS***

•       *Confirmation of diagnosis*

•       *Making diagnosis*

•       *Estimate of disease severity*

•       *Monitoring effect of treatment*

•       *Monitoring recurrence*

•       *Screening for disease*

### Common investigations in obstetrics

***Antenatal care (ANC)***

•        *[Hb] & HCT*

–    *To screen or confirm anemia*

–    *Forms a basis for supplementation*

•        *VDRL*

–    *Screening for syphylis – affects fetal outcome*

–    *Specific treponemal tests before treatment (e.g. TPHA)*

•        *Blood group*

–    *Establishes risk of feto-maternal incompartibility & hemolytic disease*

–    *Prepares for transfusion needs*

•       *Urinalysis - Screening for protein, glycosuria, UTI, etc*

•       *HIV - PMTCT; Entry into prevention of transmission; Enables provision of support - psycho-social & medical*

•       *Obstetric ultrasound - Appraisal of fetal growth; Fetal abnormality screening; Fetal well-being assessment: BPPS; Doppler studies; PET prediction*

***Full blood count (FBC)***

*Has multiplicity of value*

*Examples of significance:*

•         *[Hb] & HCT*

–    *Presence/absence of anemia; Etiology not implied*

•         *Cell indices - MCV, NCH, MCHC – may allot anemia to broad categories:*

–    *Microcytic*

–    *Hypochromic*

–    *Megaloblastic*

–    *Mixed*

•       *WBC count*

–   *Total count*

•     *Marked elevation - acute infections (bacterial/viral)*

•     *Moderate elevation – chronic infections*

–   *Differential count*

•     *Neutrophilia – acute bacterial infections*

•     *Lymphocytosis –viral or chronic bacterial infections*

•       *Peripheral blood film (PBF)*

–   *RBC’s*

•     *Normocytic normochromic; hypochromic microcytic*

•     *Polychromasia; reticulocytosis;megaloblasts*

•     *Anisocytosis; poikilocytosis;tear drop cells*

•     *Spherocytes; sickle forms*

–   *WBC,s*

•     *Polymorphs – nuclear segmentation*

•     *Toxic granulation*

•     *Malaria parasites (MP,s)*

•       *Erythrocyte sedimentation rate*

–   *None specific*

–   *May indicate infection*

•       *Platelet count*

–   *Absolute count/concentration*

***Infections in pregnant state***

1. ***Urinary tract infections (UTI)***

–            *Urinalysis:- appearanc; pH; blood; sugar; proteins; nitrites; urobilinogen; leukocytes; bilirubin; specific gravity; ketones*

–            *Microscopy:- pus cells; epithelial cells;casts; crystals; RBC,s; bacteria; yeast cells; trichomona vaginalis*

–            *Culture*

–            *sensitivity*

**2.       *Respiratory tract infections***

–   *FBC; chest X-ray; sputum – AAFB,s*

**3.      *Chorioamnionitis***

–   *FBC +ESR; vaginal swab – M/C/S*

**4.       *Malaria***

–   *MP’s*

5.        ***Blood coagulation disorders –thrombo-embolic disease & DIC***

•       *Coagulation screen*

–   *Bleeding time; clotting time; APTT/KCCT; PTI/INR; platelet count*

•       *Thrombus localization*

–   *Doppler studies*

–   *Venography; radioisotope studies; thermography*

6.        ***Hypertensive disease in pregnancy***

•       *Renal function tests (RFT,s)*

–   *Urinalysis – protein, blood*

–   *U/E; uric acid; creatinine; creatinine clearance*

–   *Renal ultrasound*

•       *Liver function tests (LFT,s)*

–   *Bilirubin, liver enzymes*

•       *Coagulation screen; platelet count*

•       *Obstetric utrasound –FWB; doppler studies*

***7.     Value of ultrasound in obstetrics***

•        *Diagnosis of pregnancy*

–    *Intaruterine; extra-uterine*

•        *Fetal growth monitoring*

–    *Appropriateness of growth (AGA;SGA; LGA)*

–    *IUGR (asymmetrical; symmetrical)*

–    *Fetal weight, sex, viability*

•        *Fetal well-being*

–    *BPPS (fetal tone; fetal movements; respiratory movements; AF volume; placental echogenicity)*

•       *Pelvimetry*

•       *Gestation estimation-timing of specific epiphyseal plates’ closure*

•       *Diagnosis of twins*

•       *Diagnosis of extra-uterine pregnancy*

8.          ***Diabetes mellitus***

•       *Urinalysis*

–   *Glycosuria; ketonuria; proteinuria*

•       *Blood sugar*

–   *RBS; FBS; OGTT; PPBS; Serial BS*

•       *Glycolysated Hb (Hb1c) - [N<6%]*

•       *Obstetric ultrasound – IUGR; Macrosomia; BPPS; Polyhydramnios; etc*

9.        ***Rhesus isoimmunization***

•       *Indirect Coombs test (ICT)*

•       *AF spectrophotometry –absobance deviation at 450nm*

•       *Cord blood at birth*

–   *[Hb] + hct*

–   *Direct Coombs test*

–   *Bilirubin*

*10.*       ***Anemia & Antepartum hemorrhage***

•        *Anemia*

•     *FBC+PBF*

•     *Stool o/c*

•     *MPs*

•       *Antepartum hemorrhage (APH)*

•     *Placental localization*

•     *Ultrasound; EUA*

•     *Coagulation screen*

•     *[Hb] = hct*

11.   ***Antepartum hemorrhage (APH); Establishment of fetal maturity***

*Septicemia & endotoxic shock*

•     *RFTs including urine output*

•     *Blood cultures*

•     *Coagulation screen, especially platelet count*

•     *Critical care investigations (spo2, spco2, etc)*

*Establishment of fetal maturity*

•     *Amniocentesis*

–   *Surfactant or ‘shake’ test*

–   *Lecithin:sphingomyelin (L/S) ratio (N > 1:2)*

### Common investigations in Gynaecology

•       ***FULL BLOOD COUNT***

“***Important in differentiating  infective from none-infective  conditions”***

•       ***Value of ultrasound scan in gynecology***

•        *Diagnosis of pelvic masses*

–    *Solid – fibroids; ovarian; other*

–    *Cystic – ovarian; TOM; PCOD*

–    *Fluid in the pelvis – PID; pelvic abscess; ectopic pregnancy*

•        *Management of infertility*

–    *Follicular growth monitoring; TVS for ovum retrieval*

•        *Intrauterine diagnosis*

–    *Endometrial hyperplasia; ‘lost’ IUCD; hematometra*

•       ***Radiology in gynecology***

•       *Hysterosalpingogram*

–   *Scope – uterine cavity; endoslpinx; spill*

–   *Pathology:*

•     *Submucous/intramural fibroids*

•     *Tubal block (cornual, mid-section,terminal)*

•     *Tubal loculations/peri-tubal adhesions*

•     *Fimbrial adhesions*

•     *hydrosalpinges*

•       *X-ray sella turcica – hyperprolactinaemic galactorrhoea syndrome*

•     *Erosion of clinoid processes & pituitary fossa*

•       *Embryonal tumours – cystic teratomas*

•       *‘Lost’ IUCD – tracer IUCD inserted*

•       *Extra-uterine pregnancy*

•       ***Infertility –MALE***

•         *Semen analysis*

•      *Appearance*

•      *Volume*

•      *pH*

•      *Liquefaction*

•      *count*

•      *Motility*

–    *Progressive*

–    *Sluggish*

–    *non-progressive*

–    *non-motile*

•      *Vitality*

•      *Morphology*

•      *Agglutination*

•      *Pus cells*

•       *Post-coital test*

•     *Motility; agglutination*

•       *Sperm-mucous interaction tests*

•     *Direct (spouses) or crossed*

•       *Hormonal profile*

•       *Testicular biopsy*

•       ***Infertility – FEMALE***

•       *Radiological – HSG; X-ray sella turcica*

•       *Pelvic ultrasound scan*

–   *Uterine pathology (fibroids; adenomyosis)*

–   *ovarian pathology (PCOS)*

•       *Hormonal profile*

–   *FSH; LH; PRL; testosterone; progesterone; estradiol*

•       *PCT*

•       *Sperm-mucus interaction tests*

•       *Visual fields*

•       *CT-scan/MRI*

•       *Karyotype*

•       ***Diagnostic endoscopy in gynecology***

•        *Laparoscopy*

•     *Chronic pelvic pain*

•     *Endometriosis*

•     *Infertility*

•     *Ectopic pregnancy*

•     *Pelvic inflammatory disease*

•        *hysteroscopy*

•     *Endometrial pathology*

–    *Submucous myomas; Uterine synachie; Endometrial polyps; abnormal uterine bleeding*

•        *Culdoscopy*

•       ***Carcinoma of the cervix***

•                 *Papanicolou (pap) smear*

•                 *Colposcopy*

•                 *Cone biopsy*

•                 *Loop electro-excision procedure (LEEP)*

•                 *EUA, bopsy and staging*

•       ***Abnormal uterine bleeding***

–   *D&C*

–   *Fractional curettage*

–   *Hysteroscopy*

–   *Hormonal profile*

•       ***Gestational trophoblastic disease***

•       *Hydatidiform mole*

–   *PDT/B-hCG*

–   *US scan – ‘snow storm appearance’*

•       *Choriocarcinoma*

–   *B-hCG*

–   *Metastatic disease*

•     *CXR (cannon balls):CT-scan/MRI (liver.renal,brain)*

•     *Lumbar puncture; LFTs; RFTs*

•       ***Ectopic pregnancy***

•       *PDT/b-hCG*

•       *Pelvic U/S scan*

•       *Laparoscopy*

•       *Paracentesis*

•       *Culdocentesis*

•       ***Sexually transmitted infections (STIs)***

•       *Vaginal swab – M/C/S*

–   *HVS*

–   *Cervical swab*

•       *VDRL*

•       *HIV ELISA*

Unit Four Content..

Topic 1: PUBERTY

 Period when the endocrine and gametogenic functions of the gonads have first developed to the point where reproduction is possible

This is characterized by sequence of events by which a child becomes a young adult:

The beginning of gametogenesis

Secretion of gonadal hormones

Development of secondary sexual characters and reproductive functions

Sexual dimorphism is accentuated.

**Factors affecting the onset of puberty**

The age of onset of puberty varies and is more closely correlated with osseous maturation than with chronological age

Genetic/Ethnic factors

Environmental/Geographical factors

**Prepubertal stage (8–9 yr of age)**

The hypothalamic-anterior pituitary-gonadal axis is suppressed by;

Neuronal restraint pathways

Negative feedback provided by minute amounts of circulating gonadal steroids

Thus there are **undetectable serum levels of;**

luteinizing hormone (LH)

sex hormones (i.e., estradiol in girls, testosterone in boys)

PREPUBERTY STAGE

Evidence of hypothalamic-anterior pituitary-gonadal interaction during the prepubertal period resides in the fact that serum **follicle-stimulating hormone (FSH) concentrations are detectable in most children and may be increased (with serum LH concentrations) in;**

Turner syndrome

Anorchia

**Peripubertal period (1-3 yr before the onset of puberty)**

Pulsatile secretion of low levels of LH during sleep secondary to endogenous episodic discharge of hypothalamic gonadotropin-releasing hormone (GnRH).

Nocturnal pulses of LH continue to increase in amplitude and, to a lesser extent, in frequency as clinical puberty approaches.

Serum LH concentrations rise earlier in the course of the pubertal process in boys than in girls.

This pulsatile secretion of gonadotropins is responsible for;

Enlargement and maturation of the gonads

The secretion of sex hormones

Appearance of the secondary sex characteristics

NB

**GnRH is the major, if not the only, hormone responsible for the onset and progression of puberty.**

A second critical event occurs in middle or late adolescence in girls, in whom cyclicity and ovulation occur.

 A positive-feedback mechanism develops whereby rising levels of estrogen in midcycle cause a distinct increase of LH.

**Puberty in Girls (8-13yr)**

**Thelarche** (Development of Breasts) - Breast bud - 10–11 yrs

**Pubarche** (Development of axillary and pubic hair) - Appearance of pubic hair - 6–12 mo later

Peak height velocity occurs early (at breast stage II–III, typically between 11 and 12 yr of age) in girls and always precedes menarche.

**Menarche** (first menstrual period) Interval to menarche - 2–2.5 yr but may be as long as 6 yr after thelarche.

Mean age of menarche - 12.75 yr. (13.5 yrs in rural girls)

**Puberty in Boys (9-14yr)**

Growth of the testes (>3 mL in volume or 2.5 cm in longest diameter)

Thinning of the scrotum

Pigmentation of the scrotum

Growth of the penis, seminal vesicles and prostrate

Pubic hair then appears

Appearance of axillary hair usually occurs in midpuberty, 2 yr after pubic hair.

In boys, unlike girls, acceleration of growth (5-15cm/yr in early adolescence but later drops) begins after puberty is well under way and is maximal at genital stage IV–V (typically between 13 and 14 yr of age).

In boys, the growth spurt occurs approximately 2 yr later than in girls, and growth may continue beyond 18 yr of age.

**Adrenarche**

Adrenal cortical androgens also play a role in pubertal maturation.

Serum levels of **dehydroepiandrosterone (DHEA) and its sulfate (DHEAS) begin to rise at approximately 6–8 yr of age, before any increase in LH or sex hormones and before the earliest physical changes of puberty are apparent.**

NB

DHEAS is the most abundant adrenal C-19 steroid in the blood, and its serum concentration remains fairly stable over 24 hr; **a single measurement of this hormone is commonly used as a marker of adrenal androgen secretion.**

Although adrenarche typically antedates the onset of gonadal activity (i.e., gonadarche) by a few years, the two processes do not seem to be causally related, because adrenarche and gonadarche are dissociated in conditions such as;

Central precocious puberty

Adrenocortical failure

**ENDOCRINOLOGY IN PUBERTY**

The levels of gonadal steroids and gonadotropins are low until the age of 6–8 yrs

This is mainly due to the negative feedback effect of estrogen to the hypothalamic pituitary system (Gonadostat).

The gonadostat remains very sensitive (6–15 times) to the negative feedback effect, even though the level of estradiol is very low (10 pg/ml) during that time.

As puberty approaches this negative feedback effect of estrogen is gradually lost.

This results in some significant changes in the endocrine function of the girl.

**Hypothalamopituitary gonadal axis**

The GnRH pulses from hypothalamus results in pulsatile gonadotropin secretion (first during the night then by the day time).

GnRH → FSH, LH → Estradiol

The tonic and episodic secretion of gonadotropins in prepubertal period is gradually changed to one of cyclic release in postpubertal period

..

**Adrenal glands (Adrenarche)**

increase their activity of sex steroid synthesis (androstenedione, DHA, DHAS) from about 7 years of age.

Increased sebum formation, pubic and axillary hair and change in voice are primarily due to adrenal androgen production**.**

**Gonadarche:**

Increased amplitude and frequency of GnRH → ↑ secretion of FSH and LH → ovarian follicular development → ↑ estrogen.

Gonadal estrogen is responsible for the development of uterus, vagina, vulva and also the breasts

**Menarche**

The onset of first menstruation in life is called menarche.

It may occur anywhere between 10 and 16 years, the peak time being 13 years**.**

The first period is usually anovular.

The ovulation may be irregular for a variable period following menarche and may take about 2 years for regular ovulation to occur.

The menses may be irregular to start with.

**ADOLESCENCE**

The period of life beginning with puberty and ending with completed growth and physical maturity.

Between the ages of **10 - 19 yr (WHO), children undergo rapid changes in;**

Phenotypic changes: - Body size & Body shape

Neuroendocrine changes - Hormones set the developmental agenda in conjunction with social structures designed to foster the transition from childhood to adulthood.

Physiology

Psychological functioning

Social functioning

NB: 10-24 yr - Young Adults

**Marshall - Tanner Classification of Sex Maturity Stages in Girls**

**(Tanner JM, Growth of Adolescence, 1962)**

**SMR = sexual maturity rating.**

|  |  |  |
| --- | --- | --- |
| SMR Stage | Pubic Hair | Breasts |
| 1 | Preadolescent | Preadolescent |
| 2 | Sparse, lightly pigmented, straight, medial border of labia | Breast and papilla  elevated as small mound;  areolar diameter  increased |
| 3 | Darker, beginning to curl, increased amount | Breast and areola enlarged  , no contour  separation |
| 4 | Coarse, curly, abundant but amount less than in adult | Areola and papilla form  secondary mound |
| 5 | Adult feminine triangle, spread to medial surface of thighs | Mature; nipple projects,  areola part of general  breast contour |

**Marshall - Tanner Classification of Sex Maturity Stages in Boys**

|  |  |  |  |
| --- | --- | --- | --- |
| SMR Stage | PUBIC HAIR | PENIS | TESTES |
| 1 | None | Preadolescent | Preadolescent |
| 2 | Scanty, long, slightly pigmented | Slight enlargement | Enlarged scrotum, pink texture altered |
| 3 | Darker, starts to curl, small amount | Longer | Larger |
| 4 | Resembles adult type, but less in quantity; coarse, curly | Larger; glans and  breadth increase in size | Larger, scrotum dark |
| 5 | Adult distribution, spread to medial surface of thighs | increase in size  Adult size | Adult size |

**Adolescence**

Developmental lines occur within three periods of adolescence;

**Early Adolescence – 10 – 13YRS**

**Middle Adolescence – 14-16yrs**

**Late Adolescence – 17-20 yrs and beyond**

|  |  |  |  |
| --- | --- | --- | --- |
| Variable | Early Adolescence | Middle Adolescence | Late Adolescence |
| Age (yr) | 10–13 | 14–16 | 17–20 and beyond |
| SMR\* | 1–2 | 3–5 | 5 |
| Somatic | Secondary sex characteristics; beginning of rapid growth; awkward | Height growth peaks; body shape and composition change; acne and odor; menarche; spermarche | Slower growth |
| Sexual | Sexual interest usually exceeds sexual activity | Sexual drive surges; experimentation; questions of sexual orientation | Consolidation of sexual identity |
| Cognitive and moral | Concrete operations; conventional morality | Emergence of abstract thought; questioning mores; self–centered | Idealism; absolutism |
| Self–concept | Preoccupation with changing body; self–conscious | Concern with attractiveness, increasing introspection | Relatively stable body image |
| Family | Bids for increased independence; ambivalence | Continued struggle for acceptance of greater autonomy | Practical independence; family remains secure base |
| Peers | Same–sex groups; conformity; cliques | Dating; peer groups less important | Intimacy; possibly commitment |
| Relationship to society | Middle–school adjustment | Gauging skills and opportunities | Career decisions (e.g., dropout, college, work) |

Bottom of Form

### Common Disorders of Puberty

•           Precocious puberty

•           Delayed puberty

•           Menstrual abnormalities (amenorrhea, menorrhagia, dysmenorrhea)

•          Others (infection, neoplasm, hirsutism, etc.)

**PRECOCIOUS PUBERTY**

The term precocious puberty is reserved for girls who exhibit any secondary sex characteristics before the age of 8 or menstruate before the age of 10.

Precocious puberty may be isosexual where the features are due to excess production of estrogen.

It may be heterosexual where features are due to excess production of androgen (from ovarian and adrenal neoplasm).

**FORMS OF PRECOCIOUS PUBERTY**

**GnRH dependent—80% (complete, central, isosexual or true)**

Constitutional—most common

Juvenile primary hypothyroidism

Intracranial lesions—trauma, tumor or infection

**Incomplete**

Premature thelarche

Premature puberche

Premature menarche

**CAUSES OF PRECOCIOUS PUBERTY**

**GnRH independent (precocious pseudopuberty or peripheral)**(Excess estrogen or androgen)

**Ovary**

Granulosa cell tumor

Theca cell tumor

Leydig cell tumor

Chorionic epithelioma

Androblastoma

McCune-Albright syndrome

**Adrenal**

Hyperplasia

Tumor

**Liver**

Hepatoblastoma

**Iatrogenic**

Estrogen or androgen intake

Precocious puberty in a girl aged  
2 years and 3 months

**DIAGNOSIS**

History

Basic investigations

X-ray, ct scan, MRI

Serum hCG, FSH, LH

Thyroid profile (TSH, T4)

Serum estradiol, testosterone, 17 OH progesterone, dehydroepiandrosterone (DHEA).

Electroencephalogram.

TREATMENT

**The goals are:**

 To reduce gonadotropin secretions.

 To suppress gonadal steroidogenesis or counteract the peripheral action of sex steroids.

 To decrease the growth rate to normal and slowing the skeletal maturation.

 To protect the girl from sex abuse.

DRUG MNX

**GnRH agonist therapy arrests the pubertal**precocity and growth velocity significantly.

The agonists suppress the premature activation of hypothalamopituitary axis due to down regulation and thereby diminished estrogen secretion.

**GnRH agonist therapy is the drug of choice in cases with GnRH dependent precocious puberty.**

GnRH agonist therapy suppresses FSH, lH secretion, reverses the ovarian cycle, establishes amenorrhea, causes regression of breast, pubic hair changes, and other secondary sexual characteristics.

This drug should be continued till the median age of puberty

**GnRH agonist**

•           Buserelin nasal spray 100 mg daily. It can slow down the process of skeletal maturation

•           **Medroxyprogesterone acetate—30 mg daily**orally or 100–200 mg. IM weekly to suppress gonadal steroids. It can suppress menstruation and breast development but cannot change the skeletal growth rate.

•           **Cyproterone acetate—It acts as a potent**progestogen, having agonist effects on progesterone receptors.

**Dose—70–100 mg/m2/day orally for 10 days starting**from 5th day of cycle.

**4. Danazol—It produces amenorrhea and arrest**breast development. But there is no effect on growth rate or skeletal maturation.

**2. DELAYED PUBERTY**

**Puberty is said to be delayed when the breast tissue and/or pubic hair have not appeared by 13–14 years or menarche appears as late as 16 years.**

The normal upper age limit of menarche is 15 years.

**CAUSES OF DELAYED PUBERTY**

•           Hypergonadotropic hypogonadism

Gonadal dysgenesis, 45 XO

Pure gonadal dysgenesis 46 XX, 46 XY

Ovarian failure 46 XX

2. Hypogonadotropic hypogonadism

Constitutional delay

Chronic illness, malnutrition

Primary hypothyroidism

Isolated gonadotropin defi ciency (Kallmann’s syndrome)

Intracranial lesions—tumors: craniopharyngioma, pituitary adenomas

3. Eugonadism

Anatomical causes

Müllerian agenesis

Imperforate hymen

Transverse vaginal septum

Androgen insensitivity syndrome

### Menstrual cycle

A periodic physiologic vaginal hemorrhage, occurring at approximately **28 ± 7 days interval (from the start of one menstrual period to the start of the next), and having its source from the shedding of uterine mucous membrane (menstruation); usually the bleeding is preceded by ovulation and predecidual changes in the endometrium.**

This may be teleologically regarded as periodic preparations for **fertilization and pregnancy.**

Menstruation is the visible manifestation of cyclic physiologic uterine bleeding due to shedding of the endometrium following invisible interplay of hormones mainly through hypothalamo-pituitaryovarian axis.

**For the menstruation to occur, the**axis must be actively coordinated, endometrium must be responsive to the ovarian hormones (estrogen and progesterone) and the outflow tract must be patent.

The first menstruation (**menarche**) occurs between 11–15 years with a mean of 13 years. It is more closely related to bone age than to chronological age

For the past couple of decades, the age of menarche is gradually declining with improvement of nutrition and environmental condition.

Physiologically, it is kept in abeyance due to pregnancy and lactation

Women have around **400 menstrual cycles**during the course of their lifetimes

Ultimately, it ceases between the ages 45–50 when **menopause** sets in

The duration of menstruation (menses) is about 4–5 days and the amount of blood loss is estimated to be 20 to 80mL with an average of 35mL.

Nearly 70% of total menstrual blood loss occurs in the first 2 days.

The menstrual discharge consists mainly of:

 dark altered blood,

mucus,

vaginal epithelial cells,

fragments of endometrium,

prostaglandins,

enzymes and bacteria.

**Prenatal follicular development**

During intrauterine fetal development, the ovary develops through **3 stages;**

**Genital ridge stage -**Sex cells can first be identified and begin as hypertrophy of the coelomic epithelium (future peritoneum) overlying the developing mesonephroi. Further growth of the ridges is dependent upon the arrival of germ cells.

**Indifferent stage -**Proliferation of germinal cells by mitosis and somatic cells

**Sexual differentiation stage -**Fundamental histologic differences between the ovary and testis are established

To maintain species-specific chromosome complement;

* the male gametes go through meiosis after puberty and continues throughout life owing to persistence of mitotically active “stem cells”, (spermatogonia)
* the female gametes undergo meiosis during fetal life and all stem cells are eliminated during birth when meiosis is suspended in the middle of the first meiotic division to resume shortly before ovulation in response to LH surge

The normal human menstrual cycle can be divided into two segments:

* the **ovarian cycle**and
* the **uterine cycle**, based on the organ under examination

**The ovarian cycle**

**Def:**is the cyclic hormonal changes and other series of changes that occur in the ovary to mature the immature follicle and recruit the oocyte.

It may be further divided into:

**Follicular phase**extends from the beginning of menstruation (day 1) to the onset of ovulation. The average length of the human follicular phase ranges from 10 to 14 days, and variability in this length is responsible for most variations in total cycle length.

**Ovulation.**

***luteal phase****(post ovulstory phase) extends from ovulation to the beginning of menstruation.*Unlike the follicular phase this phase is **most predictable and constant**(14 days) in length

Ovarian cycle

**the ovarian cycle consists of:**

Follicular phase:

Recruitment of groups of follicles

Selection of dominant follicle and its maturation.

Ovulation

Luteal phase:

Corpus luteum formation

Demise of the corpus luteum.

**Recruitment of groups of follicles (Preantral phase)**

The cohort of the growing follicles undergoes a process of development and differentiation which takes about **85 days and spreads over 3 ovarian cycles.**

It is not clear as to how many and which of the primordial follicles amidst several thousands are recruited for a particular cycle.

It is presumed that about 20 antral follicles (about 5–10 per ovary) proceed to develop in each cycle.

..

The initial recruitment and growth of primordial follicles are not under the control of any hormone.

After a certain stage (2–5 mm in size), the growth and differentiation of primordial follicles are under the control of FSH.

**Unless the follicles are rescued by FSH at this stage, they undergo atresia.**

..

With FSH, the oocyte is now surrounded by an acellular barrier of glycoprotein produced by the follicular cells and is called **zona pellucida.**

The flattened outer single layer pregranulosa cells become cuboidal and multilayered—now called **granulosa cells**

…

Then, there is appearance of channels (gap junctions) between the granulosa cells and the oocyte.

Through these gap junctions nutrition to the oocyte is maintained.

There is noticeable beginning of differentiation of the theca interna layer of ovarian stroma surrounding the follicle.

The granulosa cells now acquire FSH receptors.

**Antrum formation**

Then, there is accelerated growth of all the components of the follicles of the prentral phase.

The granulosa cells grow faster than the theca cells.

There is production of follicular fluid which is primarily an **ultrafiltrate** of blood from the vessels within theca interna.

The fluidfilled space is formed amidst the granulosa cells.

The spaces coalesce to form an **antrum**

**Dominant Follicle**

**As early as day 5–7, one of the follicles**out of so many becomes dominant and undergoes further maturation.

It seems probable that the one with **highest antral concentration of estrogen and lowest androgen** and whose granulosa cells contain the **maximum receptors for FSH**, becomes the dominant follicle.

The rest of the follicles become atretic by day 8

Further growth of dominant follicle

There is marked enlargement of the granulosa cells.

The granulosa cells surround the ovum to form **cumulus oophorus**which infact anchors the ovum to the wall of the follicle.

The cells adjacent to the ovum are arranged radially and is called **corona radiata**.

At this stage, **FSH induces LH receptors on the granulosa cells of the dominant follicle.**

**LH receptor**induction is essential for the mid-cycle LH surge to induce ovulation, luteinization of the granulosa cells to form corpus luteum and secretion of progesterone (two cell, two gonadotropin therapy)

Mature **Graafian follicle**

**The fully mature Graafian follicle**just prior to ovulation measures about 20 mm, and is composed of the following structures from outside inward:

•                Theca externa.

•                Theca interna.

•                Membrana granulosa (limitans).

•                Granulosa cell layer.

•                Discus proligerus in which the ovum is incorporated with cells arranged radially (corona radiata).

•                Antrum containing vesicular fluid.

NB

it takes 3 months for the follicle to grow and mature to ovulation—2 months to reach an antral stage measuring 1 mm; 2 weeks to reach 5 mm and another 2 weeks to reach 20mm before ovulation.

Hormonal changes during follicular phase of ovarian cycle…

At the start of the menstrual cycle, FSH levels begin to rise as the pituitary is released from the negative feedback effects of progesterone, oestrogen and inhibin.

Rising FSH levels rescue a cohort of follicles from atresia, and initiate **steroidogenesis.**

Under influence of FSH, a cavity forms around the ovum **(antrum formation).**

**NB. On steroidogenesis**

The basis of hormonal activity in pre-antral to pre-ovulatory follicles is described as the**'two cell, two gonadotrophin' hypothesis.**

Steroidogenesis is compartmentalized in the two cell types within the follicle: **the theca and granulosa cells.**

The two cell, two gonadotrophin hypothesis states that these cells are responsive to the gonadotrophins **LH and FSH respectively**.

Within the theca cells, **LH stimulates**the **production of androgens**from cholesterol.

Within the granulosa cells, **FSH stimulates**the conversion of thecally derived androgens to oestrogens (**aromatization)**.

In addition to its effects on aromatization, FSH is also responsible for the proliferation of granulosa cells.

Androgen production within the follicle **regulate** the development of the pre-antral follicle.

**Low levels of androgens**enhance aromatization and therefore increase oestrogen production.

In contrast, **high androgen levels inhibit aromatization**and produce follicular atresia.

A delicate balance of FSH and LH is required for early follicular development.

**The ideal**situation for the initial stages of follicular development is **low LH levels and high FSH levels**, as seen in the early menstrual cycle.

If LH levels are too high, theca cells produce large amounts of androgens, causing follicular atresia.

The selection of the dominant follicle is the result of complex signalling between the ovary and the pituitary.

Such a follicle has the most efficient aromatase activity and the **highest** concentration of FSH-induced LH **receptors.**

The dominant follicle therefore produces the greatest amount of **oestradiol and inhibin**.

Inhibin further amplifies LH-induced androgen synthesis, which is used as a substrate for oestradiol synthesis.

These features mean that the largest follicle therefore requires the **lowest levels of FSH (and LH)**for continued development.

At the time of follicular selection, FSH levels are declining in response to the negative-feedback effects of oestrogen.

The dominant follicle is therefore the only follicle that is capable of continued development in the face of falling FSH levels.

in-vitro fertilization (IVF) & multiple pregnancy

During in-vitro fertilization (IVF), the production of many ovulatory follicles is desired to harvest many oocytes.

with the administration of exogenous gonadotrophins, many follicles continue to develop and are released at ovulation, with an ensuing multiple gestation rate of around 30%

INHIBIN & ACTIVIN

Granulosa cell inhibin enhances LH -induced androgen synthesis.

The production of inhibin is a further mechanism by which FSH levels are reduced below a threshold at vlhich only the dominant follicle can respond, ensuring atresia of the remaining follicles.

Activin augments pituitary FSH secretion and increases FSH binding to granulosa cells.

**Ovulation**

The dominant follicle, shortly before ovulation reaches the surface of the ovary.

The cumulus becomes detached from the wall, so that the **ovum with the surrounding cells (corona radiata)** floats freely in the liquor folliculi.

The oocyte completes the **first meiotic division**with extrusion of the first polar body which is pushed away.

The follicular wall near the ovarian surface becomes thinner.

**Hormonal changes at ovulation**

As the dominant follicle develops further, follicular **oestrogen production increases**.

Eventually the production of oestrogen is sufficient for it to reach the threshold required to exert a **positive-feedback** effect on pituitary **LH secretion**

LH levels increase, at first quite slowly (day 8 to day 12 of the menstrual cycle) and then more rapidly (day 12 onwards).

Hormonal changes at ovulation..

During this time, LH induces **luteinization** of granulosa cells in the dominant follicle, so that progesterone is produced.

Progesterone further amplifies the positive-feedback effect of oestrogen on pituitary LH secretion, leading to a surge of LH.

Ovulation occurs 36 hours after the onset of the LH surge.

**Hormonal changes at ovulation…**

In addition to the rise in LH, FSH and oestrogen that occurs around ovulation, a rise in serum androgen levels also occurs.

These **androgens** are derived from the stimulatory effect of LH on theca cells, particularly those of the non-dominant follicle.

This rise in androgens may have an important physiological effect in the **stimulation of libido**, ensuring that sexual activity is likely to occur at the time of ovulation, when the woman is at her most fertile and **enhance the process of atresia**of the small follicles.

**Hormonal changes at ovulation…**

Prior to the release of the oocyte at the time of ovulation, the LH surge stimulates the resumption of **meiosis**, a process which is completed after the sperm enters the egg (fertilization)

Hormonal changes at ovulation…

Additionally, the LH surge stimulates increased follicular leukocytes eg macrophage chemotactic protein-1 (MCP-I), interleukin 8 (IL-8) & neutrophils into the pre-ovulatory follicle.

Once activated, these leukocytes secrete mediators which cause the follicle wall to break down, releasing the oocyte at ovulation.

**LUTEAL PHASE**

Following ovulation, the follicle is changed to **corpus luteum**.

The ovum is picked up into the fallopian tube and undergoes either degeneration or further maturation, if fertilization occurs.

**Menstruation is unrelated to ovulation**and**anovular menstruation**is quite common during adolescence, following childbirth and in women approaching menopause.

**Corpus Luteum**

**Stage of Proliferation**

The opening through which the ovum escapes soon becomes plugged with fibrin.

The granulosa cells undergo hypertrophy without multiplication.

The cells become larger, polyhedral with pale vesicular nuclei and frothy cytoplasm.

The cells are called **granulosa lutein cells**.

The color of thecorpus luteum at this stage is **greyish yellow**due to presence of lipids

**Corpus luteum..**

**Stage of Vascularization**

Within 24 hours of rupture of the follicle, small capillaries grow into granulosa layer towards the lumen accompanied by lymphatics and fibroblasts.

Extensive vascularization within the corpus luteum ensures that the granulosa cells have a rich blood supply providing the precursors for steroidogenesis.

**Corpus luteum..**

**Stage of Maturation**

By 4th day, the luteal cells have attained the maximum size.

Approximately about 7–8 days following ovulation, the corpus luteum attains a size of about 1–2 cm and reaches its secretory peak.

The lutein cells become greatly enlarged and develop lipid inclusion, giving the cells a distinctive yellowish color. Cell contain a **yellow pigment**called **lutein**

**Corpus luteum**

**Stage of Regression**

On the day 22–23 of cycle, retrogression starts.

The first evidence of degeneration is appearance of vacuolation in the cells.

The lutein cells atrophy and the corpus luteum becomes **corpus albicans**.

Regression of corpus luteum is due to withdrawal of tonic LH support

If, however, fertilization occurs in the particular cycle, regression fails to occur, instead it is converted into **corpus luteum of pregnancy**.

**Hormonal changes in luteal phase**

is characterized by the production of progesterone from the corpus luteum within the ovary.

The production of progesterone from the corpus luteum is dependent on continued pituitary LH secretion.

However, serum levels of progesterone are such that LH and FSH production is relatively suppressed by inhibin.

The **low levels of gonadotrophins**mean that the initiation of **new follicular growth is inhibited**for the duration of the luteal phase.

**Hormonal changes in luteal phase**

In the absence of pregnancy and the production of human chorionic gonadotrophin (hCG) from the implanting embryo, the corpus luteum regresses at the end of the luteal phase, a process known as **luteolysis.**

As the corpus luteum dies, **oestrogen, progesterone and inhibin** levels decline.

The pituitary is released from the **negative-feedback** effects of these hormones, and gonadotrophins, particularly FSH, **start to rise**.

A cohort of follicles that happen to be at the pre-antral phase is rescued from atresia and a further menstrual cycle is initiated.

**Corpus Luteum of Pregnancy**

There is a surge of hyperplasia of all the layers between 23rd to 28th day due to chorionic gonadotropin.

**hCG, like LH will stimulate**the corpus luteum to secrete progesterone.

The growth reaches its peak at about 8th week when it measures about 2–3 cm.

Regression occurs following low levels of chorionic gonadotropin and the degenerative changes take place most frequently at about 6 months of gestation.

**Corpus luteum secretions**

predominantly progesterone is secreted by the corpus luteum to support the endometrium of the luteal phase.

There is also secretion of estrogen, inhibin and relaxin.

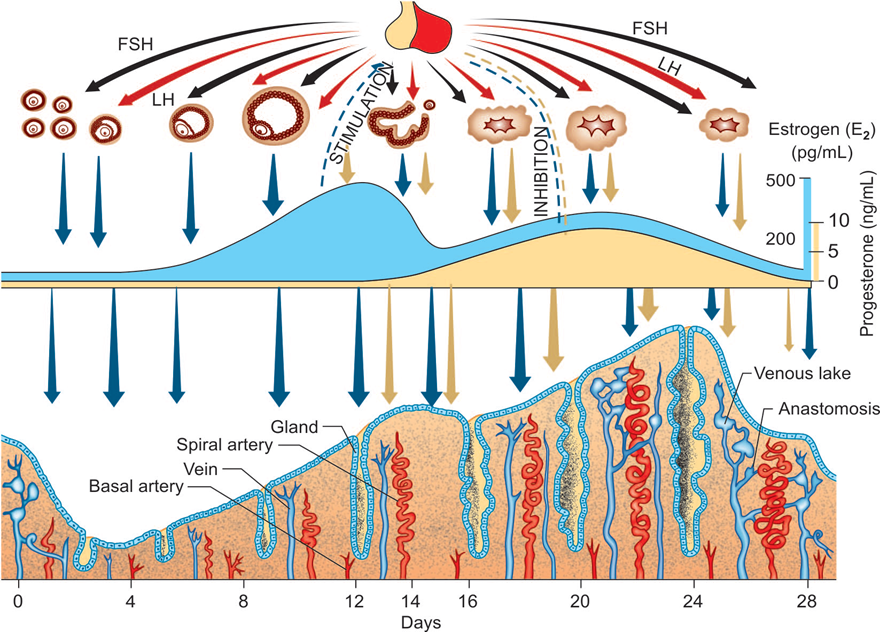
Progesterone along with estrogen from corpus luteum maintain the growth of the fertilized ovum.

This is essential till the luteal function is taken over by the placenta.

This turn over of function from corpus luteum of pregnancy to placenta is called **luteal-placental shift.**

**This transition period continues from seven weeks to ten weeks.**

**UTERINE CYCLE**



ENDOMETRIUM

The endometrium is the lining epithelium of the uterine cavity above the level of internal os.

It consists of **surface epithelium, glands, stroma and blood vessels.**

Two distinct divisions are established:**—**

**basal zone (stratum basalis) and**

**the superficial functional zone.**

**BASAL LAYER**

It is about one-third of the total depth of the endometrium and lies in contact with the myometrium.

The base of the endometrial glands extends into the layer.

The zone is uninfluenced by hormone and as such, no cyclic changes are observed.

 After shedding of the superficial part during menstruation, the regeneration of all the components occurs from this zone. **It measures about 1 mm.**

**FUNCTIONAL LAYER**

This zone is under the influence of fluctuating cyclic ovarian hormones, estrogen and progesterone.

The changes in different components during an ovulatory cycle has been traditionally divided into four stages:

Regenerative phase.

Proliferative phase.

Secretory phase.

Menstruation

**STAGE OF REGENERATION**

starts even before the menstruation ceases and is completed 2–3 days after the end of menstruation.

New blood vessels grow from the stumps of the old one.

The glands and the stromal cells are regenerated from the remnants left in the basal zone.

The glands are lined by the cubical epithelia.

**The thickness averages 2 mm.**

**STAGE OF PROLIFERATION**

extends from 5th or 6th day to 14th day (till ovulation).

The proliferative changes occurs due to rise in level of ovarian estrogens.

The glands become tubular and lie perpendicular to the surface.

The epithelium becomes columnar.

Unbranched spiral vessels with capillary congestion

**The thickness measures about 3–4 mm.**

**SECRETORY PHASE**

The changes of the components are due to the combined effects of estrogen and progesterone liberated from the corpus luteum after ovulation.

The endometrium contains receptors for progesterone which are induced by estrogen.

**Thus, the progesterone can only act on the endometrium previously primed by estrogen.**

It begins on day 15 and ceases 5–6 days prior to menstruation.

**The surface epithelium becomes more columnar**and ciliated at places.

The glands show predominant changes.

**The glands increase in size.**

**The lining**epithelium become taller.

There is appearance of vacuoles due to secretion of glycogen between the nuclei and the basement membrane.

**The glands become corkscrew-shaped. The blood vessels undergo marked spiraling.**

**The thickness of the endometrium reaches its highest (6–8 mm)**

The endometrial growth ceases 5–6 days prior to menstruation (22nd or 23rd day of cycle) in an infertile cycle.

The subepithelial capillaries and the spiral vessels are engorged.

**MENSTRUAL PHASE**

It is essentially degeneration and casting off an endometrium prepared for a pregnancy.

Regression of corpus luteum with fall in the level of estrogen and progesterone is an invariable preceding feature

Thes marked **spiralling** of the arteries and the **withdrawal of hormones estrogen and progesterone** causes intense spasm of the spiral arterioles at the basal part.

These two lead to **stasis and tissue anoxemia**.

There are evidences of infiltration of leucocytes and monocytes in the stroma.

Stasis of blood and spasm of the arterioles lead to damage of the arteriolar walls.

There is enzymatic autodigestion of the functional zone.

The bleeding occurs from the broken arteries, veins and capillaries and also from the stromal hematoma, with the superficial functional layer being shed into the uterine cavity

**The menstrual flow stops as a result of combined effect**of prolonged vasoconstriction, myometrial contraction and local aggregation of platelets with deposition of fibrin around them.

**ANOVULAR MENSTRUATION**

In an anovulatory cycle, the follicles grow without any selection of dominant follicle. The estrogen is secreted in increasing amount.

There may be imbalance between estrogen and FSH or because of temporary unresponsiveness of the hypothalamus to the rising estrogen, GnRH is suppressed → no ovulation.

The net effect is unopposed secretion of estrogen till the follicles exist.

The endometrium remains in either proliferative or at times hyperplastic state.

When the estrogen level falls, there is asynchronus shedding of the endometrium and menstruation.

The bleeding may be heavy or prolonged and irregular

This type of bleeding is mostly found during adolescence, following childbirth and abortion and in premenopausal period.

**ARTIFICIAL POSTPONEMENT**

The hormones used for deferment of the period are—combined oral pill, 2 tablets daily or progestogen such as norethisterone 5 mg twice daily.

The drug should be taken at least 3–6 days before the expected date of the period and continued until the crisis is over.

The period is expected 2–3 days after the drug is suspended.

**The normal menstrual cycle-clinical features**

normal menstrual cycle is 28 days +/- 7days

duration of menstrual flow is 3-7 days.

Menstrual cycles are longest immediately after puberty and in the 5 years leading up to the menopause, corresponding to the peak incidence of anovulatory cycles.

The length of the menstrual cycle is determined by the length of the follicular phase.

Once ovulation occurs, **luteal phase length is fairly fixed at 14 days** in almost all women.

Clinical features

The amount of menstrual flow peaks on the first or second day of menstruation.

The normal volume of menstrual loss is **35 mL**per month.

A menstrual loss of greater than **80 mL**is considered to be excessive - this level is rather arbitrary and corresponds to the threshold at which iron deficiency anaemia may ensue unless treated.

**New developments**

**IVF** - exogenous gonadotrophins are administered to stimulate follicular growth within the ovary

**GnRH antagonists**

**GnRH agonists**

### Menstrual Disorders

* •           Dysmenorrhea, mittelschmerz’s syndrome and Premenstrual syndrome

•           Abnormal uterine bleeding:

•           Amenorrhea and Oligomenorrhea

•           Polycystic ovarian syndrome

•           Dysfunctional uterine bleeding

•           Postmenopausal bleeding

**1. MITTELSCHMERZ’S SYNDROME (Ovular Pain)**

* Ovular pain is not an infrequent complaint.
* It appears  in the midmenstrual period.
* The pain is usually situated in the hypogastrium or in either iliac fossa.
* The exact cause is not known.

**The probable factors are:**

* •                  Increased tension of the Graafian follicle just prior to rupture,
* •                  Peritoneal irritation by the follicular fluid following ovulation and
* •                  Contraction of the tubes and uterus

**2. DYSMENORRHEA**

* Dysmenorrhea literally means painful menstruation.
* But a more realistic and practical definition includes cases of **painful menstruation of sufficient magnitude so as to incapacitate day-to-day activities.**
* **Prevalence:**Dysmenorrhea is a very common complaint, experienced by 45-95 per cent of women of reproductive age.

**Types:**

**Primary  
Secondary**

**PRIMARY DYSMENORRHEA (Spasmodic)**

* The primary dysmenorrhea is one where there is no identifiable pelvic pathology.
* The incidence of primary dysmenorrhea of sufficient magnitude with incapacitation is about 15–20%.
* The risk factors for primary dysmenorrhoea include:
* duration of menstrual flow of > 5 days,
* younger than normal age at menarche,
* cigarette smoking.
* There is some evidence to support the assertion that dysmenorrhoea improves after childbirth, and it also appears to decline with increasing age.
* The severity of pain usually lasts for few hours, may extend to 24 hours but seldom persists beyond 48 hours.

**SECONDARY DYSMENORRHEA  
(Congestive)**

* is normally considered to be menstruation — associated pain occurring in the presence of pelvic pathology.
* The pain may be related to increasing tension in the pelvic tissues due to pre-menstrual pelvic congestion or increased vascularity in the pelvic organs.

**Common causes of secondary dysmenorrhea:**

* chronic pelvic infection,
* Pelvic endometriosis,
* adenomyosis
* pelvic adhesions / Asherman's syndrome
* Uterine fibroid,
* endometrial polyp,
* IUCD *in utero*
* *Pelvic*congestion.
* (rarely) cervical stenosis.
* Obstruction due to mullerian malformations
* NB: some definitions
  + ***Endometriosis:***Endometrial stroma & glands remote of the uterine cavity, responsive to ovarian hormone variations with fibrosis of surrounding tissues
  + *Sites;*Uterine ligaments, bowel, urinary tract, pelvic peritonuem, ovaries, abdominal wall, perineum & vagina.
  + ***Adenomyosis/endometriosis interna:***Extension of endometrial glands and stroma into the myometrium

**Clinical features of secondary dysmenorhea**

* The pain is dull, situated in the back and in front without any radiation.
* It usually appears 3–5 days prior to the period and relieves with the start of bleeding.
* The onset and duration of pain depends on the pathology producing the pain.
* There is no systemic discomfort unlike primary dysmenorrhea.
* The patients may have got some discomfort even in between periods

**Treatment of dysmenorhea**

* NSAIDs, such as naproxen, ibuprofen and mefenamic acid, are reasonably effective. Aspirin,  Mefenamic acid (500mg TID)
* Oral contraceptives are widely used but, surprisingly, there is little evidence.
* Surgical treatments aimed at interrupting the nerve pathways from the uterus have been employed
* Treat underlying conditions
* Conservative mgt; taking a hot bath, using a hot water bottle and relaxing in bed

**3. PREMENSTRUAL SYNDROME (PMS) (Syn : Premenstrual Tension)**

* is the occurrence of cyclical psychoneuroendocrine disorder of unknown etiology, often noticed just prior to menstruation (in luteal phase-premenstrual) and resolve by the time menstruation ceases.
* Occur during the last 7–10 days of the menstrual cycle.

**It should fulfil the following criteria:**

* Not related to any organic lesion.
* cyclic symptoms occurring only during luteal phase
* Symptoms must be severe enough to disturb the lifestyle of the woman or she requires medical help.
* Symptom-free period during rest of the cycle.
* symptoms relieved with onset of menses
* symptoms present for at least 3 cycles

**Pathophysiology:**

The exact cause is not known but the **following hypotheses are postulated :**

(a) Alteration in the level of estrogen and progesterone starting from the midluteal phase. Either there is altered estrogen : progesterone ratio or diminished progesterone level.

(b) Neuroendocrine factors :

* **Serotonin is an important neurotransmitter in**the CNS. During the luteal phase, decreased synthesis of serotonin is observed in women suffering from PMS.
* **Endorphins: The symptom complex of PMS**is thought to be due to the withdrawal of endorphins (neurotransmitters) from CNS during the luteal phase.
* γ**-aminobutyric acid (GABA) suppresses the**anxiety level in the brain. Medications that are GABA agonist, are effective.

(c) Psychological and psychosocial factors may be involved to produce behavioral changes.

**Unfortunately, nothing is conclusive**

CLINICAL FEATURES

* Bloating
* cyclical weight gain
* Mastalgia
* abdominal cramps
* Fatigue
* Headache
* depression
* irritability.

**TREATMENT OF PMS**

* As the etiology is multifactorial and too often obscure, various drugs are used either on speculation or empirically with varying degrees of success.
* **Life style modification and congnitive behavior therapy are important steps.**
* **Nonpharmacological:**
  + **Assurance, Yoga,**Stress management, Diet manipulation.
  + Avoidance of salt, caffeine and alcohol specially in second half of cycle improves the symptoms.

**Nonhormonal :**

* + - Tranquilizers or antidepressant drugs, may be of help logically.
    - Pyridoxine 100 mg Bid  is helpful by correcting tryptophan metabolism - depression.
    - D**iuretics** - Frusemide 20 mg OD for consecutive 5 days a week reduces fluid retention.
    - **Anxiolytic**agents are found to be helpful to women having persistent anxiety. Alprazolam 0.25 mg, BID) is given during the luteal phase of the cycle.
    - **Selective Serotonin Reuptake Inhibitors (SSRI**) (eg Fluoxetine 20mg) **and Noradrenaline Reuptake Inhibitors (SNRI)**are found to be very effective.

**Hormones:**

* + **Oral contraceptive pills:**The idea is to suppress ovulation and to maintain an uniform hormonal milieu. The therapy is to be continued for 3–6 cycles.
  + **Progesterone is not effective in treating PMS.**
  + **Spironolactone:**It is a potassium sparing diuretic. It has anti-mineralocorticoid and anti-androgenic effects. It is given in the luteal phase (25–200 mg/day).
  + **Bromocriptine: 2.5 mg daily or twice daily**may be helpful, at least to relieve the breast complaints.
  + **Oophorectomy -**primary PMS with recurrence of symptoms and approaching to menopause, hysterectomy with bilateral oophorectomy is a last resort
  + **Suppression of ovarian cycle**
    - * Medical oopherectomy - GnRH agonist for 6 months
      * **Danazol 200 mg daily**is to be adjusted so as to produce amenorrhea

### Menstrual Disorders: Abnormal Uterine Bleeding

Abnormal Uterine Bleeding

**Any uterine bleeding outside the normal volume, duration, regularity or frequency is considered abnormalnuterine bleeding (AUB).**

Description of alteration in the normal pattern of menstrual flow

 Forms;

Excessive flow

Prolonged flow

Intermenstrual bleeding

Types;

          -         hypermenorrhoea                 -        metrorrhagia

          -         polymenorrhoea               -         amenorrhoea

          -         menometrorrhagia                -        menorrhagia

          -         oligomenorrhoea

**1. MENORRHAGIA  
(Syn : Hypermenorrhea)**

Menorrhagia is defined as cyclic bleeding at normal intervals; the bleeding is either excessive in amount (> 80 mL) or duration (>7 days) or both.

The term **menotaxis**is often used to denote prolonged bleeding.

**Classification of menorrhagia**

Menorrhagia can be classified as:

idiopathic, where no organic pathology can be found: also known as dysfunctional uterine bleeding-(DUB).

secondary to an organic cause, such as fibroids.

**Causes:**

**1. Organic:**

a**. Pelvic causes:**

* Fibroid uterus
* Adenomyosis
* Pelvic endometriosis
* IUCD inutero
* Chronic tubo-ovarian mass
* Tubercular endometritis (early cases)
* Retroverted uterus—due to congestion
* Granulosa cell tumor of the ovary

***b. Systemic:***

***Liver dysfunction—f****ailure to conjugate and*thereby inactivates the estrogens.

* Congestive cardiac failure.
*  Severe hypertension

***c. Endocrinal***

* Hypothyroidism.
* Hyperthyroidism.

***d. Hematological***

*  Idiopathic thrombocytopenic purpura.
*  Leukemia.  von Willebrand’s disease.
*  Platelet deficiency.

2. **Functional**

* Due to disturbed hypothalamo-pituitary-ovarianendometrial axis.

**COMMON CAUSES OF MENORRHAGIA**

* Dysfunctional uterine bleeding
* Fibroid uterus
* Adenomyosis
* Chronic tubo-ovarian mass

**Diagnosis & treatment**

* number of towels and tampons used per day is useful
* irregular, intermenstrual or postcoital bleeding, a sudden change in symptoms, dyspareunia, pelvic pain or premenstrual pain, and excessive bleeding from other sites or **in other situations (e.g. after tooth**extraction).
* Long duration of flow, passage of big clots, use of increased number of thick sanitary pads, pallor, and low level of hemoglobin give an idea about the correct diagnosis and magnitude of menorrhagia.

**Treatment**: The definitive treatment is appropriate to the cause for menorrhagia**.**

Some Definitive Treatment

*Drugs that are compatible with ongoing attempts at conception*

* Mefenamic acid and other non-steroidal, anti-inflammatory drugs (NSAIDs)
* Tranexamic acid
* *Drugs that are incompatible with ongoing attempts at conception but not licensed for use as contraceptives*
* Danazol
* *used as contraceptives that are effective in the treatment of menorrhagia*
* Combined oral contraceptive pill
* levonorgestrel intrauterine system.
* *Second-line drugs with few advantages over the forgoing, and whose side effects limit long-term use*
  + - * Danazol
      * Gestrinone
      * Gonadotrophin-releasing hormone analogues
      * Medical and surgical treatments that are ***not*effective**in the treatment of menorrhagia
      * Ethamsylate
      * Luteal phase progestogens
      * Uterine curettage

**Surgical treatments for menorrhagia**

* **ablation of the endometrial lining**of the uterus to sufficient depth prevents regeneration of the endometrium.
* ***Hysterectomy***

**2. POLYMENORRHEA (SYN : EPIMENORRHEA)**

* is defined as cyclic bleeding where the cycle is reduced to an arbitrary limit of less than 21 days and remains constant at that frequency.
* If the frequent cycle is associated with excessive and or prolonged bleeding, it is called **epimenorrhagia**.

**Causes & treatment**

**Dysfunctional:**

* It is seen predominantly during adolescence, preceding menopause and following delivery and abortion.
* Hyperstimulation of the ovary by the pituitary hormones may be the responsible factor.

**Ovarian hyperemia**as in PID or ovarian endometriosis.

**Treatment:**

**Persistent dysfunctional type is to be treated**by hormone as in DUB

**3. METRORRHAGIA**

* is defined as irregular, acyclic bleeding from the uterus.
* Amount of bleeding is variable.
* While metrorrhagia strictly concerns uterine bleeding but in clinical practice, the bleeding from any part of the genital tract is included under the heading.
* Then again, irregular bleeding in the form of **contact bleeding or** **intermenstrual bleeding**in an otherwise normal cycle is also included in metrorrhagia.
* In fact, it is mostly related to surface lesion in the uterus.

**Menometrorrhagia**is the term applied when the bleeding is so irregular and excessive that the menses (periods) cannot be identified at all.

**CAUSES OF ACYCLIC BLEEDING**

* DUB—usually during adolescence, following childbirth and abortion and preceding menopause
* Submucous fibroid
* Uterine polyp
* Carcinoma cervix and endometrial carcinoma

**CAUSES OF CONTACT BLEEDING**

* Carcinoma cervix
* Mucos polyp of cervix
* Vascular ectopy of the cervix especially during pregnancy, pill use cervix
* Infections—chlamydial or tubercular cervicitis
* Cervical endometriosis

**CAUSES OF INTERMENSTRUAL BLEEDING**

* Apart from the causes of contact bleeding, other causes are:
*  Urethral caruncle 
* Ovular bleeding
*  Breakthrough bleeding in pill use
*  IUCD in utero 

**Treatment of metrorhagia**

Treatment is directed to the underlying pathology.

**Malignancy is to be excluded prior to any definitive treatment**ecubitus ulcer

**4. OLIGOMENORRHEA**

* **Definition: Menstrual bleeding occurring more than 35 days apart and which remains constant at**that frequency is called oligomenorrhea.

**COMMON CAUSES OF OLIGOMENORRHEA**

*  Age-related—during adolescence and preceeding menopause
* Weight-related—obesity
* Stress and exercise related
* Endocrine disorders—PCOS (commonest), hyperprolactinemia, hyperthyroidism
* Androgen producing tumors—ovarian, adrenal
* Tubercular endometritis—late cases
* **Drugs:**
* Phenothiazines
* Cimetidine
* Methyldopa

**5. AMENORRHOEA**

* absence of menstruation.
* It may be classified as either primary or secondary.
* There are, physiological situations in which amenorrhoea is normal, namely:
* pregnancy,
* lactation and
* prior to the onset of puberty.

**Classification of amenorrhoea**

* Primary amenorrhoea: condition in which girls fail to develop secondary sexual characteristics by 14 years of age or fail to menstruate by 16 years of age.
* Secondary amenorrhoea: describes the cessation of menstruation for more than 6 months in a normal female of reproductive age that is not due to pregnancy.

**Causes of amenorrhoea**

* Reproductive outflow tract disorders
* Asherman 's syndrome
* Mullerian agenesis
* Transverse vaginal septum
* Imperforate hymen
* Testicular feminization syndrome
* Ovarian disorders
* Anovulation, e.g. polycystic ovarian synd rome (peaS)
* Gonadal dysgenesis, e.g. Turner's syndrome
* Premature ovarian failure
* Resistant ovary syndrome
* Pituitary disorders
* Adenomas such as prolactinoma
* Pituitary necrosis, e.g. Sheehan's syndrome
* Hypothalamic malfunctions
* Resulting from excessive exercise
* Resulting from weight loss/anorexia nervosa
* Resulting from stress
* Craniopharyngioma
* Kallman 's syndrome

**Diagnosis & treatment**

* Detailed history and examination
* Investigations
* *Initial hormone tests:*
  + Pregnancy test,
  + Prolactin,
  + Thyroid function,
  + LH and FSH,
  + Testosterone – androgen producing tumours
* Treat the cause.

**6. HYPOMENORRHEA**

**Definition:**When the menstrual bleeding is unduly scanty and lasts for less than 2 days, it is called hypomenorrhea.

**Causes**

* The causes may be:
* **local** (uterine synechiae or endometrial tuberculosis),
* **endocrinal** (use of oral contraceptives, thyroid dysfunction, and premenopausal period), or systemic (malnutrition).

**7. DYSFUNCTIONAL UTERINE BLEEDING (DUB)**

* **DUB** is defined as a state of abnormal uterine bleeding without any clinically detectable organic, systemic, and iatrogenic cause (Pelvic pathology, e.g. tumor, inflammation or pregnancy is excluded).
* **NB: Heavy menstrual bleeding (HMB)**is defined as a bleeding that interferes with woman's physical, emotional, social and maternal quality of life.

DUB

* The bleeding may be abnormal in frequency, amount, or duration or combination of any three.
* As **the diagnosis is based with the exclusion of ‘organic lesion’,***Currently DUB is defined as a state of abnormal uterine bleeding following anovulation due to dysfunction of hypothalamo-pituitary-ovarian axis (endocrine origin).*

Pathophysiology

* The endometrial abnormalities may be *primary or secondary to****incoordination in the hypothalamopituitary-*ovarian axis.**
* It is thus *more prevalent in extremes of reproductive period—adolescence and*premenopause or following childbirth and abortion.
* The abnormal bleeding may be associated with or without ovulation and accordingly grouped into :
  + - * **Ovular bleeding**
      * **Anovular bleeding**

**OVULAR BLEEDING**

* **Polymenorrhea or polymenorrhagia:**
* usually occurs following childbirth and abortion, during adolescence and premenopausal period, and in pelvic inflammatory disease.
* The follicular development is speeded up with resulting shortening of the follicular phase.
* This is probably due to hyperstimulation of the follicular growth by FSH.
* Rarely, the luteal phase may be shortened due to premature lysis of the corpus luteum.
* Sometimes, it is related to stress induced stimulation.
* **Oligomenorrhea:**
* Primary ovular oligomenorrhea is rare.
* Common in adolescence and premenopause.
* The disturbance may be due to ovarian unresponsiveness to FSH or secondary to pituitary dysfunction.
* There is undue prolongation of the proliferative phase with normal secretory phase.

**ANOVULAR BLEEDING**

* **Menorrhagia**
* Anovular bleeding is usually excessive.
* In the absence of growth limiting progesterone due to anovulation, the endometrial growth is under the influence of estrogen throughout the cycle.
* There is inadequate structural stromal support and the endometrium remains fragile.
* Thus, with the withdrawal of estrogen due to negative feedback action of FSH, the endometrial shedding continues for a longer period in asynchronous sequences because of lack of compactness
* **Cystic glandular hyperplasia**
* This type of abnormal bleeding is usually met in premenopausal women
* There is slow increase in secretion of estrogen but no negative feedback inhibition of FSH.
* The net effect is gradual rise in the level of estrogen with concomittant phase of amenorrhea for about 6–8 weeks.
* As there is no ovulation, the endometrium is under the influence of estrogen without being opposed by growth limiting progesterone for a prolonged period.

**diagnosis**

* History, physical examination & Investigations aim at:
* To confirm the menstrual abnormality as stated by the patient.
* To exclude the systemic, iatrogenic, and ‘organic’ pelvic pathology.
* To identify the possible etiology of DUB.
* To work out the definite therapy protocol.

**MANAGEMENT**

* Because of diverse etiopathology of DUB in different phases of woman’s life, the management protocols have been grouped accordingly.
  +  Pubertal and adolescent menorrhagia < 20 years.
  +  Reproductive period (20–40 years).
  +  Premenopausal (> 40 years).
  +  Postmenopausal.

Management

**General**

* Rest is advised during bleeding phase.
* Assurance and sympathetic handling are helpful particularly in adolescents.
* Anemia should be corrected energetically by diet, hematinics, and even by blood transfusion.
* Clinically evident systemic or endocrinal abnormalities should be investigated and treated accordingly.

**MEDICAL**

* potent **orally active progestins, are the mainstay in the management of DUB in all age groups.**
* **Mechanism of antiestrogenic action of progestins are:**
  + •                It stimulates the enzyme (17-β-hydroxy steroid dehydrogenase) that converts estradiol to estrone (less potent).
  + •                Inhibits induction of estrogen receptor.
  + •                It has antimitotic effect on the endometrium.
* While isolated progestins therapy is highly effective in anovular DUB, in ovular DUB combined preparations of progestogen and estrogen (combined oral pills) are effective.
* **To stop bleeding and regulate the cycle :**Norethisterone preparations (5 mg tab) are used thrice daily till bleeding stops, which it usually does by 3–7 days.

**Cyclic Therapy:**

* *In ovular bleeding,***Any low dose combined oral pills** are effective when given 5th to 25th day of cycle for 3 consecutive cycles. It causes endometrial atrophy. It is more effective compared to progesterone therapy as it suppress the hypothalamopituitary axis more effectively. Normal menstruation is expected to resume with restoration of normally functionating pituitary–ovarian-endometrial axis
* *In anovular bleeding: Cyclic progestogen preparation*of medroxyprogesterone acetate (MPA) 10 mg or norethisterone 5 mg is used from 5th to 25th day of cycle for 3 cycles

**NON-HORMONAL MANAGEMENT**

* **Anti-fibrinolytic agents**- Tranexamic acid
* **Prostaglandin synthetase**inhibitors: eg Mefenamic acid 150-600mg is much effective in women aged more than 35 years and in cases of ovulatory DUB during bleeding phase.
* **Desmopressin: It is a synthetic analogue of**arginine-vasopressin. indicated in von Willebrand’s disease and factor VIII deficiency. It is given IV (0.3 μg/kg) or intranasally.

**SURGICAL MANAGEMENT OF DUB**

* Uterine curettage
* Endometrial ablation/resection
* Hysterectomy

**8. POLYCYSTIC OVARIAN SYNDROME**

* is a syndrome of ovarian dysfunction along with the cardinal features of hyperandrogenism and polycystic ovary morphology.
* PCOS remains a syndrome, and as such no single diagnostic criterion (such as hyperandrogenism or polycystic ovary) is sufficient for clinical diagnosis.
* Its clinical manifestations may include:
  + - * menstrual irregularities,
      * signs of androgen excess
      * obesity.
      * Insulin resistance
      * elevated serum LH levels.
      * PCOS is associated with an increased risk of *type 2 diabetes and cardiovascular events.*

Prevalence:

* affects around 5-10% of women of reproductive age.
* The prevalence of polycystic ovaries seen on ultrasound is much higher - around 25%.

**Aetiology:**

* remains unclear but is self-perpetuating once starts.
* Women with this syndrome have increased ovarian androgen due partly to disordered ovarian cytochrome P450 activity and partly to increased LH stimulation.
* Additionally, increasing evidence suggests a role for (peripheral) insulin resistance in the pathophysiology, with the resulting hyperinsulinaemia also promoting ovarian androgen production.

**Clinical features**

* **Oligomenorrhoea/amenorrhoe**a: this occurs in up to 65-75% and is predominantly related to chronic anovulation.
* **Hirsutism**: this occurs in 30-70%.
* **Subfertility**: up to 75% of women with PCOS who try to conceive have difficulty doing so.
* **Obesity**: at least 40% are clinically obese.
* **Recurrent miscarriage**: is seen in 50-60% of women with more than three early pregnancy losses.
* **Acanthosis nigricans**: areas of increased skin pigmentation that are velvety in texture and occur in the axillae and other flexures occur in around 2% of women with PCOS.

**Diagnosis**

* Elevated testosterone levels.= testosterone & androstenedione
* Decreased sex hormone binding globulin (SHBG) levels.
* Elevated LH levels.
* •          Elevated LH:FSH ratio. Typically LH:FSH > 2:3.1
* Increased fasting insulin levels.
* Free testosterone is higher than normal, since SHBG levels are low. Testosterone levels of > 5 *nmol/L should prompt a search for an androgensecreting*tumour.
* Transvaginal U/S:
  + - ≥ 10 peripheral cysts btn 2-8 mm diameter (‘string of pearls’ sign)
    - Increased ovarian stromal volume to > 8cm3

**Management of PCOS**

Aim of Rx depends on main complaint

* Weight loss
* Cyclic progestagens or limited use of COC (menstrual disturbance)
* Anti-androgens; cyproterone acetate, spironolactone,
* cosmetic therapy (waxing, bleaching)
* Induction of ovulation with anti-oestrogens (clomiphene, tamoxifen) and gonadotropins
* Anti-DM meds; metformin

* \*\*Advise on high risk of dev’pt of type II DM, gestational DM, arterial disease like HT (androgen), endometrial hyperplasia & CA\*\*

Mnx of pcos

**Oligomenorrhoea/amenorrhoea**

* tend to be anovulatory, normal or high oestrogen levels.
* endometrium that develops under the influence of oestrogen eventually becomes unsustainable and sheds.
* For these reasons, cyclical progesterone is often useful in the treatment
* Medroxyprogesterone acetate 10 mg daily for 10 days. The woman will normally bleed a few days after progesterone treatment stops.
* metformin, increases insulin sensitivity, is partially effective in its treatment.

**Hirsutism**

* arises from the growth-promoting effects of androgen at the hair follicle.
* Some of these growth-promoting effects are irreversible, even when androgen levels fall.
* However, lowering free androgen levels will slow the rate of hair growth, which most patients see as a benefit.
* The possible treatment options include the following.
* **Eflornithine cream**, applied topically.
* **Cyproterone acetate**: an anti-androgen that competitively inhibits the androgen receptor.
* **GnRH analogues with low-dose HRT**: this regime should be reserved for women intolerant to other therapies, or for short-term treatment, since bone loss is an inevitable side effect.
* **Surgical treatments**aimed at destroying the hair follicle, such as laser or electrolysis

**Subfertility**

* may respond to treatment either with clomiphene or with gonadotrophin therapy.
* there is some evidence that metformin may increase ovulation rates, either alone or when used in combination with clomiphene.
* metformin has been shown to increase ovulation rates (and therefore frequency of menses) by around once every 5 months.

**ABNORMAL UTERINE BLEEDING (AUB) (FIGO, ACOG-2011)**

* Any uterine bleeding outside the normal volume, duration, regularity or frequency is considered abnormal uterine bleeding (AUB).

**NORMAL MENSTRUATION**

* Cycle interval =28 days (21–35 days)
* Menstrual flow = 4–5 days
* Menstrual blood loss = 35 mL (20–80 mL)
* Abnormal menstrual bleeding pattern have been traditionally expressed by terms like menorrhagia, metrorrhagia, polymenorrhea, and oligomenorrhea

AUB

* In order to create an universally accepted nomenclature to describe abnormal uterine bleeding, International Federation of Gynecology and Obstetrics (FIGO) and American College of Obstetricians and Gynaecologists (ACOG) introduced newer system of terminology to describe AUB.
* The newer classification system is known by the acronym **PALM–COEIN (FIGO–2011).**
* It is used to classify the abnormal uterine bleeding on the basis of etiology:
  + - * **Polyp,**
      * **Adenomyosis,**
      * **Leiomyoma,**
      * **Malignancy and hyperplasia,**
      * **Coagulopathy,**
      * **0vulatory dysfunction,**
      * **Endometrial,**
      * **Iatrogenic, and**
      * **Not yet classified are the different etiological factors expressed by one (or more) letters.**

**Etiopathology of AUB**

Structural causes (PALM)

* **Polyp**
* **Adenomyosis**
* **Leiomyoma**
* **Malignancy and**hyperplasia

Nonstructural systemic causes (COEIN)

* **Coagulopathy**
* **Ovulatory**dysfunction
* **Endometrial**
* **Iatrogenic**
* **Not yet**identified

**Diagnosis of Abnormal Uterine Bleeding**

•           **Detailed history taking and physical examination**

**History:**Age,patterns of abnormal uterine bleeding, severity, associated pain, family history and use of medication

**physical examination:**Pallor, edema, neck glands, thyroid, and systemic examination, and pelvic examination (per speculum, Pap smear, and bimanual examination) are included

•           **Laboratory investigations:**Complete hemogram**,**thyroid profile, pregnancy test, coagulation profile.

•           **Imaging studies:**Ultrasonography (Transvaginal), hysteroscopy

•           **Magnetic resonance imaging (MRI):**second line procedure especially in cases with adenomyosis.

E. Histological confirmation of pathology

**POSTMENOPAUSAL BLEEDING**

* Vaginal bleeding after the menopause.
* In women who are not taking HRT, any bleeding is abnormal.
* In women on combined cyclical HRT, bleeding in the progesterone free period is normal.
* Unscheduled bleeding refers to bleeding at other times, and this is abnormal and should be investigated.

AETIOLOGY OF PMB

* majority of women have atrophic vaginitis, whereby the vaginal epithelium thins and breaks down in response to low oestrogen levels.
* This is a benign condition, which is relatively easily treated with topical oestrogens.
* 10% of women with PMB will be found to have endometrial cancer, the risk of which is greater for those who are not currently taking HRT, and progressively increases with increasing age.

**Differential diagnosis OF PMB**

* endometrial carcinoma
* endometrial hyperplasia
* endometrial polyps
* cervical malignancy
* atrophic vaginitis.
* Summary
* AUB
* •In order to create an universally accepted nomenclature to describe abnormal uterine bleeding, International Federation of Gynecology and Obstetrics (FIGO) and American College of Obstetricians and Gynaecologists (ACOG) introduced newer system of terminology to describe AUB.
* •The newer classification system is known by the acronym PALM–COEIN (FIGO–2011).
* •It is used to classify the abnormal uterine bleeding on the basis of etiology:
* –Polyp,
* –Adenomyosis,
* –Leiomyoma,
* –Malignancy and hyperplasia,
* –Coagulopathy,
* –0vulatory dysfunction,
* –Endometrial,
* –Iatrogenic, and
* –Not yet classified are the different etiological factors expressed by one (or more) letters.

### ENDOMETRIOSIS

**DEFINITION**

***ENDOMETRIOSIS***:

       Abnormal growths of tissue histologically resembling the endometrium in locations other than the uterine lining.

***ADENOMYOSIS*:**

* n     Presence of endometrial glands and stroma within the myometrium on hitological examination.
* n  Also called endometriosis interna
* n  ENDOMETRIOSIS
* n  It is a benign but it is locally invasive
* n  disseminates widely.
* n  Cyclic hormones stimulate growth
* n  Does NOT Discriminate by Race
* n  The prevalence is about 10 percent.

sites of endometriosis

* **Abdominal**
* **Extra-abdominal:**
* –   The common sites are abdominal scar of hysterotomy, cesarean section, tubectomy and myomectomy, umbilicus, episiotomy scar, vagina and cervix.

**Commonest sites**

* Ovary-50%. Pod, utero-sacral ligaments,posterior visceral surface of the uterus,broad ligament, bowel,bladder&ureters.
* *Rare*- deep in the cervix,vaginal fornices,wounds contaminated with endometrial tissue.
* *Distant*- out of the pelvis- lungs,brain&kidney.
* Risk factors
* Single/nulliparous
* Early menarche
* Non oral contraception
* Non smoker shorter cycle/longer duration of flow

**PATHOGENESIS**

* still remains unclear and is **full of theories**:
* **1. Retrograde Menstruation (Sampson’s theory):**
* –           There is retrograde flow of menstrual blood through the uterine tubes. Endometrial fragments get implanted in the peritoneal surface of the pelvic organs.
* 2. **Coelomic metaplasia (Meyer and Ivanoff):**
* –   Chronic irritation of the pelvic peritoneum by the menstrual blood may cause coelomic metaplasia which results in endometriosis.
* **3. Lymphatic Theory (Halban):**
* –   It may be possible for the normal endometrium to metastasize the pelvic lymph nodes through the draining lymphatic channels
* **4. Vascular Theory:**
* –   This is sound at least to explain endometriosis at distant sites such as lungs, arms or thighs.
* 5. others: **Genetic and Immunological Factors, Direct Implantation, Environment theory.**

**PATHOLOGY**

* Endometrial lesions appear as red velvety implants on the peritoneal surface. Further growth gives them a cystic, darkblue or black appearance. Lesions may grow to 5-10 mm surrounded by extensive adhesions. In the ovaries the cysts may enlarge to several cm; endometriomas or ‘chocolate cysts’.

**CLINICAL FEATURES**

* The age is 30–45.
* Infertility,
* patients are mostly nulliparous
* 25% have no symptom
* Dysmenorrhea (70%)
* Menorrhagia
* Dyspareunia
* Chronic Pelvic Pain

**Other Symptoms**

* n  Urinary—frequency, dysuria, back pain or even hematuria
* n  Sigmoid colon and rectum—painful defecation (dyschezia), diarrhea, constipation, rectal bleeding or even melena
* n  Chronic fatigue, perimenstrual symptoms (bowel, bladder)
* n  Hemoptysis (rarely), catamenial chest pain
* n  Surgical scars—cyclical pain and bleeding

Diagnosis

* confirm by laparoscopy\ laparotomy and biopsy for histology. (“Gold Standard)
* Inconclusive Ix:
* –   CA-125 - moderate elevationin patients with severe endometriosis,
* –   History & Pelvic Exam,
* –   Imaging Studies
  + - * Ultrasonography is not much helpful to the diagnosis.
      * MRI & CT scan

Treatment: Overall Approach

Recognize Goals:

       – Pain Management

       – Preservation / Restoration of Fertility

Discuss with Patient:

       – Disease may be Chronic and Not Curable

       – Optimal Treatment Unproven or Nonexistent

**TREATMENT**

Depends on desire for future fertility, symptoms, disease stage and age of the patient.

  Minimal disease – observe on NSAIDS and prostaglandin inhibitors.

Moderate – pseudo pregnancy – ocps.

Severe disease – pseudomenopause – e.g.. Danazol, gnrh agonists - Buserelin , Goserelin, Leuprorelin .

Surgery – excision & adhesionolysis, For those with DFS – TAH + BSO, Appendicectomy and excision of all lesions.

Pain Management: Medical Therapy

* NSAIDs
* OCPs (Continuous)
* Progestins
* Danazol
* GnRH-a
* GnRH-a + Add-Back Therapy
* Misc: Opoids, TCAs, SSRIs

Surgical Treatment

* (Laparoscopy / Laparotomy
* Excision / Fulgeration
* Resection of Endometrioma
* Lysis of Adhesions, Cul-de-sac Reconstruction
* Uterosacral Nerve Ablation
* Presacral Neurectomy
* Appendectomy
* Uterine Suspension
* Hysterectomy +/- BSO

**PROGNOSIS**

* Counseling after diagnosis and staging is vital for decision of management mode.
* May reccur even after definitive surgery

### ADENOMYOSIS

* Adenomyosis is a condition where there is in-growth of the endometrium, both the glandular and stromal components, directly into the myometrium.
* The cause of such in-growth is not known.
* It may be related to repeated childbirths, vigorous curettage or excess of estrogen effect
* It is thought to be direct contamination of endometrial surface where isolate islands have lost the connection with the surface endometrium from fibrosis or musculature.

**CLINICAL FEATURES**

* one-third remains **asymptomatic**
* **Menorrhagia (70%) *-***unresponsive to hormonal therapy or uterine curettage
* **Dysmenorrhea (30%)**
* **Pelvic pain**
* **Dyspareunia or frequency of urination**
* **Sub-Infertility & pregnancy loss:**
* –   abnormal function of the subendometrial myometrium,
* –   retrograde myometrial contractions,
* –   interference in sperm transport and blastocyst implantation and
* –   abnormal endomerial immune respose and nitric oxide level

diagnosis

**Ultrasound and Color Doppler (TVS)**characteristics are:

* –   Myometrium normally has three distinct zones of different echogenecity.
* –   The inner layer is hypoechoic relative to the middle and outer layer.
* –   This subendometrial halo is characteristic in adenomyosis.
* –   Other features are:
  + - § heterogenous echogenecity,
    - § hypoecoic myometrium with multiple small cysts in the myometrium (honeycomb appearance),
    - § increased vascularity within the myometrium

**Magnetic Resonance Imaging (MRI)**

MRI should be expected to be excellent in recognizing uterine masses like fibroids, cysts, and adenomyomas if they reach 5 mm. or greater in size.

MRI may be able to lead us to expect adenomyosis if the myometrial thickness is increased or the consistency of the myometrium is changed.

**Hysterography**

the presence of ill defined areas of contrast intravasation extending perpendicularly from the uterine cavity into the myometrium is the most characteristic feature of adenomyosis on hysterography.

Unfortunately, the sensitivity of this technique is too low for clinical practice.

n  **TREATMENT:**

n  The only definitive treatment for adenomyosis is total hysterectomy, with or without ovarian conservation.

n  Chemotherapy – ocps reduce pain and bleeding.

n  DXT – destroys ovaries and reduces I.e. for  those who cannot stand surgery.

n  Prognosis – Hysterectomy is curative.

n  Levonorgestrel–releasing-IUS is found to improve the menorrhagia and dysmenorrhea.

n  Danazol—loaded (300–400 mg) intrauterine device (IUD) is also found to improve the symptoms of menorrhagia and dysmenorrhea.

n  .

A good gynecologist may suspect adenomyosis based on the clinical factors, but the final diagnosis usually has to wait until hysterectomy is performed.

### Summary

ENDOMETRIOSIS:

  Abnormal growths of tissue histologically resembling the endometrium in locations other than the uterine lining  
Diagnosis: confirm by laparoscopy\ laparotomy and biopsy for histology. (“Gold Standard)  
Treatment: Depends on desire for future fertility, symptoms, disease stage and age of the patient.

* Minimal disease – observe on NSAIDS and prostaglandin inhibitors.
* Moderate – pseudo pregnancy – ocps.
* Severe disease – pseudomenopause – e.g.. Danazol, gnrh agonists - Buserelin , Goserelin, Leuprorelin .
* Surgery – excision & adhesionolysis, For those with DFS – TAH + BSO, Appendicectomy and excision of all lesions.

ADENOMYOSIS:

Presence of endometrial glands and stroma within the myometrium on hitological examination.

Also called endometriosis interna

The only definitive treatment for adenomyosis is total hysterectomy, with or without ovarian conservation

A good gynecologist may suspect adenomyosis based on the clinical factors, but the final diagnosis usually has to wait until hysterectomy is performed.

## Unit Five Content..

### Topic 1: Fertilization and Implantation

**FERTILIZATION**

Fertilization is the process of fusion of the spermatozoon with the mature ovum. It begins with sperm egg collision and ends with production of a mononucleated single cell called the zygote. Its objectives are:

(1) To initiate the embryonic development of the egg and

(2) To restore the chromosome number of the species.

Almost always, fertilization occurs in the ampullary part of the uterine tube.

**Process of Fertilization**

After the male ejaculates semen into the vagina during intercourse, a few sperm are transported within 5 to 10 minutes upward from the vagina and through the uterus and fallopian tubes to the  
*ampullae*of the fallopian tubes near the ovarian ends of the tubes. This transport of the sperm is aided by contractions of the uterus and fallopian tubes stimulated by prostaglandins in the male seminal fluid and also by oxytocin released from the posterior pituitary gland of the female during her orgasm. Of the almost half a billion sperm deposited in the vagina, a few thousand succeed in  
reaching each ampulla.

·         Complete dissolution of the cells of the corona radiata occurs by the chemical action of the hyaluronidase liberated from the acrosomal cap of the hundreds of sperm present at the site.

·         Penetration of the zona pellucida is facilitated by the release of hyaluronidase from the acrosomal cap. More than one sperm may penetrate the zona pellucida.

·         Out of the many sperms, one touches the oolemma. Soon after the sperm fusion, penetration of other sperm is prevented by zona reaction (hardening) and oolemma block. This is due to release of cortical granules by exocytosis from the oocyte.

·         Completion of the second meiotic division of the oocyte immediately follows, each containing haploid number of chromosomes (23, X). The bigger one is called the female pronucleus and the smaller one is called second polar body which is pushed to the perivitelline space.

·         In the human, both the head and tail of the spermatozoon enter the cytoplasm of the oocyte but the plasma membrane is left behind on the oocyte surface. Head and the neck of the spermatozoon become male pronucleus containing haploid number of chromosomes (23, X) or (23, Y).

·         The male and the female pronuclei unite at the center with restoration of the diploid number of chromosomes (46) which is constant for the species. The zygote, thus formed, contains both the paternal and maternal genetic materials. In some instances, an antigen called fertilizin present on the cortex and its coat of the ovum, reacts with the antibody called antifertilizin liberated at the plasma membrane of the sperm head. Thus the union between the two gametes may be an immunological reaction (chemotaxis).

**IMPLANTATION *(Nidation)***

Implantation occurs in the endometrium of the anterior or posterior wall of the body near the fundus on the 6th day after fertilization which corresponds to the 20th day of a regular menstrual cycle. Implantation occurs through four stages e.g. apposition, adhesion, penetration and invasion.

**APPOSITION:**Occurs through pinopod formation. Pinopods are long finger like projections (microvilli) from the endometrial cell surface. These pinopods absorb the endometrial fluid which is secreted by the endometrial gland cells. This fluid, rich in glycogen and mucin provides nutrition to the blastocyst initially. Unless this fluid is absorbed, adhesion phase cannot occur.

**ADHESION**of blastocyst to the endometrium occurs through the adhesion molecules like integrin, selectin and cadherin (glycoproteins).

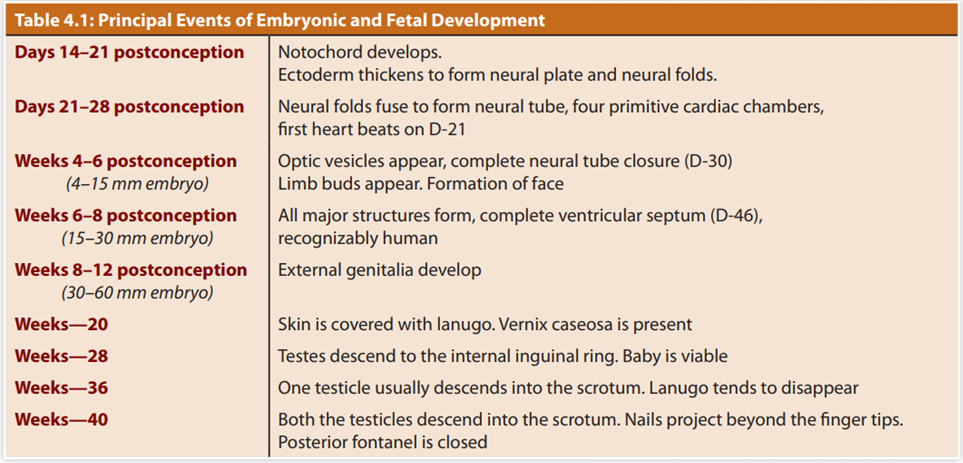
**PENETRATION AND INVASION:**Actual penetration and invasion occur through the stromal cells in between the glands and is facilitated by the histolytic action of the blastocyst. With increasing lysis of the stromal cells, the blastocyst is burrowed more and more inside the stratum compactum of the decidua. Vacuoles appear in the advancing syncytium which fuse to form large lacunae. These are more evident at the embryonic pole. Concurrently, the syncytial cells penetrate deeper into the stroma and erode the endothelium of the maternal capillaries. The syncytium by penetrating the vessels, not only becomes continuous with the endothelial lining but permits the maternal blood to enter into the lacunar system. Ultimately erosion of few maternal arteries with formation of blood space (lacunae) occurs. Nutrition is now obtained by aerobic metabolic pathway from the maternal blood. Further penetration is stopped probably by the maternal immunological factor and the original point of entry is sealed by fibrin clot and later by epithelium. The process is completed by 10th or 11th day which corresponds to D 24-25 from LMP. This type of deeper penetration of the human blastocyst is called interstitial implantation and the blastocyst is covered on all sides by the endometrium (decidua). Occasionally, there may be increased blood flow into the lacunar spaces at the abembryonic pole. This results in disruption of the lacunae and extravasation of blood into the endometrial cavity. This corresponds approximately to 13th day after fertilization (at about the expected day of the following period). This may produce confusion in determination of the expected date of delivery.

### Normal Pregnancy and the Fetus

**Normal Pregnancy**

**Pregnancy**(gestation) is the physiologic process of a developing fetus within the maternal body.  
Several terms are used to define the developmental stage of human conception and the duration of pregnancy. For obstetric purposes, the gestational age or menstrual age is the time elapsed since the first day of the last normal menstrual period (LNMP), which actually precedes the time of oocyte fertilization. The gestational age is expressed in completed weeks. The start of the gestation (based on the LNMP) is usually 2 weeks before ovulation, assuming a 28-day regular menstrual cycle. The developmental or fetal age is the age of the conception calculated from the time of implantation, which is 4 to 6 days after ovulation is completed. The menstrual gestational age of pregnancy is calculated at 280 days or 40 completed weeks. The estimated due date (EDD) may be estimated by adding 7 days to the first day of the last menstrual period and subtracting 3 months plus 1 year (Naegele’s rule).

The period of gestation can be divided into units consisting of 3 calendar months each or 3 trimesters.  
Three periods are distinguished in the prenatal development of the fetus. (1) **Ovular period or germinal period**—which lasts for first 2 weeks following ovulation. In spite of the fact that the ovum is fertilized, it is still designated as ovum. (2) **Embryonic period**—begins at 3rd week following ovulation and extends up to 10 weeks of gestation (8 weeks post conception). The crown-rump length (CRL) of the embryo is 4 mm. (3) **Fetal period**begins after 8th week following conception and ends in delivery. **The chronology in the fetal period is henceforth expressed in terms of menstrual age and not in embryonic age**. The principal events of embryonic and fetal development are shown in the table below:



**THE FETUS**

**GROWTH OF THE FETUS**

**Normal fetal growth**is characterized by cellular hyperplasia followed by hyperplasia and hypertrophy and lastly by hypertrophy alone. The fetal growth increases linearly until 37th week. It is controlled by genetic factor in the first half and by environmental factors in the second half of pregnancy. **The important physiological factors are:**Race (European babies are heavier than Indians); Sex (male baby weighs > female); Parental height and weight (tall and heavier mother have heavier babies); Birth order (weight rises from first to second pregnancy) and Socioeconomic factors (heavier babies in social class I and II). Fetal growth is predominantly controlled by IGF-1, insulin and other growth factors. Growth hormone is essential for postnatal growth. **At term**, **the average fetal weight in India varies from 2.5 kg to 3.5 kg**.

**FETAL NUTRITION**

**There are three stages of fetal nutrition following fertilization:**

(1) ***Absorption:***In the early postfertilization period, nutrition is stored in deutoplasm within cytoplasm and the very little extra nutrition needed is supplied from the tubal and uterine secretion.  
(2) ***Histotrophic transfer:***Following nidation and before the establishment of uteroplacental circulation, nutrition is derived from eroded decidua by diffusion and later on from the stagnant maternal blood in the trophoblastic lacunae.

(3) ***Hematotrophic:***With the establishment of the fetal circulation, nutrition is obtained by active and passive transfer from the 3rd week onwards.

The fetus is a separated physiological entity and it takes what it needs from the mother even at the cost of reducing her resources. While all the nutrients are reaching the fetus throughout the intrauterine period, the demand is not squarely distributed. **Two-thirds of the total calcium, three-fifths of the total proteins and four-fifths of the total iron are drained from the mother during the last 3 months**. Thus, in preterm births, the store of the essential nutrients to the fetus is much low. The excess iron reserve is to compensate for the low supply of iron in breast milk which is the source of nutrients following birth.

**THE FETAL CIRCULATION**

**The umbilical vein carrying the oxygenated blood (80% saturated) from the placenta**, enters the fetus at the umbilicus and runs along the free margin of the falciform ligament of the liver. In the liver, it gives off branches to the left lobe of the liver and receives the deoxygenated blood from the portal vein. The greater portion of the oxygenated blood, mixed with some portal venous blood, short circuits the liver through the **ductus venosus**to enter the inferior vena cava (IVC) and thence to right atrium of the heart. The O2 content of this mixed blood is thus reduced. Although both the ductus venosus and hepatic portal/fetal trunk bloods enter the right atrium through the IVC, there is little mixing. The terminal part of the IVC receives blood from the right hepatic vein.  
In the right atrium, most of the well oxygenated (75%) ductus venosus blood is preferentially directed into the foramen ovale by the valve of the inferior vena cava and crista dividens and passes into the left atrium. Here it is mixed with small amount of venous blood returning from the lungs through the pulmonary veins. This left atrial blood is passed on through the mitral opening into the left ventricle.

Remaining lesser amount of blood (25%), after reaching the right atrium via the superior and inferior vena cava (carrying the venous blood from the cephalic and caudal parts of the fetus respectively) passes through the tricuspid opening into the right ventricle.

During ventricular systole, the left ventricular blood is pumped into the ascending and arch of aorta and distributed by their branches to the heart, head, neck, brain and arms. The right ventricular blood with low oxygen content is discharged into the pulmonary trunk. Since the resistance in the pulmonary arteries during fetal life is very high, the main portion of the blood passes directly through the **ductus arteriosus**into the descending aorta bypassing the lungs where it mixes with the blood from the proximal aorta. 70% of the cardiac output (60% from right and 10% from left ventricle) is carried by the ductus arteriosus to the descending aorta. About 40% of the combined output goes to the placenta through the umbilical arteries. **The deoxygenated blood leaves the body by way of two umbilical arteries to reach the placenta**where it is oxygenated and gets ready for recirculation. **The mean cardiac output is comparatively high in fetus and is estimated to be 350 mL/kg/min**.

Common Pregnancy Complaints and Questions

**First Trimester**

**What are the first symptoms of pregnancy?**

Missing a period is usually the first signal of a new pregnancy, although women with irregular periods may not initially recognize a missed period as pregnancy. During this time, many women experience a need to urinate frequently, extreme fatigue, nausea and/or vomiting, and increased breast tenderness. All of these symptoms can be normal. Most over-the-counter pregnancy tests are sensitive 9-12 days after conception, and they are readily available at most drug stores. Performing these tests early helps to allay confusion and guesswork. A serum pregnancy test (performed in a provider's office or laboratory facility) can detect pregnancy 8-11 days after conception.

**How long after conception does the fertilized egg implant?**

The fertilized conceptus enters the uterus as a 2- to 8-cell embryo and freely floats in the endometrial cavity about 90-150 hours, roughly 4-7 days after conception. Most embryos implant by the morula stage, when the embryo consists of many cells. This happens, on average, 6 days after conception. However, there is great variance in the time of implantation. It can occur on days 16-30 of the menstrual cycle. The new embryo then induces the lining changes of the endometrium, which is called decidualization. It then rapidly begins to develop the physiologic changes that establish maternal-placental exchange. Prior to this time, medications ingested by the mother typically do not affect a pregnancy.

**Is cramping during pregnancy normal?**

Early in pregnancy, uterine cramping can indicate normal changes of pregnancy initiated by hormonal changes; later in pregnancy, it can indicate a growing uterus. Cramping that is different from previous pregnancies, worsening cramping, or cramping associated with any vaginal bleeding may be a sign of [ectopic pregnancy](http://emedicine.medscape.com/article/2041923-overview), [threatened abortion](http://emedicine.medscape.com/article/252560-overview), or [missed abortion](http://emedicine.medscape.com/article/266317-overview).

Other physical effects that are normal during pregnancy, and not necessarily signs of disease, include nausea, vomiting, increase in abdominal girth, changes in bowel habits, increased urinary frequency, palpitations or more rapid heartbeat, upheaving of the chest (particularly with breathing), heart murmurs, swelling of the ankles, and shortness of breath.

**Why do pregnant women feel tired?**

Fatigue in early pregnancy is very normal. Many changes are occurring as the new pregnancy develops, and women experience this as fatigue and an increased need for sleep. Lower blood pressure level, lower blood sugar levels, hormonal changes due to the soporific effects of progesterone, metabolic changes, and the physiologic anemia of pregnancy all contribute to fatigue. Women should check with their health care provider to determine if an additional work up, prenatal vitamin changes, and/or supplemental iron would be beneficial.

**Second Trimester**

**When do the postural changes of pregnancy occur?**

Women experience a progressive increase in the anterior convex shape of the lumbar spine during pregnancy. This change, termed lordosis, helps keep the center of gravity stable and over the legs as the uterus enlarges. Late in pregnancy, aching, weakness, and numbness of the arms may occur secondary to compensatory anterior positioning of the neck and hunching of the shoulders in positional response to exaggerated lordosis. These positional responses put traction on the ulnar and median nerves, resulting in the previously mentioned symptoms.

In pregnancy, relaxin is secreted by the corpus luteum, the placenta, and part of the decidual lining of the uterus. It is thought to cause remodeling of the connective tissue of the reproductive tract and especially induce biochemical changes of the cervix.

**When is fetal movement usually felt?**

Most women feel the beginnings of fetal movement before 20 weeks' gestation. In a first pregnancy, this can occur around 18 weeks' gestation, and in following pregnancies it can occur as early as 15-16 weeks' gestation. Early fetal movement is felt most commonly when the woman is sitting or lying quietly and concentrating on her body. It is usually described as a tickle or feathery feeling below the umbilical area. The point at which a woman feels the baby move is termed **quickening.**

Placental location can impact the timing of quickening. An anterior placenta can "cushion" against fetal movement and delay maternal detection of fetal movements. As the fetus grows larger, the fetal movement feelings become stronger, regular, and easier to detect. While there is no absolute number that indicates fetal well-being, typical guidance may include that fetuses should move approximately 4 times an hour as they get larger, and some clinicians advise patients to count fetal movements to follow fetal well-being.

**What kind of breast changes are normal during pregnancy?**

Pregnancy-related breast changes include growth and enlargement, tenderness, darkening of the nipples, and darkened veins due to increased blood flow. In addition, small raised bumps (Montgomery tubercles) appear around the areola in mid-pregnancy.

**Third Trimester**

**How much does the uterus grow during pregnancy?**

The uterus grows from an organ that weighs 70 g with a cavity space of about 1 mL to an organ that weighs more than 1000 g that can accumulate a fluid area of almost 20 L. The shape also evolves during pregnancy from the original pearlike shape to a more round form, and it is almost a sphere by the early third trimester. By full term, the uterus becomes ovoid. The uterus is completely palpable in the abdomen (not just by pelvic examination) at about 12-14 weeks' gestation. After 20 weeks' gestation, most women begin to appear pregnant upon visual examination.

**Is it normal to secrete milk from the breast prior to delivery?**

Galactorrhea (milk secretion from the nipple) is the product of the combined effects of prolactin, glucocorticoids, progesterone, and human placental lactogen. Galactorrhea is not uncommon in the first trimester, although it usually does not occur until milk let-down soon after delivery. At that time, the high levels of progesterone, which block milk excretion, drop with the delivery of the placenta. In mid-pregnancy a woman reaches lactogenesis stage I and she is able to secrete colostrum.

Early galactorrhea does not mean that a woman will produce less milk after delivery. Some women notice secretions beginning before the fifth month of pregnancy. Many women find they spontaneously leak or express some fluid by the ninth month.

Early milk secretion, known as colostrum, is watery and pale. Colostrum has more protein and lower fat levels than mature milk.

Lactogenesis stages II and III occur postpartum and form more mature milk.

**Nutrition in Pregnancy**

**What are the most common dietary complaints during pregnancy?**

During early pregnancy, most women experience an increased appetite, with extra caloric needs of approximately 300 kcal/d. Stomach motility decreases, probably due to decreased production of motilin. A decreased incidence of peptic ulcer disease is due to a reduction in gastric acid secretion. Prolonged transit times through the colon are also reported, with transit from the stomach to the cecum occurring in about 58 hours instead of 52 hours.

Some women have nonfood cravings, known as pica.

Women who experience nausea or hyperemesis may develop ptyalism (spitting). Reported fluid losses of 1-2 L/d can occur in these women.

**Should certain foods be avoided during pregnancy?**

Pregnant women are at increased risk of bacterial food poisoning. For the safety of both mother and fetus, it is important to take steps to prevent foodborne illnesses, including the following:

* Properly cook food to kill bacteria. Use a meat thermometer to determine the appropriate temperature, although cooking until well done is safe for most meat. Ground beef should be cooked to at least 160°F, roasts and steaks to 145°F, and whole poultry to 180°F.
* Cook eggs until they have a firm yolk and are white. Eggnog and hollandaise sauce have raw or partially cooked eggs and are not considered safe.
* Eat liver in moderation. Liver can contain extremely high levels of vitamin A.
* Avoid products containing unpasteurized milk, including soft cheeses like brie, feta, and blue cheese. Also avoid unpasteurized juice.
* Carefully wash all fruits and vegetables to eliminate harmful bacteria. Avoid raw sprouts altogether.
* Limit caffeine intake. Caffeine crosses the placenta and can affect fetal heart rate. Some clinicians recommend limiting caffeine to less than 200 mg/day (about 2 cups of coffee).

**Can women safely eat fish while pregnant?**

The American College of Obstetricians and Gynecologists (ACOG) issued a warning regarding eating fish in response to the US FDA's consumer advisory about the dangers of eating fish for nursing mothers and women who are or who may become pregnant. The fish themselves are not harmful, but extensive fish consumption increases exposure to the naturally occurring compound methylmercury, levels of which have been increasing in the waters because of industrial pollution. Mercury is very toxic and can cause danger to the fetus and to the newborn nursing infant. Mercury exposure can actually occur via inhalation and/or skin absorption, and all fish contain trace amounts. However, longer-lived and larger fish, such as shark, swordfish, king mackerel, and tilefish, have increased mercury levels and cause the most concern for consumption by pregnant women.

**Other Questions Related to Pregnancy**

**What is the recommended weight gain in pregnancy?**

Important variables to consider regarding weight gain recommendations include the presence of twin or triplet pregnancies, maternal age, and maternal prepregnancy weight. These variables can add to the burden of chronic disease for the mother and baby; excessive weight gain is associated with an increased risk for gestational diabetes, pregnancy-associated hypertension, and delivery of large-for-gestational-age (LGA) infants.

Guidelines for weight gain during pregnancy\* are as follows:

* Underweight women (BMI < 18.5) should gain 12.5-18 kilograms.
* Normal-weight women (BMI, 18.5-24.9) should gain 11-16 kilograms.
* Overweight women (BMI, 25-29.9) should gain 6.8-11 kilograms.
* Obese women (BMI, 30 or higher) should gain 5-9 kilograms.

\*Weight gain guidelines are for singleton pregnancy; weight gain should be higher for multiple pregnancies but the ideal amounts are unknown.

Clinicians are urged to supplement these guidelines with individualized counseling about diet and exercise, and preconception counseling should emphasize the importance of conceiving when the mother is at a normal body mass index (BMI). To help mothers attain these goals, dietary, lifestyle, and exercise interventions have been shown to be safe and effective at reducing excessive weight gain in pregnancy

**Do older fathers have an increased risk of fathering children with birth defects?**

No medical information exists to support the hypothesis that increased paternal age causes increased numerical chromosomal abnormalities in the manner that increased maternal age does. However, as males age, structural spermatozoa abnormalities increase, and affected sperm usually cannot fertilize eggs.

The literature suggests a 0.3%-0.5% risk of autosomal dominant disease in offspring of fathers aged 40 years or older.Autosomal dominant disorders include [neurofibromatosis](http://emedicine.medscape.com/article/950151-overview), [Marfan syndrome](http://emedicine.medscape.com/article/946315-overview), [achondroplasia](http://emedicine.medscape.com/article/941280-overview), and [polycystic kidney disease](http://emedicine.medscape.com/article/244907-overview).

**Should women wear seatbelts during pregnancy?**

Seatbelts should absolutely be worn during pregnancy. Trauma to the mother is more devastating to the child than any potential entrapment of the pregnant abdomen in the seatbelt. The seatbelt should be placed low, across the hip bones and under the pregnant abdomen. The shoulder strap should be placed to the side of the abdomen, between the breasts, and over the midportion of the clavicle. No information indicates that air bags are unsafe during pregnancy. Pregnant women should try to keep their abdomen 10 inches from the airbag.

**Can pregnant women go to the dentist?**

Dental care during pregnancy is an important part of overall healthcare.During pregnancy, the gums naturally become more edematous and may bleed after brushing. Epulis gravidarum, a type of gingivitis with violaceous pedunculated lesions, can occur. If treatment of cavities, surgery, or infection care is required, be sure the dentist is aware of the pregnancy. Most antibiotics and local anesthetics are safe to use during pregnancy. Radiographs can be obtained with abdominal shielding but are best avoided during pregnancy because a small, but statistically significant, increase in childhood malignancies exists in children exposed to in-utero radiographic irradiation.

**Why is heartburn more common during pregnancy?**

Stomach emptying was thought to be retarded during pregnancy, but hormonal influences of increased progesterone and/or decreased levels of motilin may be more responsible for pyrosis (heartburn) than the actual mechanical obstruction in the third trimester. Some studies have also shown decreased lower esophageal sphincter tone, which can lead to an excess of gastric acid in the esophagus.

**Why is back pain prevalent during pregnancy and can it be treated?**

Half of women report having back pain at some point during pregnancy. The pain can be lumbar or sacroiliac. The pain may also be present only at night. Back pain is thought to be due to multiple factors, which include shifting of the center of gravity caused by the enlarging uterus, increased joint laxity due to an increase in relaxin, stretching of the ligaments (which are pain-sensitive structures), and pregnancy-related circulatory changes.

Treatment is heat and ice, acetaminophen, massage, proper posturing, good support shoes, and a good exercise program for strength and conditioning. Pregnant women may also relieve back pain by placing one foot on a stool when standing for long periods of time and placing a pillow between the legs when lying down.

**Is sexual intercourse safe during pregnancy?**

Research indicates that sexual intercourse is safe in the absence of ruptured membranes, bleeding, or placenta previa, but pregnant women engage in sex less often as their pregnancy progresses. ACOG states that sexual activity during pregnancy is safe for most women right up until labor, unless there is a specific contraindication.

ACOG specifically cautions that a woman should limit or avoid sex if she has a history of preterm labor or birth, more than one miscarriage, placenta previa, infection, bleeding, and/or breaking of the amniotic sac or leaking amniotic fluid. ACOG discusses that, as part of natural sexuality, couples may need to try different positions as the woman's stomach grows. Vaginal penetration by the male is not as deep with the male facing the woman's back, and this may be more comfortable for the pregnant woman.

**Why do women get varicose veins during pregnancy?**

Varicose veins are more common as women age; weight gain, the pressure on major venous return from the legs, and familial predisposition increase the risk of developing varicose veins during pregnancy. These can occur in the vulvar area and be fairly painful. Rest, leg elevation, acetaminophen, topical heat, and support stockings are typically all that is necessary. Determining that the varicosities are not complicated by superficial thrombophlebitis is important. Having a venous thromboembolism in association with superficial thrombophlebitis is rare. Hemorrhoids, essentially varicosities of the anorectal veins, may first appear during pregnancy for the same reasons and are aggravated by constipation during pregnancy.

**Why are urinary tract infections more common during pregnancy?**

Pregnancy predisposes women with bacteriuria, which in the nonpregnant state is usually self-limiting, to developing urinary tract infections (UTIs). Normal pregnancy-related physiologic changes contribute to UTIs, including dilatation of the upper collecting systems, hypotonic renal pelvises, increases in urinary tract dead space and vesicoureteral reflux, and reductions in the natural antibacterial activity in the urine and in the phagocytic activity of leukocytes at the mucosal surfaces. UTIs in pregnant women usually do not present with typical symptoms, and they may be asymptomatic. All of these factors increase the likelihood for infections to ascend to the kidneys; pyelonephritis is a serious complication of UTIs.

**How can stretch marks be prevented?**

Unfortunately, striae (stretch marks) cannot be prevented. The degree to which a woman experiences stretch marks is determined genetically. Stretch marks usually occur when weight is lost or gained quickly. Using creams and gels rarely make a difference. Fortunately, striae fade with time and marks become silvery white, but they do not tan. Striae managed early can be reduced with new medical laser technology.

**Work and Exercise During Pregnancy**

**What kind of exercise can women engage in during pregnancy?**

Regular physical activity during pregnancy is felt to improve or maintain physical fitness as well as assisting with weight management, decreasing gestational diabetes in obese women and enhancing well-being. A goal for pregnancy exercise would include working up to moderate-intensity exercise for at least 20-30 minutes daily on most days of the week. Although some preexisting conditions such as chronic bronchitis and heavy smoking call for cautionary increases in activity, the majority of women in pregnancy would benefit from becoming or remaining active. Contact sports such as boxing as well as those with a high risk of falling (such as downhill snow skiing) should be avoided. Walking, swimming and stationary cycling are well suited to pregnancy.  Running, racquet sports and strength training can be safe in pregnancy, especially when these were done regularly prior to pregnancy. Women doing these for the first time should use caution due to the pregnancy causing a change in their center of balance and more laxity in their joints.

**Should women restrict work during pregnancy?**

Maintaining an active and productive lifestyle helps make time pass faster and adds to a feeling of accomplishment. Working during pregnancy is usually not a problem unless a woman has risk factors or a complicated pregnancy. Women should check with their healthcare providers for specific restrictions. With an uncomplicated pregnancy, working close to or near the due date should not be a problem. Pregnant women should wear comfortable clothing, move around frequently if sedentary, drink plenty of fluids, and have time to rest and take breaks. Women with strenuous jobs, those who work with heavy machinery, or those who work with toxic chemicals should consult their healthcare providers and their job's occupational department for restrictions or concerns.

**Postpartum**

**When will the uterus return to normal size?**

The uterus returns to prepregnancy size after approximately 6 weeks. This is accomplished through a process called involution. During involution, the uterus has contractions that women may be able to feel, especially during breastfeeding.

**When can women resume sexual intercourse after pregnancy?**

Women usually can resume sexual intercourse when they feel ready, typically 4-6 weeks after delivery and when bleeding has substantially decreased. Clinically, this is the period when the cervix has closed, which usually occurs at 4 weeks postpartum, and when uterine bleeding is minimal.

The following are other issues to consider:

* Breastfeeding may cause increased vaginal dryness due to slightly decreased estrogen levels.
* Women who have had an episiotomy need at least 2-3 weeks to heal before intercourse.
* An ACOG bulletin indicated that some women may find that they do not have much interest in sex after giving birth because of fatigue, stress, fear of pain, lack of opportunity, and/or lack of desire. This lack of postpartum sexual interest is usually temporary.

**Reference:** Medscape (WebMD LLC)

### Diagnosis of Pregnancy

**DIAGNOSIS OF PREGNANCY**

It is crucial to diagnose pregnancy as soon as possible in order to initiate appropriate prenatal care, avoid teratogen (an agent that can cause a deleterious fetal effect) exposure, and diagnose nonviable or ectopic pregnancies.

**Clinical Findings**

**A. Symptoms & Signs**

A number of clinical signs and symptoms may presumptively indicate pregnancy.

1. **Amenorrhea**—Cessation of menses is caused by hormones (estrogen and progesterone) produced by the corpus luteum. The abrupt cessation of menses in a healthy reproductive-aged female with predictable cycles is highly suggestive of pregnancy.

2. **Nausea & vomiting**—This is a common symptom (50% of pregnancies) that begins as early as 2 weeks’ gestational age and customarily resolves at between 13 and 16 weeks’ gestation. Hyperemesis gravidarum is an extreme form of nausea and vomiting and is characterized by dehydration, weight loss (up to 5%), and ketonuria. In extreme cases of hyperemesis gravidarum, hospitalization, intravenous therapy, antiemetics, and, if needed, parenteral nutrition are given. Uncomplicated nausea and vomiting is treated with frequent small meals, a dry diet, and emotional support.

3. **Breast changes**

A. MASTODYNIA—Breast tenderness may range from tingling to pain caused by hormonal changes affecting the mammary duct and alveolar system.

B. BREAST ENGORGEMENT—Breast engorgement and periareolar venous prominences are also seen early in pregnancy, especially in primiparous patients. Montgomery’s tubercles are the portion of the areolar glands visible on the skin surface. These tubercles can be more pronounced during pregnancy secondary to hormonal changes occurring as early as 6–8 weeks’ gestation.

C. COLOSTRUM SECRETION—Protein and antibody production may occur during pregnancy as early as 16 weeks’ gestation. This secretion is not associated with preterm delivery.

D. DEVELOPMENT OF SECONDARY BREAST TISSUE—Development of secondary breast tissue may occur across the nipple line. Hypertrophy of secondary breast tissue may occur in the axilla and cause a symptomatic mass.

4. **Fetal movement**—The initial perception of fetal movement occurs at 18–20 weeks’ gestation in primiparous patients and as early as 14 weeks’ gestation in multiparous patients. Maternal perception of movement is called quickening, but this is not a dependable sign of pregnancy.

5. **Elevated basal body temperature**—Progesterone produces a 0.5°F increase in the basal body temperature, which persists after the missed menses. The rise in temperature occurs within the luteal phase of the menstrual cycle.

6. **Skin changes**

A. CHLOASMA—The mask of pregnancy is skin darkening of the forehead, bridge of the nose, or cheek bones. This pregnancy-associated change is linked to genetic predisposition and usually occurs after 16 weeks’ gestation. Chloasma is exacerbated by sunlight.

B. LINEA NIGRA—Melanocyte-stimulating hormone increases, causing darkening of the nipples and the lower midline from the umbilicus to the pubis (linea nigra). This skin change is genetically based; skin lightens slightly after delivery of the fetus.

C. STRIAE—Striae marks of the breast and abdomen appear as irregular scars. The striae appear late in pregnancy and are caused by collagen separation.

D. SPIDER TELANGIECTASIA—These are common skin lesions of pregnancy that result from elevated plasma estrogen. Both the vascular stellate skin lesions as well as palmar erythema may be seen in pregnancy and also occur in patients with liver failure.

7. **Pelvic organ changes**

A. CHADWICK’S SIGN—Congestion of the pelvic vasculature causes bluish discoloration of the vagina and the cervix. This is a presumptive sign of pregnancy.

B. HEGAR’S SIGN—There is widening and softening of the body or isthmus of the uterus. This occurs at 6–8 weeks’ menstrual age or gestational age. Estrogen and progesterone cause increased cervical softening and dilation at the external os.

C. LEUKORRHEA—There is an increase in vaginal discharge, containing epithelial cells and cervical mucous, secondary to hormonal changes.

D. PELVIC LIGAMENTS—There is relaxation of the sacroiliac and pubic symphysis during pregnancy. Relaxation is pronounced at the pelvic symphysis.

E. ABDOMINAL ENLARGEMENT—There is progressive abdominal enlargement with growth of uterus during pregnancy. From 18 to 34 weeks there is a good correlation between the uterine fundal measurement in centimeters and the gestational age in weeks.

F. UTERINE CONTRACTIONS—Painless uterine contractions (Braxton Hick’s contractions) are felt as tightening or pressure. They usually begin at approximately 28 weeks’ gestation and increase in regularity with advancing gestational age. These contractions usually disappear with walking or exercise, whereas true labor contractions become more intense.

**Diagnosis**

**A. Fetal Heart Tones**

Fetal heart tones (FHTs) are detectable by handheld Doppler (after 10 weeks’ gestation) or by fetoscope (after 18–20 weeks’ gestation). The normal heart rate is 110–160 beats per minute, with a higher fetal heart rate observed early in pregnancy.

**B. Uterine Size/Fetal Palpation**

Uterine size can be used to diagnose pregnancy secondary to uterine enlargement. Later in pregnancy, the fetus can be palpated through the maternal abdominal wall (after 22 weeks), and the position can be determined by Leopold’s maneuvers.

**C. Imaging Studies**

Sonography is one of the most useful technical aids in diagnosing and monitoring pregnancy. Cardiac activity is discernible at 5–6 weeks via transvaginal sonogram, limb buds at 7–8 weeks, and finger and limb movements at 9–10 weeks. At the end of the embryonic period (10 weeks by LNMP), the embryo has a human appearance. The gestational age can be determined by the crown rump length between 6 and 13 weeks’ gestation, with a margin of error of approximately 8% or 3–5 days.

**D. Pregnancy Tests**

Sensitive, early pregnancy tests measure changes in the level of human chorionic gonadotropin (hCG). There is a small degree of cross-reactivity between luteinizing hormone, follicle-stimulating hormone, and thyrotropin, which all share an α subunit with hCG. The β submit of hCG is produced by the syncytiotrophoblast 8 days after fertilization and may be detected in the maternal serum 8–11 days after conception or as early as 21–22 days after the LNMP. β-hCG levels peak at 10–12 weeks’ gestation and decrease afterward. The half-life of hCG is 1.5 days. Generally, serum and urine levels return to normal (<5 mIU/mL), 21–24 days after delivery or after a fetal loss.

1. **Home pregnancy test**—hCG is a qualitative test that is performed on the first voided morning urine sample. A positive test is usually indicated by a color change. Because the accuracy of the home pregnancy test depends on technique and interpretation, it should always be repeated in the office.

2. **Urine pregnancy test**—An antibody assay recognizing the β-hCG subunit is the initial lab test performed in the office to diagnose pregnancy. The test is reliable, rapid (1–5 minutes), and inexpensive, with a positive test threshold between 5 and 50 mIU/mL, characterized by a color change. This is the most common method to confirm pregnancy.

3. **Serum pregnancy test**—β-hCG can be detected within 7 days after conception or at a menstrual age of 21 days’ gestation. The threshold for a positive test can be as low as 2–4 mIU/mL. Serial quantitative tests of β-hCG are be used to evaluate threatened abortion, ectopic pregnancy, or a molar pregnancy.

### Maternal Physiological Changes in Pregnancy

##### Introduction

v  Maternal physiology undergoes many changes during pregnancy.

v  These changes, which are largely secondary to the effects of progesterone and estrogen, begin as early as 4 weeks gestation and are progressive.

v  These changes both enable the fetus and placenta to grow and prepare the mother and baby for childbirth.

**Reproductive System Changes**

Ø  In the non-pregnant woman, the **uterus** weighs approximately 70g and is almost solid, except for a cavity of 10 mL or less.

Ø  By the end of pregnancy, the uterus has achieved a capacity that is 500 to 1000 times greater (approx. 5L) & weighs about 1100g!

Ø  The size of the uterus increases 5-6 times (from 7x5x3 cm to 35x25x22 cm) mostly due to hypertrophy of muscle cells.

Ø  Uterine ligaments show hypertrophy.

Ø  There is dextrorotation of the uterus (twisting to the right) in 80% of cases.

Ø  Blood flow to the **uterus** increases 10-fold (from 50-500 ml/min)

Ø  The shape of the uterus changes from its original pear shape to be spherical by week 12 and finally attains an ovoid shape towards term.

Ø  By the end of week 12, the enlarged uterus extends out of the pelvis and starts displacing abdominal organs upwards and outwards.

Ø  The lower uterine segment (LUS) starts forming at 4 months gestation and reaches full development (10 cm) at full term.

Ø  The uterine walls thin up but are supported by fibrous tissue.

Ø  As early as 1 month after conception, the **cervix** begins to soften and gain bluish tones. These result from increased vascularity and edema of the entire cervix; and from changes in its collagen network.

Ø  Cervical glands undergo marked proliferation, and there is increased cervical secretions.

Ø  A tenacious mucus plug that obstruct the cervical canal forms soon after conception. This mucus is rich in immunoglobulins and cytokines and may act as an immunological barrier to protect the uterine contents against infection.

Ø  The **fallopian tube** elongates and its musculature undergoes little hypertrophy during pregnancy.

Ø  The **ovaries** show increased vascularity and one ovary contains the active corpus luteum in early pregnancy.

Ø  Ovulation is suspended during pregnancy because of pituitary inhibition.

Ø  The **vulva** becomes edematous and more vascular; superficial varicosities may appear especially in multiparae.

Ø  **Vaginal** walls become hypertrophied, edematous and more vascular.

Ø  The vaginal secretion becomes copious, thin and curdy white due to marked exfoliated cells and bacteria.

Ø  The vaginal pH becomes more acidic (3.5–6), which prevents bacterial infections but promotes yeast infection.

Ø  The changes in the **breasts** are best evident in a primigravida.

Ø  In early pregnancy there is breast tenderness. After the second month, the breasts grow in size, and delicate veins are visible just beneath the skin.

Ø  The **nipples** become considerably larger, more deeply pigmented, and more erectile.

Ø  The **areolae** become broader and more deeply pigmented. Secondary areola may appear.

Ø  **Tubercles of Montgomery** (sebaceous glands) enlarge and secrete a substance to maintain areolar suppleness.

Ø  A thick, yellowish **colostrum-like** fluid can be expressed from the breast starting from around the third month of pregnancy.

**Cardiovascular System Changes**

·         The **heart is displaced upward and outward** making the**apex beat**tobe shifted to the 4th ICS about 2.5 cm outside the midclavicular line.

·         Pulse rate is slightly raised (by 10-15 bpm), often with extrasystoles.

·         **A systolic murmur**may be audible in the apical or pulmonary area due to decreased blood viscosity and torsion of the great vessels.

·         The cardiac output (CO) increases and reaches its peak (of 40–50% increase) at about 30–34 weeks.

·         Systemic vascular resistance (**SVR**) decreases (–21%) due to smooth muscle relaxing effect of progesterone, NO, prostaglandins or ANP.

·         **Diastolic** blood pressure (**BP**) and mean arterial pressure (MAP) decrease by 5–10 mm Hg.

·         **Systolic** arterial pressure declines slightly during pregnancy, reaching a nadir at 24–28 weeks of gestation.

·         **Supine** **hypotension** **syndrome** occurs in 8% of women in the latter half of pregnancy.

**Hematological System Changes**

o   Blood (mostly Plasma) volume increases by 40-45% reaching a peak between 32-34 weeks.

o   Circulating RBCs mass increases by 20-30% (more in multiple pregnancy and with iron supplementation).

o   The disproportionate increase in plasma and RBC volume produces a state of hemodilution (fall in hematocrit) during pregnancy.

o   Erythropoietin rises esp. with no iron supplementation

o   Reticulocyte count increases by 2%

o   Human placental lactogen (**HPL**) may stimulate hemopoiesis

o   Erythrocyte Sedimentation Rate (**ESR**) level rises.

o   **WBC** count rises (increase in polymorphonuclear leucocytes)

o   **Neutrophil** count rises with estrogen levels and peaks at 33 weeks. It stabilizes thereafter until the puerperium when it rises sharply.

o   **T and B lymphocytes**counts do not change but their function is suppressed (making pregnant women more susceptible to viral infections, malaria and leprosy)

o   **Platelet** count and platelet functions are largely unchanged.

o   There is increased concentrations of **all clotting factors**except factors **XI** and **XIII**.

o   Plasma **fibrinolytic activity**decreases during pregnancy and labor and returns to normal within 1 hour of delivery of the placenta.

o   **Antithrombin** **III** level falls.

o   Because of all the above changes, pregnancy is considered as a **procoagulant** state.

**Respiratory System Changes**

§  As the uterus enlarges, the **diaphragm** is elevated by as much as 4 cm. The **rib cage**is displaced upward, increasing the angle of the ribs with the spine. May cause dyspnea.

§  **Abdominal muscles**have less tone and activity during pregnancy, causing respiration to be more **diaphragm** dependent.

§  Respiratory **dead space**volume increases because of relaxation of the musculature of conducting airways.

§  **Tidal** volume & inspiratory reserve volume rise by 30-40%.

§  Elevation of the **diaphragm** is associated with reduction in total lung capacity, expiratory reserve volume and functional residual capacity.

§  Increased levels of progesterone appear to have a critical role in the **hyperventilation** of pregnancy, which develops early in the first trimester.

§  The overall respiratory effect appears to be a decrease in the threshold and an increase in the sensitivity of central chemoreflex responses to CO2.

§  **Respiratory rate**does not change.

§  Carbon dioxide production rises sharply in the third trimester as fetal metabolism increases.

§Increased **alveolar** ventilation due to rise in PaCO2from 96.7 mmHg to 101.8 mmHg.

§  Increase in oxygen consumption of about 16% by term.

§  Fall in **arterio-venous** oxygen difference.

§  Pregnancy places a greater demand on the cardiovascular system than on the respiratory system.

**Gastrointestinal System Changes**

      The **gums** may become hypertrophic and hyperemic (epulis); often, they are so spongy and friable that they bleed easily.

      Increased **salivation** (ptyalism)

**Taste** is often altered in early pregnancy

      Increased **appetite** and thirst leading to frequent snacking

      Heart burn (**pyrosis**) due to relaxation of the **cardiac sphincter** under the influence of progesterone and relaxin

      Emesis gravidarum [**NVP**] occurs in 70-80% of pregnancies.

      Decreased gastric acidity which interferes with iron absorption

      Constipation due to reduced gut motility caused by progestin

**Hepatic** synthesis of albumin, plasma globulin and fibrinogen increase

**Gall bladder** increases in size and empties more slowly

      Relaxation of the **gall bladder** wall increases the tendency of gall stone (cholelithiasis) formation

      Secretion of bile is unchanged

**Renal System Changes**

ü  During pregnancy, the length of the **kidneys** increases by 1–1.5 cm, with a proportional increase in weight.

ü  The **renal calyces and pelves** are dilated in pregnancy, with the volume of the renal pelvis increased up to 6-fold.

ü  The **ureters** are dilated above the brim of the bony pelvis, with more prominent effects on the right.

ü  The **ureters** elongate, widen, and become more curved.

ü  The entire **dilated** collecting system may contain up to 200 mL of urine, which predisposes to ascending **urinary infections**.

ü  **GFR** **increases** reaching a peak increment of 40–65% by the end of the first trimester and remains high until term.

ü  **100** **extra** liters (40-60% increase) of fluid passes through the kidneys during pregnancy

ü  There is increased **frequency** of micturition due to pressure of the **gravid uterus**on the bladder in the first trimester and pressure of the **engaging head**towards term.

**Endocrine System Changes**

v  The **anterior pituitary** gland increases in size and activity

v  The **posterior pituitary** gland remains unchanged. It releases oxytocin at the onset of labor.

v  The **thyroid gland** increases in size (in 50%) and activity

v  Most pregnant women are euthyroid

v  The **parathyroid glands** increase in size and activity to regulate calcium metabolism

v  **Adrenals** increase in size and activity and increase total cortisol levels (free cortisol levels remain the same)

v  **Progesterone** is initially produced by the corpus luteum and later by the placenta.

v  Its levels rises steadily throughout the pregnancy to a maximum of 250mg/day

v  Its **actions** during pregnancy include: reduces gut motility, causes nausea and constipation, reduces bladder and ureteric tone, causes vascular and bronchial dilatation and raises body temperature.

v  **Estrogen** is produced by the ovary in early pregnancy and then by the placenta and fetal adrenals later in pregnancy. Levels of different estrogens peak during pregnancy to between 100- and 1000-fold above normal non-pregnant values.

v  The major **actions** of estrogen during pregnancy include the following: induce growth of the uterus and development of the breasts; alter chemical composition of connective tissues of the cervix to become more pliable; causes water retention and reduce sodium excretion.

**Musculoskeletal System Changes**

Ø  Increased **lumbar** **lordosis**. Compensating for the anterior position of the enlarging uterus, lordosis shifts the center of gravity back over the lower extremities.

Ø  Increased **relaxation** of **pelvic** **joints** and **ligaments** due to **progesterone** and **relaxin** hormones.

Ø  Joint **strengthening** begins immediately following delivery and is usually complete within 3 to 5 months.

**Skin Changes**

·         Increased skin **pigmentation** due to high melanocyte stimulating hormone

·         **Linea nigra**: pigmentation of the linea alba that is more marked below the umbilicus

·         **Chloasma gravidarum**: butterfly pigmentation of the face

·         **Striae gravidarum**: stretch marks of the abdominal wall or breasts due to rupture of subcutaneous elastic fibres.

**Weight Changes**

o   There is an overall **increase** in weight of approximately **12.5Kg** on average at term.

o   There is a slight **loss** of weight during early pregnancy if the patient experiences much nausea and vomiting.

o   She then **gains** 1 to 2 Kgs by the end of the **first** trimester.

o   The **major** **increase** occurs in the second half of the pregnancy at a rate of approx. **0.5Kg/week.**

**Metabolic Changes**

§  Basal metabolic rate is increased to the extent of 30% higher than that of the average for the nonpregnant women.

§  **Carbohydrate Metabolism:**Normal pregnancy is characterized by mild fasting hypoglycemia, postprandial hyperglycemia, and hyperinsulinemia.

§  This response reflects a pregnancy-induced state of peripheral insulin resistance, which ensures a sustained postprandial supply of glucose to the fetus. The mechanisms responsible for this reduced insulin sensitivity include numerous endocrine and inflammatory factors.

§  In particular, pregnancy related hormones such as **progesterone**, placentally derived **growth** hormone, **prolactin**, and **cortisol**; cytokines such as **tumor** **necrosis** **factor**; and hormones derived from central adiposity, particularly **leptin** and its interplay with prolactin, all have a role in the insulin resistance of pregnancy.

§  **Fat Metabolism:**The concentrations of lipids, lipoproteins, and apolipoproteins in plasma rise appreciably during pregnancy.

§  Augmented lipid synthesis and food intake contribute to maternal fat accumulation during the first two trimesters. In the third trimester, however, fat storage declines or ceases. This transition to a catabolic state favors maternal use of lipids as an energy source and spares glucose and amino acids for the fetus.

§  **Protein Metabolism**: There is a**positive nitrogenous balance**throughout pregnancy.

§  At term, the fetus and the placenta contain about 500 g of protein and the maternal gain is also about 500 g chiefly distributed in the uterus, breasts and the maternal blood.

§  As the breakdown of amino acid to urea is suppressed, the blood urea level falls to 15–20 mg/dL. Blood uric acid and creatinine level, however, either remain unchanged or fall slightly.

§  Pregnancy is an **anabolic state**.

**Nervous System Changes**

      Women often report problems with **attention**, **concentration**, and **memory** throughout pregnancy and the early puerperium.

      Beginning as early as 12 weeks’ gestation and extending through the first 2 months postpartum, women have **difficulty** with **falling** **asleep**, frequent awakenings, fewer hours of night sleep, and reduced sleep efficiency.

      The **greatest** disruption of sleep is encountered **postpartum** and may contribute to postpartum **blues** or to frank **depression**.

**Psychological Changes**

ü  Maternal Emotional Responses include:

• Ambivalence

• Introversion (nervousness)

• Acceptance

• Mood Swings

• Changes in Body Image

ü  A woman’s attitude toward a pregnancy depends a great deal on psychological aspects such as the **environment** in which she was raised, the **messages** about pregnancy her family communicated to her as a child, the **society and culture**in which she lives as an adult, and whether the pregnancy has come at a good **time** or less than a good time in her life (Darby, 2007).

ü  **Social Inﬂuences:**How well a pregnant woman and her partner feel during pregnancy and childbirth is related to their cultural background, their personal experiences, and the experiences of friends and relatives, as well as that taught by childbirth educators, and the current public philosophy of childbirth.

ü  **Individual Inﬂuences:**A woman’s ability to cope with or adapt to stress plays a major role in how she will resolve conﬂict and adapt to the new life contingencies that are coming.

ü  **Family Inﬂuences:**The family in which a woman was raised can be influential to her beliefs about pregnancy because it is part of her cultural environment.

ü  **Cultural Inﬂuences:**A woman’s cultural background may strongly influence how active a role she wants to take in her pregnancy, because certain beliefs and taboos may place restrictions on her behavior and activities (Andrews & Boyle, 2007).

**Emotional Changes**

v  Emotional changes during pregnancy may include feeling uncomfortable with having a baby, feeling anxious about caring for the new baby and even negative emotions about the baby from time to time as sleep deprivation takes its toll on the new mother and father.

v  Feeling sad at times throughout pregnancy is completely normal.

v  Mood Swings occur during pregnancy because of the overwhelming amount of emotion. This is as a result of the progesterone hormone.

### Antenatal Care (Concept and Practice)

Pregnancy is a normal physiologic process; however, complications that increase the mortality or morbidity to the mother and/or fetus occur in 5–20% of pregnancies. The present system of antenatal care focuses on prevention.

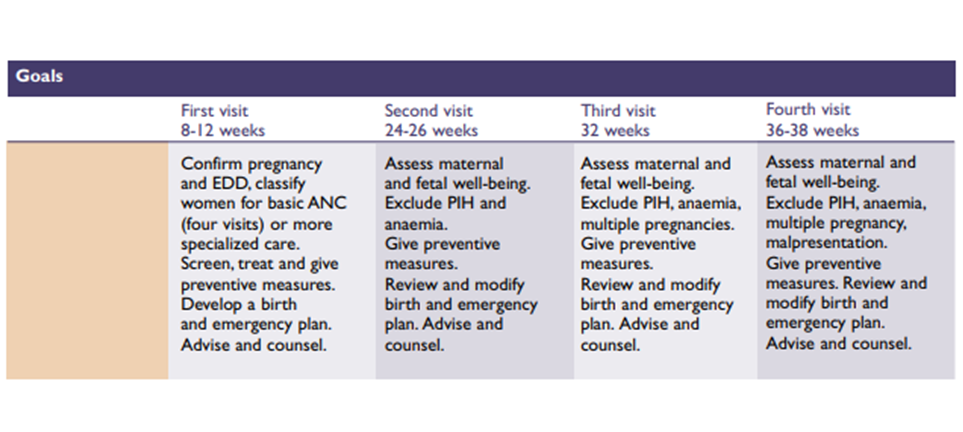
**Antenatal care (ANC)** is the medical care given to a pregnant woman from the moment she realizes she is pregnant until she delivers. The purpose of antenatal care is to ensure a successful pregnancy outcome when possible, including the delivery of a live, healthy fetus. It’s proven that mothers receiving antenatal care have a lower risk of complications, and one of the principal aims of antenatal care is the identification and special treatment of the high-risk patient—the one whose pregnancy, because of some factor in her medical history or an issue that develops during pregnancy, is likely to have a poor outcome. Antenatal care providers must be familiar with the normal changes of pregnancy and the possible pathologic changes that may occur so that therapeutic measures can be initiated to reduce any risks to the mother or fetus.

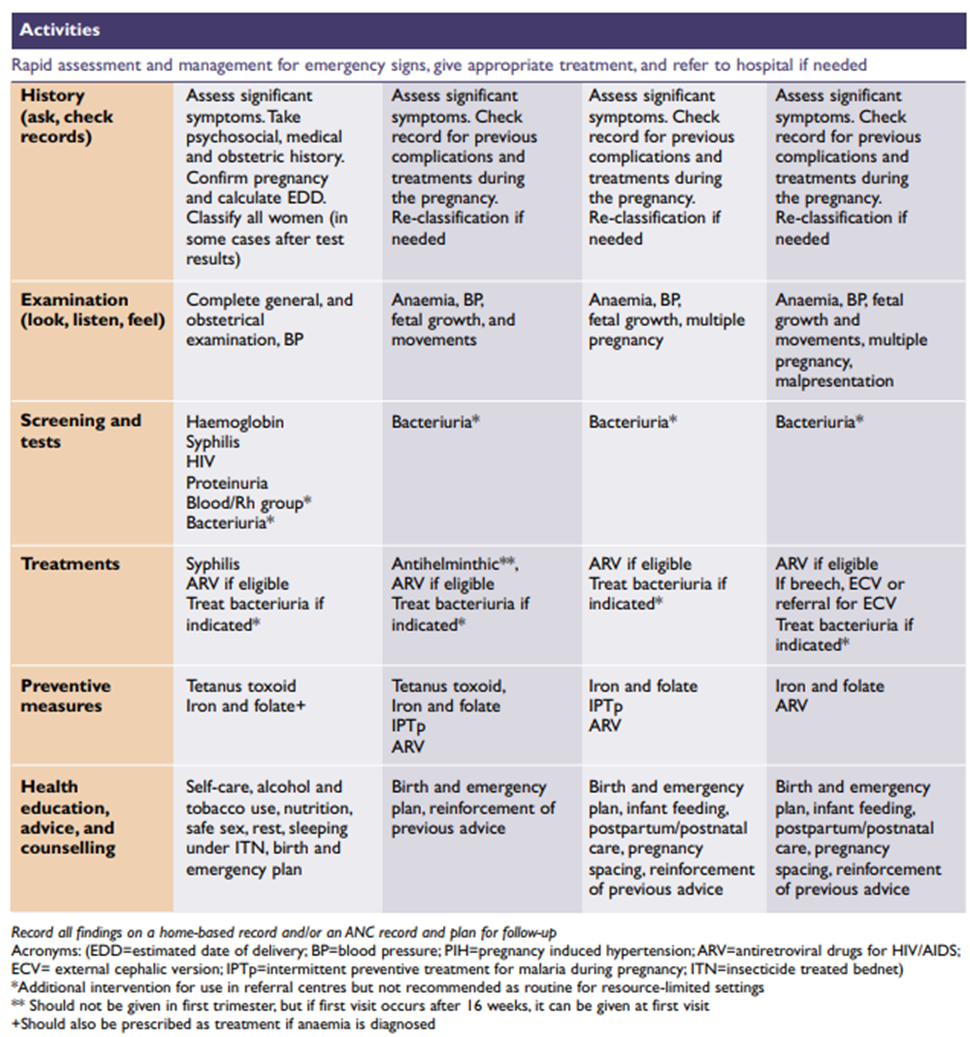
Antenatal care aims to make the woman the focus. Women should be treated with kindness and dignity at all times, and due respect given to personal, cultural and religious beliefs. Services should be readily accessible and there should be continuity of care. There is a need for high-quality, culturally appropriate, verbal and written information on which women can base their choices, through a truly informed decision-making process, which is led by them.

#### ANC Schedule Models

1.      **High risk ANC model:** The high risk approach intended to classify pregnant women as “low risk” or “high risk” based on predetermined criteria and involved many ANC visits. This approach was hard to implement effectively since many women had at least one risk factor and not all developed complications; at the same time, some low risk women did develop complications, particularly during childbirth.

2.      **Focused antenatal care (FANC) model:** Focused or goal oriented ANC services provides specific evidence-based interventions for all women, carried out at certain critical times in the pregnancy. The essential elements of this package are outlined in the boxes below:





3.      **The standard ANC model:** The frequency of office visits is dependent on the gestational age, maternal condition, and any fetal complications. The standard schedule for prenatal office visits in uncomplicated patients is every 4 weeks from 0 to 32 weeks’ gestation, every 2 weeks from 32 to 36 weeks’ gestation, and weekly visits after 36 weeks’ gestation. At each visit, maternal weight, uterine fundal height, maternal blood pressure, and urinalysis by dipstick are documented. The FHTs should be documented. All findings should be recorded and compared with those from previous visits.

4.      **Revised WHO (2016) ANC model:** Antenatal care models with a minimum of eight contacts are recommended to reduce perinatal mortality and improve women’s experience of care. The table below compares the FANC model and the revised WHO (2016) ANC model:

|  |  |
| --- | --- |
| **FANC model** | **WHO (2016) ANC model** |
| *First trimester* | |
| Visit 1: 8–12 weeks | Contact 1: up to 12 weeks |
| *Second trimester* | |
| Visit 2: 24–26 weeks | Contact 2: 20 weeks  Contact 3: 26 weeks |
| *Third trimester* | |
| Visit 3: 32 weeks Visit 4: 36–38 weeks | Contact 4: 30 weeks Contact 5: 34 weeks Contact 6: 36 weeks Contact 7: 38 weeks Contact 8: 40 weeks |
| Return for delivery at 41 weeks if not given birth. | |

**WHO RECOMMENDATIONS ON ANTENATAL CARE**

**A. Nutritional interventions**

**Dietary interventions**

**A.1.1:**Counselling about healthy eating and keeping physically active during pregnancy is recommended for pregnant women to stay healthy and to prevent excessive weight gain during pregnancy

**A.1.2:**In undernourished populations, nutrition education on increasing daily energy and protein intake is recommended for pregnant women to reduce the risk of low-birth-weight neonates.

**A.1.3:**In undernourished populations, balanced energy and protein dietary supplementation is recommended for pregnant women to reduce the risk of stillbirths and small-for-gestational-age neonates.

**A.1.4:**In undernourished populations, high-protein supplementation is not recommended for pregnant women to improve maternal and perinatal outcomes.

**Iron and folic acid supplements**

**A.2.1:**Daily oral iron and folic acid supplementation with 30 mg to 60 mg of elemental iron and 400   g (0.4 mg) of folic acid is recommended for pregnant women to prevent maternal anaemia, puerperal sepsis, low birth weight, and preterm birth.

**A.2.2:**Intermittent oral iron and folic acid supplementation with 120 mg of elemental iron and 2800   g (2.8 mg) of folic acid once weekly is recommended for pregnant women to improve maternal and neonatal outcomes if daily iron is not acceptable due to side-effects, and in populations with an anaemia prevalence among pregnant women of less than 20%.

**Calcium supplements**

**A.3:**In populations with low dietary calcium intake, daily calcium supplementation (1.5–2.0 g oral elemental calcium) is recommended for pregnant women to reduce the risk of pre-eclampsia.

**Vitamin A supplements**

**A.4:**Vitamin A supplementation is only recommended for pregnant women in areas where vitamin A deficiency is a severe public health problem, to prevent night blindness.

**Zinc supplements**

**A.5:**Zinc supplementation for pregnant women is only recommended in the context of rigorous research.

**Restricting caffeine intake**

**A.10:**For pregnant women with high daily caffeine intake (more than 300 mg per day), lowering daily caffeine intake during pregnancy is recommended to reduce the risk of pregnancy loss and low-birth-weight neonates.

**B. Maternal and fetal assessment**

**B.1: Maternal assessment**

**Anaemia**

**B.1.1:**Full blood count testing is the recommended method for diagnosing anaemia in pregnancy. In settings where full blood count testing is not available, on-site haemoglobin testing with a haemoglobinometer is recommended over the use of the haemoglobin colour scale as the method for diagnosing anaemia in pregnancy.

**Asymptomatic bacteriuria (ASB)**

**B.1.2:**Midstream urine culture is the recommended method for diagnosing asymptomatic bacteriuria (ASB) in pregnancy. In settings where urine culture is not available, on-site midstream urine Gram staining is recommended over the use of dipstick tests as the method for diagnosing ASB in pregnancy.

**Intimate partner violence (IPV)**

**B.1.3:**Clinical enquiry about the possibility of intimate partner violence (IPV) should be strongly considered at antenatal care visits when assessing conditions that may be caused or complicated by IPV in order to improve clinical diagnosis and subsequent care, where there is the capacity to provide a supportive response (including referral where appropriate) and where the WHO minimum requirements are met.

**Gestational diabetes mellitus (GDM)**

**B.1.4:**Hyperglycaemia first detected at any time during pregnancy should be classified as either gestational diabetes mellitus (GDM) or diabetes mellitus in pregnancy, according to WHO criteria.

**Tobacco use**

**B.1.5:**Health-care providers should ask all pregnant women about their tobacco use (past and present) and exposure to second-hand smoke as early as possible in the pregnancy and at every antenatal care visit.

**Substance use**

**B.1.6:**Health-care providers should ask all pregnant women about their use of alcohol and other substances (past and present) as early as possible in the pregnancy and at every antenatal care visit.

**Human immune deficiency virus (HIV) and syphilis**

**B.1.7:**In high-prevalence settings, provider-initiated testing and counselling (PITC) for HIV should be considered a routine component of the package of care for pregnant women in all antenatal care settings. In low-prevalence settings, PITC can be considered for pregnant women in antenatal care settings as a key component of the effort to eliminate mother-to-child transmission of HIV, and to integrate HIV testing with syphilis, viral or other key tests, as relevant to the setting, and to strengthen the underlying maternal and child health systems.

**Tuberculosis (TB)**

**B.1.8:**In settings where the tuberculosis (TB) prevalence in the general population is 100/100 000 population or higher, systematic screening for active TB should be considered for pregnant women as part of antenatal care.

**B.2: Fetal assessment**

**Daily fetal movement counting**

**B.2.1:**Daily fetal movement counting, such as with “count-to-ten” kick charts, is only recommended in the context of rigorous research.

**Symphysis-fundal height (SFH) measurement**

**B.2.2:**Replacing abdominal palpation with symphysis-fundal height (SFH) measurement for the assessment of fetal growth is not recommended to improve perinatal outcomes. A change from what is usually practiced (abdominal palpation or SFH measurement) in a particular setting is not recommended.

**Ultrasound scan**

**B.2.4:**One ultrasound scan before 24 weeks of gestation (early ultrasound) is recommended for pregnant women to estimate gestational age, improve detection of fetal anomalies and multiple pregnancies, reduce induction of labour for post-term pregnancy, and improve a woman’s pregnancy experience.

**C. Preventive measures**

**Antibiotics for asymptomatic bacteriuria (ASB)**

**C.1:**A seven-day antibiotic regimen is recommended for all pregnant women with asymptomatic bacteriuria (ASB) to prevent persistent bacteriuria, preterm birth and low birth weight.

**Antibiotic prophylaxis to prevent recurrent urinary tract infections**

**C.2:**Antibiotic prophylaxis is only recommended to prevent recurrent urinary tract infections in pregnant women in the context of rigorous research.

**Antenatal anti-D immunoglobulin administration**

**C.3:**Antenatal prophylaxis with anti-D immunoglobulin in non-sensitized Rh-negative pregnant women at 28 and 34 weeks of gestation to prevent RhD alloimmunization is only recommended in the context of rigorous research.

**Preventive anthelminthic treatment**

**C.4:**In endemic areas, preventive anthelminthic treatment is recommended for pregnant women after the first trimester as part of worm infection reduction programmes.

**Tetanus toxoid vaccination**

**C.5:**Tetanus toxoid vaccination is recommended for all pregnant women, depending on previous tetanus vaccination exposure, to prevent neonatal mortality from tetanus.

**Malaria prevention: intermittent preventive treatment in pregnancy (IPTp)**

**C.6:**In malaria-endemic areas in Africa, intermittent preventive treatment with sulfadoxine-pyrimethamine (IPTp-SP) is recommended for all pregnant women. Dosing should start in the second trimester, and doses should be given at least one month apart, with the objective of ensuring that at least three doses are received.

**Pre-exposure prophylaxis (PrEP) for HIV prevention**

**C.7:**Oral pre-exposure prophylaxis (PrEP) containing tenofovir disoproxil fumarate (TDF) should be offered as an additional prevention choice for pregnant women at substantial risk of HIV infection as part of combination prevention approaches.

**D. Interventions for common physiological symptoms**

**Nausea and vomiting**

**D.1:**Ginger, chamomile, vitamin B6 and/or acupuncture are recommended for the relief of nausea in early pregnancy, based on a woman’s preferences and available options.

**Heartburn**

**D.2:**Advice on diet and lifestyle is recommended to prevent and relieve heartburn in pregnancy. Antacid preparations can be offered to women with troublesome symptoms that are not relieved by lifestyle modification.

**Leg cramps**

**D.3:**Magnesium, calcium or non-pharmacological treatment options can be used for the relief of leg cramps in pregnancy, based on a woman’s preferences and available options.

**Low back and pelvic pain**

**D.4:**Regular exercise throughout pregnancy is recommended to prevent low back and pelvic pain. There are a number of different treatment options that can be used, such as physiotherapy, support belts and acupuncture, based on a woman’s preferences and available options.

**Constipation**

**D.5:**Wheat bran or other fibre supplements can be used to relieve constipation in pregnancy if the condition fails to respond to dietary modification, based on a woman’s preferences and available options.

**Varicose veins and oedema**

**D.6:**Non-pharmacological options, such as compression stockings, leg elevation and water immersion, can be used for the management of varicose veins and oedema in pregnancy, based on a woman’s preferences and available options.

**ELIMINATION OF MOTHER TO CHILD TRANSMISSION OF HIV AND SYPHILIS**

Mother-to-child transmission (MTCT) of HIV is a significant contributor to the HIV pandemic, accounting for 9% of new infections globally. The Joint United Nations Programme on HIV/AIDS (UNAIDS) reported that in 2016 an estimated 160 000 children were newly infected with HIV, and an estimated 3.1 million children were living with HIV globally. Although this is still a large number of new infections, at the peak of the HIV epidemic, there were close to 500 000 children infected with HIV through MTCT each year.

MTCT of HIV occurs when HIV is transmitted from a woman living with HIV to her baby during pregnancy, labour or delivery, or after delivery through breastfeeding. Without treatment, approximately 15–30% of infants born to HIV-positive women will become infected with HIV during gestation and delivery, with a further 5–15% becoming infected through breastfeeding. HIV infection of infants results in early mortality for many or creates a lifelong chronic condition that greatly shortens life expectancy and contributes to substantial human, social and economic costs.

Globally, an estimated 1.3 million women living with HIV become pregnant every year. Primary prevention of HIV, prevention of unintended pregnancies, effective access to HIV testing and counselling, initiation of lifelong antiretroviral therapy (ART) with support for adherence, retention and viral suppression for mothers living with HIV, safe delivery practices, optimal infant feeding practices and access to postnatal antiretroviral (ARV) prophylaxis for infants all contribute to the prevention of mother-to-child transmission (PMTCT), thereby reducing maternal and child mortality. With the global shift to highly effective and simplified interventions based on lifelong maternal ART, it is now feasible to virtually eliminate new HIV infections in infants, while assuring the health of the mother.

In 2012, WHO estimated that over 900 000 pregnant women were infected with syphilis. These maternal infections resulted in more than 350 000 estimated adverse pregnancy outcomes, over 200 000 of which were stillbirths or neonatal deaths. Syphilis is caused by the Treponema pallidum bacterium, renowned for its invasiveness. It can be transmitted via sexual exposure or vertically from mother to child early in pregnancy (in utero infection). If the infection remains untreated, adverse pregnancy outcomes are frequent. Indeed, over half of the pregnancies among women with active syphilis will result in stillbirth, early neonatal death, a preterm or low-birth-weight infant, or serious neonatal infection. Screening for maternal syphilis early in pregnancy and prompt treatment of seropositive mothers with intramuscular benzathine benzylpenicillin, a long-acting penicillin, cures syphilis in both mother and infant, and prevents most complications associated with MTCT of syphilis.

Dual elimination serves to improve a broad range of maternal and child health (MCH) services and outcomes. This achievement directly contributes to Sustainable Development Goals (SDGs) 3, 5 and 10, which aspire to ensure health and well-being for all, achieve gender equality and empower women and girls, and reduce inequalities in access to services and commodities. Additionally, the similarity of the control interventions necessary to prevent transmission of HIV and syphilis in pregnancy adds to the feasibility and benefit of such an integrated approach to the elimination of MTCT (EMTCT) of both infections.

The following strategies are important components of successful EMTCT programmes:

• interruption of transmission through quality ANC and prevention services that provide timely identification and treatment of pregnant women infected with HIV or syphilis, their sexual partners, and their infants;

ü  reduction in the number of HIV and/or syphilis infections among pregnant women through:  
prevention of HIV and/or syphilis infection in women of reproductive age, including in HIV negative pregnant and breastfeeding women and their sexual partners;

ü  promotion of a healthy reproductive life, including prevention of unintended pregnancies and support for safer conception among women with known HIV infection;

ü  control of HIV and syphilis in the general and key populations (including sex workers, drug users, men who have sex with men) to decrease prevalence.

• promotion and protection of the human rights and gender equality of women living with HIV;  
• greater engagement of women living with HIV in HIV programming, decision-making and service delivery.

**SPECIFIC EMTCT MEASURES**

**Guidelines on infant feeding for mothers living with HIV**

WHO bases its recommendations on infant feeding for mothers living with HIV on the comparative risk of infants acquiring HIV through breastfeeding with the increased risk of infants dying from illnesses such as malnutrition, diarrhea and pneumonia, which increases if they are not breastfed.

In 2016, WHO released guidelines recommending that mothers living with HIV who are on treatment and are being fully supported to adhere to it should exclusively breastfeed their infants for the first six months of life, then introduce appropriate complementary foods while continuing to breastfeed for at least 12 months and up to 24 months or longer (similar to the general population).

When ARV drugs are not immediately available, the WHO guidelines still recommend mothers exclusively breastfeed for the first six months of an infant’s life and continue, unless environmental and social circumstances are safe for, and supportive of, replacement feeding. This decision should be based on international recommendations and consideration of:

* the socioeconomic and cultural contexts of the population groups served by maternal and child health services
* the availability and quality of health services
* the local epidemiology (which diseases are common and who they affect), including HIV prevalence among pregnant women
* the main causes of under-nutrition among mothers and children, and infant and child mortality.

When ARV drugs are unlikely to be available, such as in acute emergencies, mothers living with HIV are still recommended to breastfeed their infants to increase their chances of survival.

Currently, most high-income countries recommend women living with HIV do not breastfeed whether they are virally suppressed or not. This is because formula feed and clean, boiled water are widely accessible. So any risks around dirty water or malnutrition have been eliminated. In low- and middle-income countries this risk is far greater, leading WHO’s advice on infant feeding to differ.

**Guidelines for HIV-exposed infants**

If an HIV-exposed infant is given ART within the first 12 weeks of life, they are 75% less likely to die from an AIDS-related illness. The treatment should be linked to the mother's course of ARV drugs and would vary according to the infant feeding method as follows:

* **breastfeeding:** the infant should receive once-daily nevirapine from birth for six weeks
* **replacement feeding:** the infant should receive once-daily nevirapine (or twice-daily zidovudine) from birth for four to six weeks.

This is one of the reasons WHO recommends that infants born to mothers living with HIV are tested between four and six weeks old. This is often referred to as ‘early infant diagnosis’.

WHO further recommends that another HIV test is carried out at 18 months and/or when breastfeeding ends to provide the final infant diagnosis. As proportionally more infant infections are now occurring during breastfeeding these tests are becoming increasingly important.

According to WHO guidelines, all infants who test positive for HIV should be immediately initiated on treatment (full HAART).

## unit six content

### Topic 1: Ectopic Pregnancy

**ECTOPIC PREGNANCY**

Ectopic pregnancy refers to the implantation of a fertilized egg in a location outside of the uterine cavity, including the fallopian tubes (approximately 97.7%), cervix, ovary, cornual region of the uterus, and abdominal cavity. Of tubal pregnancies, the ampulla is the most common site of implantation (80%), followed by the isthmus (12%), fimbria (5%), cornua (2%), and interstitia (2-3%).

Ectopic pregnancy is the result of a flaw in human reproductive physiology that allows the conceptus to implant and mature outside the endometrial cavity, which ultimately ends in the death of the fetus. Without timely diagnosis and treatment, ectopic pregnancy can become a life-threatening situation. As the gestation enlarges, it creates the potential for organ rupture, because only the uterine cavity is designed to expand and accommodate fetal development. [Ectopic pregnancy](http://emedicine.medscape.com/article/104382-overview) can lead to massive hemorrhage, infertility, or death

**Etiology**

An ectopic pregnancy requires the occurrence of 2 events: fertilization of the ovum and abnormal implantation. Many risk factors affect both events; for example, a history of major tubal infection decreases fertility and increases abnormal implantation.

Multiple factors contribute to the relative risk of ectopic pregnancy. In theory, anything that hampers or delays the migration of the fertilized ovum (blastocyst) to the endometrial cavity can predispose a woman to ectopic gestation. The following risk factors have been linked to ectopic pregnancy:

1. Tubal damage - Which can be the result of infections such as [pelvic inflammatory disease](http://emedicine.medscape.com/article/256448-overview) (PID) or salpingitis (whether documented or not) or can result from abdominal surgery or tubal ligation or from maternal in utero diethylstilbestrol (DES) exposure
2. History of previous ectopic pregnancy
3. Smoking - A risk factor in about one third of ectopic pregnancies; smoking may contribute to decreased tubal motility by damage to the ciliated cells in the fallopian tubes
4. Altered tubal motility - As mentioned, this can result from smoking, but it can also occur as the result of hormonal contraception; progesterone-only contraception and progesterone intrauterine devices (IUDs) have been associated with an increased risk of ectopic pregnancy
5. History of 2 or more years of infertility (whether treated or not)- Women using assisted reproduction seem to have a doubled risk of ectopic pregnancy (to 4%), although this is mostly due to the underlying infertility
6. History of multiple sexual partners
7. Maternal age - Although this is not an independent risk factor

**Clinical Presentation**

The classic clinical triad of ectopic pregnancy is pain, amenorrhea, and vaginal bleeding; unfortunately, only about 50% of patients present with all 3 symptoms. About 40-50% of patients with an ectopic pregnancy present with vaginal bleeding, 50% have a palpable adnexal mass, and 75% may have abdominal tenderness. In one case series of ectopic pregnancies, abdominal pain presented in 98.6% of patients, amenorrhea in 74.1% of them, and irregular vaginal bleeding in 56.4% of patients.

Patients may present with other symptoms common to early pregnancy, including nausea, breast fullness, fatigue, low abdominal pain, heavy cramping, shoulder pain, and recent dyspareunia. Painful fetal movements (in the case of advanced abdominal pregnancy), dizziness or weakness, fever, flulike symptoms, vomiting, syncope, or cardiac arrest have also been reported. Shoulder pain may be reflective of peritoneal irritation.

The physical examination of patients with ectopic pregnancy is highly variable and often unhelpful. Patients frequently present with benign examination findings, and adnexal masses are rarely found. Patients in hemorrhagic shock from ruptured ectopic may not be tachycardic.

Some physical findings that have been found to be predictive (although not diagnostic) for ectopic pregnancy include the following:

* Presence of peritoneal signs
* Cervical motion tenderness
* Unilateral or bilateral abdominal or pelvic tenderness - Usually much worse on the affected side

Abdominal rigidity, involuntary guarding, and severe tenderness, as well as evidence of hypovolemic shock, such as orthostatic blood pressure changes and tachycardia, should alert the clinician to a surgical emergency; this may occur in up to 20% of cases. However, midline abdominal tenderness or a uterine size of greater than 8 weeks on pelvic examination decreases the risk of ectopic pregnancy.

On pelvic examination, the uterus may be slightly enlarged and soft, and uterine or cervical motion tenderness may suggest peritoneal inflammation. An adnexal mass may be palpated but is usually difficult to differentiate from the ipsilateral ovary.

The presence of uterine contents in the vagina, which can be caused by shedding of endometrial lining stimulated by an ectopic pregnancy, may lead to a misdiagnosis of an incomplete or complete abortion and therefore a delayed or missed diagnosis of ectopic pregnancy.

**Diagnosis**

***Serum β-HCG levels***

In a normal pregnancy, the β-HCG level doubles every 48-72 hours until it reaches 10,000-20,000mIU/mL. In ectopic pregnancies, β-HCG levels usually increase less. Mean serum β-HCG levels are lower in ectopic pregnancies than in healthy pregnancies.

No single serum β-HCG level is diagnostic of an ectopic pregnancy. Serial serum β-HCG levels are necessary to differentiate between normal and abnormal pregnancies and to monitor resolution of ectopic pregnancy once therapy has been initiated.

The discriminatory zone of β-HCG (ie, the level above which an imaging scan should reliably visualize a gestational sac within the uterus in a normal intrauterine pregnancy) is as follows:

* 1500-1800 mIU/mL with transvaginal ultrasonography, but up to 2300 mIU/mL with multiple gestates
* 6000-6500 mIU/mL with abdominal ultrasonography

Absence of an intrauterine pregnancy on a scan when the β-HCG level is above the discriminatory zone represents an ectopic pregnancy or a recent abortion.

***Ultrasonography***

Ultrasonography is probably the most important tool for diagnosing an extrauterine pregnancy.

Visualization of an intrauterine sac, with or without fetal cardiac activity, is often adequate to exclude ectopic pregnancy.

Transvaginal ultrasonography, or endovaginal ultrasonography, can be used to visualize an intrauterine pregnancy by 24 days post ovulation or 38 days after the last menstrual period (about 1 week earlier than transabdominal ultrasonography). An empty uterus on endovaginal ultrasonographic images in patients with a serum β-HCG level greater than the discriminatory cut-off value is an ectopic pregnancy until proved otherwise.

Color-flow Doppler ultrasonography improves the diagnostic sensitivity and specificity of transvaginal ultrasonography, especially in cases in which a gestational sac is questionable or absent.

***Laparoscopy***

Laparoscopy remains the criterion standard for diagnosis; however, its routine use on all patients suspected of ectopic pregnancy may lead to unnecessary risks, morbidity, and costs. Moreover, laparoscopy can miss up to 4% of early ectopic pregnancies.

Laparoscopy is indicated for patients who are in pain or hemodynamically unstable.

**Management**

Therapeutic options in ectopic pregnancy are as follows:

* Expectant management
* Methotrexate
* Surgery

***Expectant management***

Candidates for successful expectant management should be asymptomatic and have no evidence of rupture or hemodynamic instability. Candidates should demonstrate objective evidence of resolution (eg, declining β-HCG levels).

Close follow-up and patient compliance are of paramount importance, as tubal rupture may occur despite low and declining serum levels of β-HCG.

***Methotrexate***

Methotrexate is the standard medical treatment for unruptured ectopic pregnancy. A single-dose IM injection is the more popular regimen. The ideal candidate should have the following:

* Hemodynamic stability
* No severe or persisting abdominal pain
* The ability to follow up multiple times
* Normal baseline liver and renal function test results

Absolute contraindications to methotrexate therapy include the following:

* Existence of an intrauterine pregnancy
* Immunodeficiency
* Moderate to severe anemia, leukopenia, or thrombocytopenia
* Sensitivity to methotrexate
* Active pulmonary or peptic ulcer disease
* Clinically important hepatic or renal dysfunction
* Breastfeeding
* Evidence of tubal rupture

***Surgical treatment***

**Conservative Surgery:**The procedure can be done either **laparoscopically or by microsurgical  
laparotomy**.  
**Indications:**(a) Cases not fulfilling the criteria of medical therapy. (b) Cases where b-hCG levels are not decreasing despite medical therapy. (c) persistent fetal cardiac activity.

1. **Linear Salpingostomy**: A longitudinal incision is made on the antimesenteric border directly over the site of ectopic pregnancy. After removing the products (by fingers, scalpel handle or by suction), the incision line is kept open to be healed later on by secondary intention. Hemostasis is achieved by electrocautery or laser.

2. **Linear Salpingotomy**: The procedures are the same as those of salpingostomy. But the incision line is closed in two layers with 7-0 interrupted vicryl sutures. This is not commonly done.

3. **Segmental Resection**: This is of choice in isthmic pregnancy. End-to-end anastomosis can be done immediately or at a later date after appropriate counseling of the patient.

4. **Fimbrial Expression:**This is ideal in cases of distal ampullary (fimbrial) pregnancy and is done digitally.

**Salpingectomy**is done when (i) whole of the affected tube is damaged, (ii) contralateral tube is normal or (iii) future fertility is not desired.

Following conservative surgery or medical treatment, estimation of b-hCG should be done weekly  
till the value becomes less than 5.0 mlU/mL. Additional monitoring by TVS is preferred. Following laparoscopic salpingostomy, persistent ectopic pregnancy ranges between 4% and 20%.

**Persistent ectopic pregnancy**is due to incomplete removal of trophoblast. It is high after fimbrial  
expression and in cases where initial serum b-hCG level is greater than 3,000 IU/L. Prophylactic single dose MTX (1 mg/kg) IM is effective to resolve the problem.

**GESTATIONAL TROPHOBLASTIC DISEASES (GTDs)**

Gestational trophoblastic disease (GTD) refers to a spectrum of interrelated but histologically distinct tumors originating from the placenta. These diseases are characterized by a reliable tumor marker, which is the β-subunit of human chorionic gonadotropin (β-hCG), and have varied tendencies or local invasion and spread.

Gestational trophoblastic neoplasia (GTN) refers to the subset of GTD that develops malignant sequelae. These tumors require normal staging and typically respond favorably to chemotherapy. Most commonly, GTN develops after a molar pregnancy but may follow any gestation. The prognosis or most GTN cases is excellent, and patients are routinely cured, even with widespread  
metastases. The outlook or preservation of fertility and or successful subsequent pregnancy outcomes is equally bright. Accordingly, although GTD is uncommon, because the opportunity or cure is great, clinicians should be familiar with its presentation, diagnosis, and management.

**Classification of GTD**

1. Hydatidiform mole: – Complete – Partial
2. Invasive mole
3. Placental site trophoblastic tumor (PSTT)
4. Choriocarcinoma
5. Epithelioid trophoblastic tumor (ETT)

**RISK FACTORS**

·         Maternal age at the upper and lower extremes carries a higher risk of GTD. This association is much greater for complete moles, whereas the risk of partial molar pregnancy varies relatively little with age.

·         A history of prior unsuccessful pregnancies also increases the risk of GTD. For example, previous spontaneous abortion at least doubles the risk of molar pregnancy. More significantly, a personal history of GTD increases the risk of developing a molar gestation in a subsequent pregnancy at least 10-fold.

·         Prior COC use approximately doubles the risk, and longer duration of use also correlates positively with risk.

·         Some epidemiologic characteristics differ markedly between complete and partial moles. For example, vitamin A deficiency and low dietary intake of carotene are associated only with an increased risk of complete moles. Partial moles have been linked to higher educational levels, smoking, irregular menstrual cycles, and obstetric histories in which only male infants are among the prior live births. Many of these associations, however, are weak and could be explained by confounding factors other than causality.

**HYDATIDIFORM MOLE (MOLAR PREGNANCY)**

Hydatidiform moles are abnormal pregnancies characterized histologically by aberrant changes within the placenta. Classically, the chorionic villi in these placenta show varying degrees of trophoblast proliferation and edema o the stroma within villi. Hydatidiform moles are categorized as either *complete hydatidiform moles*or *partial hydatidiform moles*. Chromosomal abnormalities play an integral role in hydatidiform mole development.

**Complete Hydatidiform Mole**

These molar pregnancies differ from partial moles with regard to their karyotype, their histologic appearance, and their clinical presentation. First, complete moles typically have a diploid karyotype, and 85 to 90 percent of cases are 46,XX. The chromosomes, however, in these pregnancies are entirely of paternal origin, and thus, the diploid set is described as *diandric*. Specifically, complete moles are formed by *androgenesis*, in which the ovum is fertilized by a haploid sperm that then duplicates its own chromosomes after meiosis. The ovum fails to contribute chromosomes. Most of these moles are 46,XX, but dispermic fertilization of a single ovum, that is, simultaneous fertilization by two sperm, can produce a 46,XY karyotype.

Microscopically, complete moles display enlarged, edematous villi and abnormal trophoblastic proliferation. These changes diffusely involve the entire placenta. Macroscopically, these changes transform the chorionic villi into clusters of vesicles with variable dimensions. Indeed, the name *hydatidiform mole*literally stems from this “bunch of grapes” appearance. In these pregnancies, no fetal tissue or amnion is produced. As a result, this mass of placental tissue completely fills the endometrial cavity.

More than half of affected patients present with anemia and uterine sizes in excess of that predicted or their gestational age. In addition, hyperemesis gravidarum, preeclampsia, and theca-lutein cysts develop in approximately one quarter of women. These cysts range in size from 3 to 20 cm, and most regress with falling β-hCG titers after molar evacuation. If such cysts are present, and especially if they are bilateral, the risk of postmolar GTN is increased.

Vaginal bleeding remains the most common presenting symptom, and β-hCG levels are often greater than expected.

**Partial Hydatidiform Mole**

These moles differ from complete hydatidiform moles clinically, genetically, and histologically. The degree and extent of trophoblastic proliferation and villous edema are decreased compared with those of complete moles. Moreover, most partial moles contain fetal tissue and amnion, in addition to placental tissues. As a result, patients with partial moles typically  
present with signs and symptoms of an incomplete or missed abortion. Many women will have vaginal bleeding. However, because trophoblastic proliferation is slight and only focal, uterine enlargement in excess of gestational age is uncommon. Similarly, preeclampsia, theca-lutein cysts, hyperthyroidism, or other dramatic clinical features are rare.

Preevacuation β-hCG levels are typically much lower than those or complete moles and o ten do not exceed 100,000 mIU/mL. For this reason, partial moles are o ten not identified until after a histologic review of a curettage specimen.

Partial moles have a triploid karyotype (69, XXX, 69,XXY, or less commonly 69,XYY) that is composed of one maternal and two paternal haploid sets of chromosomes. The coexisting fetus present with a partial mole is nonviable and typically has multiple malformations with abnormal growth.

**Clinical Findings**

**A. Symptoms & Signs**

Abnormal uterine bleeding, usually during the first trimester, is the most common presenting symptom, occurring in more than 90% of patients with molar pregnancies. Three-fourths of these patients present before the end of the first trimester. Nausea and vomiting have been reported in 14–32% of patients with hydatidiform mole and may be confused with nausea and vomiting of pregnancy or hyperemesis gravidarum. Ten percent of these patients may have nausea and vomiting severe enough to require hospitalization. About half of patients will have a uterine size that is greater than expected for their gestational age.

However, in one-third of patients, the uterus may be smaller than expected. Multiple theca lutein cysts causing enlargement of one or both ovaries are seen in 15–30% of women with molar pregnancies. In about half of these cases, both ovaries are enlarged and may be a source of pain. Involution of the cysts proceeds over several weeks and usually parallels the decline of hCG values. In studies, patients with theca lutein cysts appear to have a greater likelihood of developing malignant sequelae of gestational trophoblastic neoplasia.

Preeclampsia in the first trimester or early second trimester—an unusual finding in normal pregnancies—has been said to be pathognomonic for a molar pregnancy. Hyperthyroidism from stimulation of thyrotropin receptors by hCG can also occur in 10% of patients, although the disease is usually subclinical, and most patients remain asymptomatic. Treatment involves evacuation of the mole. An occasional patient may require brief antithyroid therapy.

**B. Laboratory Findings**

The principal characteristic of gestational trophoblastic neoplasms is their capacity to produce hCG. This hormone may be detected in the serum or urine of virtually all patients with hydatidiform mole or malignant trophoblastic disease, and its levels correlate closely with the presence of viable tumor cells. Consequently, monitoring of hCG levels is a necessary tool for the diagnosis, treatment, and surveillance of the disease process.

The usefulness of a serum gonadotropin assay depends on the hCG titer and the sensitivity of the test. Today, sensitive and specific immunoassays are available to differentiate hCG from luteinizing hormone by measuring the β chain of hCG. Serial β-hCG levels are best monitored in the same laboratory using the same immunoassay technique.

The rate of decline in hCG titers is also important. Normal postmolar pregnancy hCG regression curves highlighting the weekly hCG levels in patients undergoing spontaneous remission have been constructed, hence providing a reference for the comparison of random or serial values. In most instances, the hCG values exhibit a progressive decline to non-detectable levels within 14 weeks after evacuation of a molar pregnancy. If the hCG titer rises or plateaus, it must be concluded that viable tumor continues to persist. If the levels of hCG are very low and not responsive to treatment, a false positive hCG result or “phantom hCG,” caused by cross-reaction of heterophilic antibodies with the hCG test, should be considered.

**C. Ultrasonographic Findings**

The simplicity, safety, and reliability of ultrasonography define it as the diagnostic method of choice for patients with suspected molar pregnancy. In a complete molar pregnancy, the characteristic ultrasound pattern consists of multiple hypoechoic areas corresponding to hydropic villi, at times described as a “snowstorm” pattern. A normal gestational sac or fetus is not present. Theca lutein cysts may be visualized. In a partial mole, focal areas of trophoblastic changes and fetal tissue may be noted. Focal cystic changes in the placenta are also a hallmark finding. On the other hand, an ultrasonogram of a choriocarcinoma may reveal an enlarged uterus with a necrotic and hemorrhagic pattern, whereas that of PSTT may show an intrauterine mass.

**Complications**

The maternal–fetal barrier contains leaks large enough to permit passage of cellular and tissue elements. As a result, deportations of trophoblastic tissue to the lungs are frequent. Spontaneous regression of these ectopic trophoblastic tissues can occur. Less commonly, this results in a syndrome of acute pulmonary insufficiency. Symptoms of dyspnea and cyanosis, due to massive deportation of trophoblasts to the pulmonary vasculature and subsequent formation of pulmonary emboli, can present within 4–6 hours after evacuation of a molar pregnancy. Pulmonary edema leading to high-output congestive heart failure may complicate excessive fluid administration, preeclampsia, anemia, or hyperthyroidism.

**Treatment**

After the diagnosis has been confirmed, blood type, hematocrit, and thyroid, liver, and renal function tests should be obtained. A chest radiograph can rule out metastasis to the lungs. Subsequently, the molar pregnancy should be terminated. Suction curettage under general anesthesia is the method of choice once the patient is deemed stable. This can be safely accomplished even when the uterus is the size of a 28-week gestation. Local or regional anesthesia may be an option for the stable, cooperative patient with a small uterus. Intravenous oxytocin should be administered after dilation of the cervix but before the start of evacuation and may be continued, if necessary, for 24 hours post-evacuation. Tissue should be submitted for pathologic study. Blood loss usually is moderate, but precautions should be taken for the possibility of hemorrhage requiring a transfusion.

When a large hydatidiform mole (>12 weeks in size) is evacuated by suction curettage, a laparotomy setup should be readily available, as hysterotomy, hysterectomy, or bilateral hypogastric artery ligation may be necessary if perforation or hemorrhage occurs. After the completion of the evacuation, all Rh-negative patients should receive Rh immune globulin. Hysterectomy continues to remain an option for good surgical candidates not desirous of future pregnancy and for older women (who are more likely to develop malignant sequelae). If theca lutein cysts are encountered at laparotomy, the ovaries should remain intact, as regression to normal size will occur with diminishing hCG titers. Surgical treatment of these cysts is indicated only if rupture, torsion, or hemorrhage occurs or if the enlarged ovaries become infected.

**Follow-up**

Despite earlier diagnosis of molar pregnancies, the incidence of persistent gestational trophoblastic disease has not decreased. Three-fourths of patients with malignant nonmetastatic trophoblastic disease and half of patients with malignant metastatic disease develop these tumors following a hydatidiform mole. In the remainder, disease arises subsequent to a term pregnancy, abortion, or ectopic pregnancy. Several clinical features of hydatidiform moles are recognized as having a high association with malignant trophoblastic neoplasia. In general, at diagnosis, the larger the uterus and the higher the hCG titer, the greater the risk for malignant gestational trophoblastic disease. The combination of theca lutein cysts and uterine size excessive for gestational age is associated with an extremely high risk of malignant sequelae. Pathologic specimens with marked nuclear atypia, necrosis, hemorrhage, or trophoblastic proliferation may also increase the risk of persistent disease.

Regardless of the method of termination (suction curettage or hysterectomy) or presence of high risk features, close monitoring with serial hCG titers is essential for every patient, as the incidence of malignant sequelae approaches 20–30%. After evacuation of the molar pregnancy, the patient should undergo serial hCG determinations, beginning within 48 hours after evacuation and then at weekly intervals until hCG values decline to undetectable levels (<5 mIU per milliliter) on three successive assays. If titer remission occurs spontaneously within 14 weeks and without a titer plateau, the hCG titer should then be repeated monthly for at least 6 months to 1 year before the patient is released from close medical supervision. Thereafter, the patient may enter into a routine gynecologic care program.

A gynecologic examination should be done 1 week after evacuation, at which time blood may be taken for the hCG titer. Estimates of uterine size, presence of adnexal masses (theca lutein cysts) and presence of vulvar, vaginal, or cervical lesions should be noted. Unless symptoms develop, the examination may be repeated at 4-week intervals throughout the observation period. If pre-evacuation chest radiography has revealed pulmonary metastases, chest radiographs should be repeated at 4-week intervals until spontaneous remission is confirmed, then at 3-month intervals during the remainder of the surveillance period.

Effective contraceptive measures should be implemented and maintained throughout the period of surveillance. Studies have not shown an increased risk of persistent gestational trophoblastic neoplasia after a molar pregnancy with the use of oral contraceptives. Therefore, they remain the most widely used method of birth control. A patient who has entered into spontaneous remission with negative titers, examinations, and chest radiographs for 6 months to 1 year and who is desirous of becoming pregnant may terminate contraceptive practices. Successful pregnancy is the norm, and complications are similar to those of the general population.

 Abortion/Miscarriage

**ABORTION**

An abortion is the spontaneous or induced loss of an early pregnancy. The period of pregnancy prior to fetal viability outside of the uterus is considered early pregnancy. Most (following the WHO definition of abortion) consider early pregnancy to end at 20 weeks' gestation or when the fetus weighs 500 grams. In Kenya and most of sub-Saharan Africa, the age of fetal viability is 28 weeks. Therefore, our definition of abortion is ‘the loss of a pregnancy before 28 complete weeks of gestation.’

**Spontaneous abortion (commonly termed miscarriage in layman language)**is the most common complication of pregnancy and is defined as the passing of a pregnancy at less than 28 weeks of gestation. It implies the spontaneous loss of an embryo or fetus weighing less than 500 g. **Threatened abortion**is bleeding arising from within the uterus that occurs before the 28th completed week in a viable pregnancy. The patient may or may not experience pain or cramping; however, there is no passage of products of conception and no cervical dilation. **Complete abortion**is the expulsion of all of the products of conception before the 28th completed week of gestation, whereas **incomplete abortion**is the expulsion of some, but not all, of the products of conception. **Inevitable abortion**refers to bleeding from within the uterus before the 28th week, with dilation of the cervix but without expulsion of the products of conception. The term **missed abortion**describes a nonviable pregnancy that has been retained in the uterus without cervical dilation and without the spontaneous passage of products of conception. In **septic abortion**, embryonic or fetal demise has occurred, and intrauterine infection has developed, which has the potential risk of spreading systemically.  
Although the true incidence of spontaneous abortion is unknown, approximately 15% of clinically evident pregnancies and up to 50% of chemically evident pregnancies end in spontaneous abortion. Eighty percent of spontaneous abortions occur before 12 weeks’ gestation.  
The incidence of abortion is influenced by the age of the mother and by a number of pregnancy-related factors, including the number of previous spontaneous abortions, a previous intrauterine fetal demise, and a previous infant born with malformations or known genetic defects. Additionally, chromosomal abnormalities in either parent, such as balanced translocations, and medical comorbidities, such as thyroid disease and diabetes mellitus, may influence the rate of spontaneous abortion.

**Pathogenesis**An abnormal karyotype is present in as many as 50% of spontaneous abortions occurring during the first trimester. The incidence decreases to 20–30% of second-trimester losses and to 5–10% of thirdtrimester losses. The majority of chromosome abnormalities are trisomies (56%), followed by polyploidy (20%) and monosomy X (18%).  
Other suspected causes of spontaneous abortion are less common, and these include infection, anatomic defects, endocrine factors, immunologic factors, and exposure to toxic substances. In a significant percentage of spontaneous abortions, the etiology is unknown, even with genetic testing.

**Prevention**Some miscarriages can be prevented by early obstetric care and even preconception care, with adequate treatment of maternal comorbidities such as diabetes and hypertension, and by protection of pregnant women from environmental hazards and exposure to infectious diseases.  
**Clinical Findings  
A. Threatened Abortion**Approximately 25% of pregnant women experience first-trimester bleeding. In most cases, this bleeding is caused by implantation into the endometrium. The cervix remains closed, and slight bleeding with or without cramping may be noted. Resolution of the bleeding and cramping carries a favorable prognosis; however, these women are at increased risk for subsequent miscarriage. First trimester bleeding has also been associated with preterm premature rupture of membranes and preterm labor. Other causes, such as ectopic pregnancy and molar gestation, should also be considered.  
**B. Inevitable Abortion**Bleeding with cervical dilation, often with back or abdominal pain, indicate impending abortion. Unlike an incomplete abortion, the products of conception have not passed from the uterine cavity.  
**C. Incomplete Abortion**Incomplete abortion is defined as the passage of some but not all of the products of conception from the uterine cavity. Bleeding and cramping usually continue until all products of conception have been expelled. In general, severe pain and heavy bleeding occur and often require medical evaluation.

**D. Complete Abortion**In a complete abortion, all of the products of conception have passed from the uterine cavity and the cervix is closed. Slight bleeding and mild cramping may continue for several weeks.

**E. Missed Abortion**Missed abortion is defined as a pregnancy that has been retained within the uterus after embryonic or fetal demise. Cramping or bleeding may be present, but often there are no symptoms. The cervix is closed, and the products of conception remain in situ.  
**F. Anembryonic Pregnancy**Anembryonic pregnancy (previously called *blighted ovum*) is an ultrasound diagnosis. It is a pregnancy in which the embryo fails to develop or is resorbed after loss of viability. On ultrasound, an empty gestational sac is seen without a fetal pole. Clinical presentation is similar to that of a missed or threatened abortion: Mild pain or bleeding may be present; however, the cervix is closed, and the nonviable pregnancy is retained in the uterus.

**Laboratory Findings  
A. Complete Blood Count**If significant bleeding has occurred, the patient will be anemic. Both the white blood cell count and the sedimentation rate may be elevated, even without the presence of infection.  
**B. Pregnancy Tests**Falling or abnormally rising serum levels of β-human chorionic gonadotropin (hCG) are diagnostic of an abnormal pregnancy, either a failed intrauterine gestation or an ectopic pregnancy.  
**Ultrasound Findings**Transvaginal ultrasound is an essential diagnostic tool in diagnosing early normal and abnormal pregnancies. As early as 4–5 weeks of gestation, a gestational sac may be visualized in the uterus. In a normal intrauterine pregnancy, the sac is spherical and is eccentrically placed within the endometrium. At 5–6 weeks’ gestation, a yolk sac will be present. In general, a gestational sac with a mean sac diameter (MSD) of ≥8 mm should contain a yolk sac. Similarly, a gestational sac with an MSD of >16 mm should also contain an embryo. Pregnancies with a large gestational sac and no embryo are typically anembryonic gestations and are managed in a similar manner as a missed abortion. Fetal heart motion is expected in embryos with a crown to rump length of >5 mm or at 6–7 weeks’ gestation. If a repeat ultrasound in 1 week does not show embryonic cardiac activity, the diagnosis of embryonic demise is made.

In threatened abortion, ultrasound will reveal a normal gestational sac and a viable embryo. However, a large or irregular sac, an eccentric fetal pole, and/or a slow fetal heart rate (<85 beats/min) carry a poor prognosis. Miscarriage becomes increasingly less likely the further the gestation progresses. If a viable fetus of 6 weeks or less is seen on ultrasound, the risk of miscarriage is approximately 15–30%. The risk decreases to 5–10% at 7–9 weeks’ gestation and to less than 5% after 9 weeks’ gestation.  
In an incomplete abortion, the gestational sac usually is irregularly shaped. The diagnosis of complete abortion is also based on clinical findings. On ultrasound, the endometrial lining appears thin, and no products of conception are visible within the cavity. Importantly, a complete abortion is only diagnosed with certainty if a previous intrauterine gestation was documented on ultrasound. Otherwise, hCG levels must be followed to confirm the absence of ectopic pregnancy.

When findings on ultrasound are nonspecific, correlation with hCG levels can improve the ability to distinguish normal and abnormal pregnancies. In a normal pregnancy, the minimal rise in hCG is 53% over 48 hours. hCG values that rise slower than expected may be consistent with a failed intrauterine or ectopic pregnancy. Decreasing levels of hCG are also diagnostic of an abnormal pregnancy. In spontaneous abortion, the hCG values are expected to drop 21–35% in 2 days (depending on the initial hCG value). A slower decline is suggestive of an ectopic pregnancy.

**Complications**Severe or persistent bleeding during or after spontaneous abortion may be life-threatening. The more advanced the gestation, the greater the likelihood of excessive blood loss. Infection, intrauterine adhesions (Asherman’s syndrome), and infertility are other complications of abortion.  
Perforation of the uterus may occur during procedures to remove retained products of conception, namely dilatation and curettage (D&C). The rate of perforation during the first and second trimesters is approximately 0.5% for both induced and spontaneous abortions. Uterine perforation is more common during D&C performed in pregnancy because of the soft uterine wall and may be accompanied by injury to the bowel and bladder, hemorrhage, and infection. Surgical evacuation may also lead to cervical trauma and subsequent cervical insufficiency.

**Treatment of Abortions**Successful management of spontaneous abortion depends on early diagnosis. Every patient should have a complete history taken and a physical examination performed. Laboratory studies include a complete blood count, blood type, and cervical cultures to determine pathogens in case of infection.  
If the diagnosis of threatened abortion is made, pelvic rest can be recommended, although it has not been shown to prevent subsequent miscarriage. Prognosis is good when bleeding and/or cramping resolve.  
If the diagnosis of a missed or incomplete abortion is made, options include surgical, medical, or expectant management. In the past, surgery was the standard of care because of concern that medical or expectant management would lead to higher rates of retained pregnancy tissue and subsequent infection. More recently, expectant or medical management are acceptable alternatives and have even shown lower rates of infection despite their higher rates of retained products of conception. These patients also avoid the risks of surgery, including uterine perforation, intrauterine adhesions, and cervical insufficiency. The advantages of performing a manual vacuum aspiration (MVA) include convenient timing and low rates of retained products of conception.  
Expectant management allows the spontaneous passage of products of conception and avoids risks of surgery. Risks and side effects include unpredictable timing until the abortion is completed with the possibility of significant pain and bleeding, occasionally requiring emergent dilatation and curettage (D&C) or MVA. Expectant management also has the highest rates of retained pregnancy tissue, necessitating treatment with misoprostol (prostaglandin E1) or MVA.  
Patients who choose medical management are given misoprostol, a drug that induces uterine contractions and expulsion of the products of conception. The risk of retained products is lower than with expectant management; however, repeat doses of medication may be needed to complete the abortion. As with expectant management, timing can be unpredictable, and symptoms of pain and/or bleeding may necessitate emergent D&C. Expectant or medical management of abortion assumes that prompt medical evaluation is available. Those options should not be considered if medical care is not easily accessible.  
If the diagnosis of complete abortion is made, the patient should be observed for further bleeding. If bleeding is minimal, no further treatment is necessary. All products of conception should be examined and sent for pathologic examination to confirm an intrauterine pregnancy. If an intrauterine pregnancy was not previously seen on ultrasound and no pathology specimen is available, serial hCG levels are followed to confirm spontaneous abortion. If hCG levels decline more slowly than expected (eg, <21–35%), an ectopic pregnancy or retained products of conception must be considered. Molar gestation is also a possible diagnosis if hCG levels plateau or rise abnormally without an intrauterine pregnancy.  
If a complete or partial hydatidiform molar pregnancy is diagnosed, surgical evacuation with suction D&C should be performed. As long as hCG levels are decreasing and remain undetectable after molar evacuation, there is no need for chemotherapy. However, if hCG levels start rising, plateau, or are persistent for more than 6 months, evaluation for malignant postmolar gestational trophoblastic disease is indicated.  
**Treatment of Complications**Uterine perforation may result in intraperitoneal bleeding, as well as injury to the bladder and/or bowel. In many cases, uterine perforation is asymptomatic and goes unrecognized. When perforation and bowel or bladder injury is suspected or when heavy bleeding is encountered, laparoscopy and/or laparotomy are indicated to determine the extent of the perforation and to evaluate for injury to other adjacent organs.

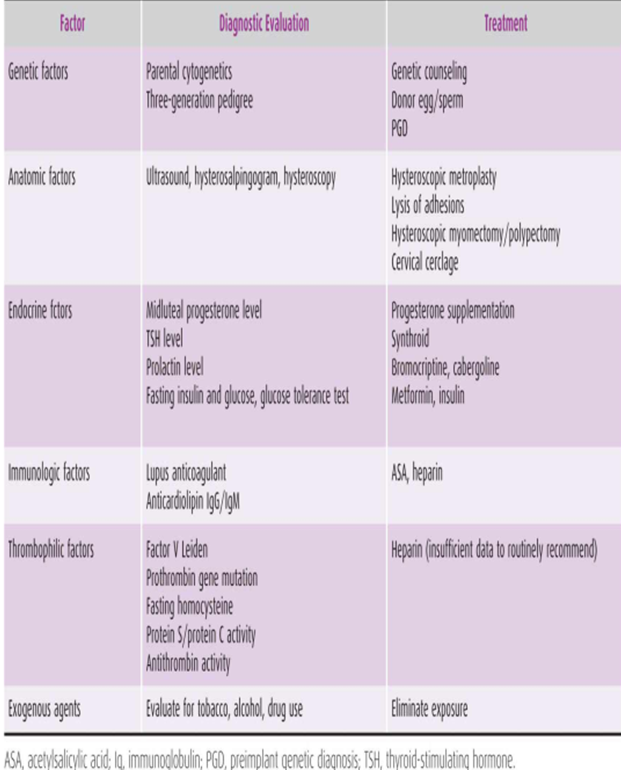
A Rh-negative patient without antibody in her system should be protected by anti-D gamma globulin 50 μg or 100 μg intramuscularly in cases of early miscarriage or late miscarriage respectively within 72 hours. However, anti-D may not be required in a case with complete miscarriage before 12 weeks of gestation where no instrumentation has been done.

**SEPTIC ABORTION**

In septic abortion, infection usually begins as endometritis involving the endometrium and any retained products of conception. These patients present with fevers, chills, abdominal pain, vaginal bleeding, and malodorous vaginal discharge. Without treatment, endometritis may spread beyond the uterus, leading to peritonitis, bacteremia, and sepsis.  
The 2 most common causes of septic abortion are retained products of conception and bacteria that have been introduced into the uterus via ascending infection. Pathogens that cause septic abortion are usually those seen in normal vaginal flora as well as sexually transmitted bacteria. Before performing D&C, screening for sexually transmitted infections is essential.  
In evaluating septic abortion, a complete blood count, urinalysis, endocervical cultures, blood cultures, and abdominal x-ray to rule out uterine perforation should be obtained. Ultrasound should be performed to look for retained products of conception.  
**Treatment**Treatment of septic abortion involves hospitalization and intravenous antibiotic therapy. Selection of antibiotic agents should provide for both anaerobic and aerobic coverage. If retained products of conception are diagnosed, a D&C is indicated.

**RECURRENT/HABITUAL MISCARRIAGE**

**Recurrent miscarriage**is defined as 3 or more consecutive pregnancy losses before 28 weeks of gestation, each with a fetus weighing less than 500 g. Recurrent pregnancy loss affects up to 5% of couples, often with no identifiable cause. The prognosis for a successful subsequent pregnancy correlates with the number of previous miscarriages. The risk of spontaneous abortion in a first pregnancy is approximately 15%, and this risk is at least doubled in women experiencing recurrent pregnancy loss.  
Overall, the prognosis after repeated losses is good, with most couples having an approximately 60% chance of a viable pregnancy. The table below shows the evaluation and management of recurrent early pregnancy loss:



Hyperemesis Gravidarum (HEG)

**HYPEREMESIS GRAVIDARUM**

Nausea and vomiting in pregnancy are extremely common; 70–80% of women experience these symptoms early in their pregnancy. Mild to moderate nausea and vomiting are especially common in pregnant women until approximately 16 weeks’ gestation.

Hyperemesis gravidarum (HEG) is defined as unexplained intractable nausea, retching, or vomiting beginning in the first trimester, resulting in dehydration, ketonuria, and typically a weight loss of more than 5% of prepregnancy weight. Severe HEG requiring hospital admission occurs in 0.3-2% of pregnancies.

**Etiology**

The pathogenesis is largely unknown, with possible contributing factors being increased levels of human chorionic gonadotropin (hCG), estradiol, and possible progesterone. It is more common among younger mothers and those with a history of motion sickness, migraines, and nausea and vomiting associated with oral contraceptives. It is more commonly seen in women carrying multiple gestations, and patients with siblings or a mother with HEG are more likely to be affected.

Other factors that increase the risk for admission include hyperthyroidism, previous or concurrent molar pregnancy, diabetes, gastrointestinal illnesses, some restrictive diets, and asthma and other allergic disorders. An association of Helicobacter pylori infection has been proposed, but evidence is not conclusive. Chronic marijuana use may cause the similar *cannabinoid hyperemesis syndrome*. And for unknown reasons—perhaps estrogen related—a female fetus increases the risk by 1.5-fold.

**Symptoms & Signs**HEG is associated with severe nausea and vomiting that may result in dehydration, weight loss, and frequently social isolation and negative impacts on relationships with family and friends. Patients with HEG, rather than nausea and vomiting of pregnancy, tend to have an earlier onset and longer duration. Excess salivation (ptyalism) may also be seen in a subset of women with HEG.

On examination features of dehydration and ketoacidosis may be present such as:dry coated tongue, sunken eyes, acetone smell inbreath, tachycardia, hypotension, rise in temperature may be noted, jaundice is a late feature.

**Complications**

Maternal complications of HEG can include Wernicke’s encephalopathy (due to thiamine deficiency), acute tubular necrosis/acute kidney injury (due to severe dehydration), central pontine myelinolysis, Mallory-Weiss tear of the esophagus, pneumomediastinum, and splenic avulsion. Additionally, significant psychological burden of the disease has been reported, with depression, anxiety, and lost work frequently seen among those with persistent or severe HEG. Fortunately, no clear fetal complications have been associated with HEG. Fetal risks may be due to low birth weight.

**Diagnosis**

The pregnancy is to be confirmed first. Suppressed thyroid-stimulating hormone/elevated free thyroxine and elevated liver enzymes, bilirubin, amylase, and lipase may all be noted in patients with severe nausea and vomiting; these are transiently abnormal and resolve with improvement of HEG. Other initial laboratory studies in the evaluation of women with HEG are:

v  Urinalysis for ketones and specific gravity

v  Serum levels of electrolytes and ketones

v  Urine culture

v  Calcium level

v  Hematocrit level

**Differential Diagnoses**

The differential diagnosis for late (onset after 9 weeks gestation) HEG should include gastroenteritis, gastroparesis, biliary tract disease, hepatitis, acute febrile infection, peptic ulcer disease, pancreatitis, appendicitis, pyelonephritis, ovarian torsion, diabetic ketoacidosis, migraines, drug toxicity or withdrawal, psychological conditions, acute fatty liver of pregnancy, and preeclampsia.

**Management**

Initial management of pregnant women with HEG should be conservative and may include reassurance, dietary recommendations and support. Dietary modifications in patients with HEG or morning sickness may include the following:

Ø  Eat when hungry, regardless of normal meal times

Ø  Eat frequent small meals

Ø  Avoid fatty and spicy foods and emetogenic foods or smells. Increase intake of bland or dry foods.

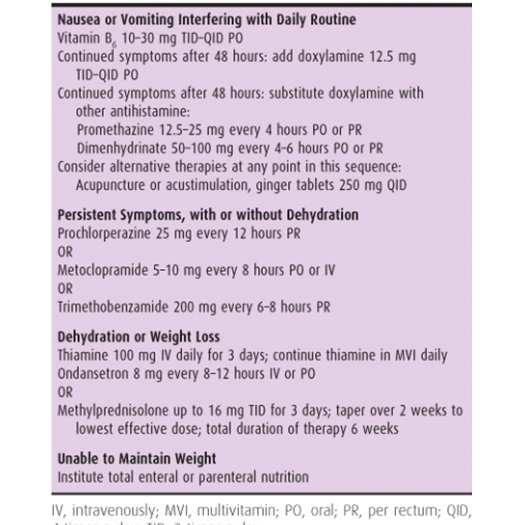
Ø  Eliminate pills with iron

Ø  High protein snacks are helpful

Ø  Crackers in the morning may be helpful

Ø  Other suggested foods include herbal teas containing peppermint or ginger, other ginger-containing beverages, broth, unbuttered toast, gelatin or frozen desserts.

If pharmacologic therapy is necessary, treatment is given using the algorithm below that balances safety and efficacy:



If medications and outpatient hydration fail or if severe electrolyte disturbance persist, inpatient admission for IV hydration may be necessary. In some refractory severe cases of HEG, if maternal survival is threatened, or if HEG is causing severe physical and psychological burden, termination of pregnancy should be considered.

**Prognosis**More than 50% of women have resolution of symptoms by 16 weeks of gestational age and 80% by 20 weeks. However, approximately 10% will be affected to some degree with severe nausea and vomiting for the duration of the pregnancy. HEG has been shown to recur in up to 80% of subsequent pregnancies, although earlier aggressive medical therapy prior to significant symptoms has been demonstrated to reduce both the severity and recurrence rate overall in future pregnancies.