GYNECOLOGY & OBSTETRICS DONE BY: YAZAN ALAWNEH

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Gynecology

Menstruation & It's Disorders

Frequen	су	Duration	Volume	Regularity
Mean is 28 da	ays +/-	Normal 4.5-8 d	Normal 25-50	Cycle to cycle
7 days		Prolonged >8d	Mean 40 ml;	variation over 12
		Shortened <4.5	Heavy >80ml	months, measured
** tend to shorten		Mean is 5 days	Light <5ml	in days
with age and its		** with aging the		
initially irreg	ular	duration decrease		
Dysmenorrhea				
- suprapubic, sharp, colicky, cyclic pain				
5/5x	- begi	ns just before or with	the onset of menses	
	- lasts	8-72 nours	-l'a sub-sa sa sa sa sa sa	
	- Associated Sx: headache, diarrhea, nausea			
• 1ry: pain just before menses and during menses) d gradually increases	
Iry/Zry	• Zry:	pain begins several u	ays before menses and	a gradually increases
	• End	enty as menses appro		
		Unethosis	Adenomyosis IllCD in utero	
2ry causes	Poly	vicadhesions	• Fibroids	
• Cervical stenosis (introgenic LLETZ/instrumentation)		tion)		
	• Con	genital abnormalities	causing genital tract of	hstruction egnon-
	comm	nunicating cornua		
	• reas	surance, analgesia		
	• Sym	ptoms control:		
	- PGSI	prostaglandin synth	etase inhibitors): mefe	enamic acid
	- COC	P to abolish ovulation		
	- data	on Mirena IUS		
	- para	cetamol		
Mx	- hot v	water bottles		
 treat underlying causes 				
	- Endo	ometriosis: COCP, pro	gestogens, GnRH	
	- PID:	antibiotics		
	- Obst	ruction: surgical		
	- Lapa	roscopy: GS for Dx/M	x of endometriosis, ac	lhesions, PID

	Premenstrual Syndror	me (PMS)	
	 Distressing psychological, physica 	l, behavioral Sx	
Info	 occur mainly during luteal phase, 	or after hysterectomy with	
	ovarian conservation		
	Physiologic PMS (95%)	Core PMS (5%)	
Types	Cyclical, Sx free week	in the follicular phase	
	Mild, no serious impact on quality	Cause impairment & impact QC	
	of life (QOL)		
	Physical signs	Psychological and behavioral	
	 breast tenderness 	- mood swings (Irritability, ange	
	 abdominal swelling 	anxiety, depression)	
Sx	- headache	- sleep disturbances, changes in	
	- skin disorders	appetite, fatigue	
	- weight gain	- poor concentration	
	 extremities swelling 	- social withdrawal, lonely	
	- joint, muscle and back pain	 lack interest, hopelessness 	
	• Daily record of PMS Sx for 2 conse	ecutive menstrual cycles	
	 we care for timing & severity of Sx more than character 		
Dx	 Cyclical or luteal phase 		
	• Must resolve by the end of menstruation to give at least one week		
	free of symptoms		
	 Mild: support, reassure, nutrition 	n, exercise, stress reduce	
	• Core:		
Мх	a. Suppression of ovulation: GnRHa, COCP, Danazol, Estrogen		
	b. ttt w/o suppression: SSRI, Diuretics, non-medication ttt (herbal,		
	behavioral therapy, vit B6, Ca ⁺² , exe	ercise)	
	Heavy menstrual bleeding (HN	1B – Menorrhagia)	
	 Idiopathic (40-60%), Subjective ratio 	ather than objective	
	Pathologic:		
	- Fibroids (20-30%) - Endomet	riosis (rare)	
Causes		· · · ·	
Causes	- Polyps (5-10%) - Malignan	ncy (rare) - Adenomyosis (59	
Causes	- Polyps (5-10%) - Malignan - HMB (Menorrhagia): Heavy menstrual	hcy (rare) - Adenomyosis (59 bleeding: HMB in excess of 80mls	
Causes	 Polyps (5-10%) Malignan HMB (Menorrhagia): Heavy menstrual MBL: Menstrual blood loss 	hcy (rare) - Adenomyosis (59 bleeding: HMB in excess of 80mls	
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Causes Abbrev.	 Polyps (5-10%) MBI: Menorrhagia): Heavy menstrual MBL: Menstrual blood loss AUB: Abnormal uterine bleeding IMB: Inter-menstrual bleeding PMP: Post-menopausal bleeding 	ncy (rare) - Adenomyosis (59 bleeding: HMB in excess of 80mls	
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6 Infection (endometritis) iatrogenic (breakthrough, smear), • Structural (polyp, fibroids, ectropion, tumors, Ca) • natural 1% • Polyps 30% Atrophy 30% • Fibroids 20% • Hyperplasia • Ca • **R/O pregnancy!!** • PALM-COEIN: - Structural: Polyp, Adenomyosis, Leiomyoma, Malignancy - Non-Structural: Coagulopathy: thrombocytopenia, leukemia, warfarin **O**vulatory dysfunction: PCOS, CAH, Cushing Endometrial disorders: Endometritis latrogenic: COCP, IUCD, progestins Not classified: AVM, endometriosis, ovarian neoplasm o Associated Sx: • Lifestyle influence Dysmenorrhea Dyspareunia (endometriosis, PID) Pressure Sx

Offensive vaginal discharge (infections)

o Medical Hx:

IMB Causes

PMP Causes

AUB Causes

(Figo

classifica.)

Hx

PE

- Anti-coagulants
 Tamoxifen • Thyroid disorders
- o General: BMI, hypothyroidism, bruises

o Abdominal:

- masses, tenderness (endometriosis, pelvic infection), Cysts, Fibroids

o Speculum:

- Local lesions
 - Severity of blood loss
 - Bimanual examination: shape, size, tenderness, mobility
 - Enlarged: fibroids, adenomyosis
 - Restricted: endometriosis, pelvic infections
 - Tenderness: endometriosis, adenomyosis, PID

 TVS USS (1st investigation): polyp, fibroids, thickness Saline infusion sonography (SIS): uterine, endometrium R/O pregnancy: MCC of abnormal bleeding Smear TFT, FBC (PID increase WBC) Biopsy: to exclude hyperplasia or cancer Sampling: D/C, hysteroscopy, Pipelle 96% of PM women with endometrial cancer will have thickness (ET) 4 mm, Women with PMB whose ET is <4 mm still have a 1–2% risk of naving endometrial cancer.
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Endometrial biopsy is required if ET is:
····
>4 mm in postmenopausal women
>16 mm in premenopausal women
May be selectively performed in postmenopausal with ET <4 mm if
other C/P or sonographic risk factors are present.
Medical:
[,] Hormonal
Mirena IUS: contains levonorgestrel progesterone to Mx
Progesterone: similar physiology
COCP: control amount
Danazol (used in endometriosis Mx): serious androgen SE
[,] Non-hormonal:
Antifibrinolytics (50% \downarrow in loss): tranexamic acid
<i>NSAIDs</i> : mefenamic acid (30-40% \downarrow in loss & \downarrow in dysme.)
Surgical:
• Endometrial ablation: thermal, curettage, balloon
y Hysterectomy

o 15 yr noted menarche at 14 but only had 3-4 periods since, she missed school due to massive bleeding:

- Coagulation problem

- Thyroid disease,

- Anovulatory cycle

"prolonged heavy bleeding because of estrogen withdrawal, but progesterone withdrawal is short time bleeding"

- PCOS

24-year case with bleeding she can't predict her cycle which is heavy and painful:

- PCOS: why heavy ??? because of oligomenorrhoea "but because of estrogen level it will be prolonged ".

- Reproductive age: usually benign, we have to R/O organic cause to say this is dysfunctional bleeding, if she uses OCPs

- Defloration injuries newly married women: vaginal tears > vaginal repair under GA.

- Vaginal and cervical cancers are unusual in this age.

- Regular cycle suggests anovulatory cycle, Irregular suggest organic disease.

- Ask about Hx of smoking, natural status, multiple partner.

o 49 years old women presented with heavy irregular menses:

- "We have to rule out endometrial cancers",

- It's usually anovulatory cycle but we have to send biopsy

o 60 years old women presented with 2 days of spotting:

- She is menopausal: Endometrial sample "to R/O cancer".

	Endometrial Polyps (localized overgrowths)
C/P	 HMB, PMB, IMB & abnormal vaginal discharge
	 Large or multiple are implicated in subfertility
Dx	 USS, SIS & hysteroscopy
Мх	 Removed hysteroscopically in postmenopausal patient or >1cm in
	size in an asymptomatic premenopausal patient

Menopause

Definition	Permanent cessation of menses for 1 y (12m) at any age	
Variations	<u>Natural</u> : lack of follicles <u>Induced</u> (medical:GnBH analog/Surgical:Oonborectomy)	
Variations	• <u>Premature Menonguse</u> (POI)	
	- Conserved ovaries may fail early after (TAH)	
Age	Avg is 51	
	if earlier <45: FHx (30-70% inherited) , DMI, Smoking	
	Premature menopause POI	
Definition	Menopause <40, unknown cause, ttt. HRT (as OF)	
	 Chromosomal abnormality (turner) 	
	 Auto-immune hypothyroidism: Addison's 	
Causes	 Enzyme deficiencies: galactocemia 	
	• Surgical	
	 CT/RT (Concurrent Chemotherapy & Radiotherapy) 	
	 Infections: TB, mumps, malaria 	
Landmarks	 Decline in fertility (no cycle dysfunction) 	
of OF	 Menstrual cycle changes (longer, till it disappears) 	
	o check ovarian reserve of the follicle we either do:	
	1) <i>FSH blood level</i> in case of subfertility in correlation with period - Days 2.3 &4)	
	2) Anti-Müllerian hormone (AMH) - (at any time) if low,	
	that means that ovarian reserve is low	
Physiology	• initially, compensated failure happens, FSH \uparrow to	
	stimulate 个 follicles to produce estrogen.	
	• Later on, Decompensated failure occur, Both FSH & LH \downarrow , mainly FSH	
	• AMH $\sqrt{2}$ as the number of follicles $\sqrt{2}$ measurement of	
	AMH could help predict the age at menopause	

	• Not only estrogen is decreased in menopause , but also Androgens ex hormone binding globulin decreases some testosterone continues to be produced that's why some women realize some hirsutism, that's a physiologic event not a pathologic problem.		
	• insulin resistance 个 after the menopause > 个 in central adiposity (android shape)		
	 Vaginal pH is acidic, after menopause alkaline 		
	 o Acute: Changes in menstrual pattern (length, amount) Hot flushes, night sweats (self-limiting) Mood swings, panic, Headache 		
S/Sx	 o Mid-term: Vaginal dryness, dyspareunia reduced libido (↓ androgen) atrophic urethritis: stress & urge urinary incontinence thinning of skin/hair loss, brittle nails Aches, pains (↓ estrogen) 		
	o Long-term:• Osteoporosis• CVS disease• Stroke• Dementia• ↑ bodyweight• Body fat redistribution (Android)		
Мх	 Life-style Psychological support HRT: oral, patches, IUS, Cream HRT alternatives 		

	 o Special group whom HRT should be prescribed: Premature OF Gonadal dysgenesis Surgical/Radiation menopause should be given HRT till menopause expected day, they have no higher risk than ladies with natural menopause given HRT at 50 or 51
	 o Types: • Estrogen only: only used in the absence of uterus (TAH) • Combined • Tissue selective Tibolone: - estrogenic, progestogenic, weak androgenic (for lipido) - only given for definite menopausal state (not before) • Testosterone: implants SC in abdominal wall
HRT	o Routes: • Oral: - 1 st choice, cost-effective - beneficial on HDL/LDL/Total cholesterol - affects liver protein synthesis (个 triglycerides) - high doses required - all tablets contain lactose
	 Transdermal (patch, gel, nasal spray): lower dose required, expensive less SE on gallbladder, coagulation factors more physiologic hormonal levels
	 Topical (Cream): for urogenital Sx/atrophy, & no systemic SE

• IUS (Mirena coil)

o HRT Benefits:

- \downarrow vasomotor Sx
- \downarrow Urogenital Sx, better sexuality
- \downarrow OP (\uparrow BMD, \downarrow vertebral, hip fractures (BOTH!)
- \downarrow colorectal cancer

o HRT Risks:

- ↑ breast cancer: P+E > E
- \uparrow endometrial cancer: E, Tibolone
- ↑ VTE: P+E > E
- \uparrow Gallbladder disease
- Note no POF!!

o RCOG on HRT:

- prescribe HRT for significant menopausal Sx
- no Sx, risk > benefit
- women have the final decision
- in POI, HRT can be used until menopause

o International menopause society IMS:

- HRT given for clear indication
- women have option
- risk & benefits clearly explained
- lowest effective dose
- HRT women, assessed at least annually

o Contraindications:

- Active liver disease, renal
- Breast cancer
- CVD, angina, MI, stroke, uncontrolled HTN
- DVT
- Endometrial cancer
- Abnormal uterine bleeding

o Exam b4 HRT:

- BMI
- BP
- Pelvic, breast (if (+) Hx)
- o Exam prior to HRT: not routinely indicated
- Mammogram (because HRT changes breast density)
- Endometrial sampling
- FSH > 30 IU/I (one reading not enough)
- TSH, T4: menopause & thyroid Sx are common

o Follow up:

- recommended: 3/12, 6/12, yearly (BP, breast, V/E)
- 3 yearly smears & 3 yearly mammography aged 50-64

o Factors influence HRT prescription:

- Hysterectomy
- Patients Preference: Oral, non-oral preparations

o HRT systemic SE:

- Oestrogen:
- fluid retention, breast tenderness/enlargement, nausea, headaches, leg cramps bloating , & dyspepsia.

• Progestogen:

- fluid retention, breast tenderness, headaches/migraine, mood swings, depression, acne, lower abdominal pain, and backache.

- Combined HRT:
- *irregular*, breakthrough bleeding (need investigation).
- All HRT:
- weight gain (not proved in RCT)

HRT Alternate	 o Lifestyle o Non pharmacological: vaginal Sx gel Replens o Pharmacological: Progestogens α-2 agonists – Clonidine SSRI (fluoxetine, paroxetine), SNRIs (venlafaxine) Gabapentin Dehydroepiandrosterone Progesterone transdermal creams o Phytoestrogens 		
HRT	o Phytoestrogens		
Complem-	o Herbal remedies		
entary	o Other: hypnotherapy, reflexology		
	o Vitamins/minerals: E&C, Selenium		
	Osteoporosis		
Definition	BND by 1-score Normal: ≥ -1 SD Osteopenia: < -1 SD - > 2.5 SD Osteoporosis: ≤ 2.5 SD Established OP: <2.5 SD with fragility fractures		
RF	 General: Age - Sex - BMI: ≤19 Previous fragility fracture Parental Hx of hip fracture Current steroid ttt 		
2ry Causes	 Estrogen deficiency: untreated POI Medical: RA, DM I, Hyperthyroid, malabsorption, chronic liver disease, COPD, organ transplantation 		
Мх	 Lifestyle: smoking, alcohol, hip protector, exercise, diet HRT: recommend in POI till the age of menopause Calcium and vit D, Calcitonin, SERMs, Strontium Bisphosphates: Alendronate (Fosamax) 		

Puberty

• Thelarche: 1st sign of puberty , breast development (breast budding), it exceeded menarche by 2-3 years

- Menarche: Last sign, 1st menstrual cycle
- Signs of puberty:
- Thelarche, Menarche, Growth spurt, Axillary, pubic hair
- **Tanner staging**: used to classify development based on breast and pubic hair (5 Stages for each)
- Pubic hair is dependent on Adrenal androgen
- Age of puberty: Avg in middle east is 12.5 year
- Leptin is an important hormone for puberty

	Precocious Puberty
Definition	Onset of puberty <i>before</i> age of 8 in females, 9 in males
	 Central, true PP, gonadotropin-dependent 80%: brain T (do MRI), CNS malformation, 75% idiopathic
Classify	 Peripheral PP or pseudo-puberty 20%: always pathologic and caused by: a. hormone producing ovarian tumor
	 b. exogenous estrogen c. <i>McCune Albright syndrome</i> (polyostotic dysplasia, café au lait lesions and precocious puberty)
Investigate	 Gonadotrophin levels (FSH, LH) Brain imaging: MRI Pelvic, abdominal imaging (ovarian, adrenal T)
Мх	 we care for bone age (maturation) 1. Lesion – Resection 2. GnRH Analogues – ↓ FSH, LH (Pituitary shutdown)

1	6
т	υ

	Delayed Puberty
Definition	Absence of all 2ry sexual characteristics by age 14
Definition	Hypogonadotronic hypogonadism: Causes:
	- Constitutional delay (mc)
	- Chronic illness (DM_CRE_CE)
	- Anorevia nervosa
	- Kallman's syndrome
	- Hydrocenhalus or CNS tumor
Classif	- Pituitary adenoma (prolactinoma)
ication	
ication	• Hypergonadotronic hypogonadism: Causes:
	- Abnormal gonadal development
	- Turner syndrome
	- Swyer syndrome
	- Premature ovarian failure (POF)
	- Following chemo, radio
	- Galactosemia
	- Infections (mumps)
	Puberty Induction:
	- low dose of ethynyl estradiol (1/10 COCP) at night.
	then dose increased at 6 m interval until breakthrough
Мх	vaginal bleeding occurs then use combined pills
	Hypogonadotropic hypogonadism:
	- Pulsatile gonadotropins via SC pump or FSH and LH
	• Long term (COCP or HRT - to prevent osteoporosis)
	Kallman's Syndrome
	• F:M – 7:1
	• X-linked Recessive
Information	 S/Sx: delayed puberty, anosmia (or hyposmia),
	commonly there are associated midline structural
	defect and mental restriction

	Turner Syndrome
Genetics	 Mosiaciasm (46 XX + 45 XO)
S/Sx	 Prenatal: cystic hygroma, non-immune hydrops, IUGR Postnatal: Short stature Gonadal failure (1/3 post menarche - 2ry Amenorrhea Shield chest: widely spaced nipples Short and webbed neck low hair line
	 Lymphoedema Cardiac (Aorta Coarctation), Renal (Horse shoe kidney) Endocrine (Hypothyroid, insulin resistance)
Dx	 Karyotyping
Мх	 GH to improve height Puberty induction Long-term HRT (to grow the uterus) Childbearing possible by ovum donation, they have inactive ovaries, but they have a small uterus
	Svwver Svndrome
Genetics	• 46 XY, 10% mutation in Sry gene, 90% idiopathic
S/Sx	 Non functioning testes (no AMH, no testosterone) Present uterus, fallopian tubes 30% gonadal malignancy risk (Dysgerminoma) Gonadal dysgenesis Female external Genetalia Tall stature Absence of pubertal development
Dx	 Karyotyping, Assess Sry mutation
Мх	 Puberty Induction Childbearing (Ovum donation) Gonadectomy to exclude malignancy Long torm HPT

1^{ry} Amenorrhea (3%)

Definition	no mens	truation, investigated at a	age 14 without 2 ^{ry} sexual
	characte	ristics (SSC) or 16 + SSC (r	mainly breast enlarge.)
	a. Histor	y :	
	- Anosmi	a (<u>Kallman's Syndrome</u> : (GnRH deficiency, X-R)
	- Excessiv	ve exercise or competitive	e sports: this is the MCC
	- Anorex	ia Nervosa (infertility cau	se - low gonadotropins)
Clinical	- Cyclic P	elvic Pain	
	b. Exami	nation:	
	- Stature	(short: <u>Turner</u> , Tall: <u>Kalln</u>	<u>nan's</u>)
	- <mark>BMI</mark> (V.	low: <u>Anorexia Nervosa</u> /	high & low are causes)
	- Breast o	development (gonads are	e functioning or used to)
	- Presence	ce of hair (absent in andro	ogen insensitivity)
	- FSH, LH		
Investiga-	- Estradio	ol level	
tions	- Periphe	ral blood karyotype	
	- Pelvic L	I/S and MRI	
Mx	- Hormoi	<mark>ne</mark> (if gonadal): induce pu	berty, protect from OP
	- Expand	the vagina to allow sex a	is in (CAIS and MRKH)
	- Psychol	ogical support for fertility	y and sexual implications
	- Fertility	by (ovum donation): in S	Swyer and turner, in
	Rokitans	ky we need a surrogate u	iterus
	Swyer:	uterus with non-functio	ning testes
	CAIS	: no uterus with function	ning tests
		1ry Amenorrhea	
Case	es	Breast Development	FSH Level
A. Centra	I defect	Absent	Low
B. Gonad	problem	Absent	High
Karyoty	/pe is	Turner synd	rome (45 XO)
require	ed to	Premature ovarian	tailure (POF – 46 XX)
ditteren	itiate:	Swyer Synd	rome (46 XY)

C. Puberty Arrest	Normal	High
Karyotype +	POF, Uteru	us Present
imaging study	Complete Androgen Ins	ensitivity (CAIS - 46 XY)
D. Anatomical	Normal	Normal
	Absent Uterus Rokitans	ky syndrome or MRKH,
Do Pelvic imaging	Obstructive anom	alies: might cause
to classify into:	<u>hematometra</u> . Or press	ure effect urinary <u>acute</u>
	<u>urine re</u>	tention_

2^{ry} Amenorrhea (97%)

Definition	the absence of menstrual periods for 6 m in a woman
	who had previously been regular, & 12m in irregular
Causes	 The MCC for 2ry Amenorrhea is pregnancy,
	• the MCC pathologically is PCOS
	a. History
Clinical	b. Physical Examination:
	 Hirsutism, clitoromegaly, galactorrhea
	- E2 def: smooth vagina, lacks rugae, dry endocervix
	Hormonal profile
	• TFT : hyperthyroidism/hyperprolactinemia cause 1ry/2ry
	• Endometrium thickness:
	- If thick then high estrogen
	 if think then no estrogen (POF): we check FSH, LH, E2
	 - in POF: low E2, high FSH (<25) in two occasions
Investiga-	• FSH testing:
tions	FSH is greater than 30-40, indicates OF
	• estradiol lvl:
	With ovarian failure, estrogen is low (<20-40)
	 Long period of anovulation: Biopsy: R/O hyperplasia,
	malignancy
	 Progesterone challenge test

Progesterone challenge test			
	-	+	-
Anovulation, PCOS		ion, PCOS	Low estrogen, Anatomical, POF
Wants to	o get	No:	
pregna	nt:	- OCP	
- Clomip	hene	- Periodic	
citrate, i	nject	progestin	
gonadotr	opins	withdrawal	
- do not bleed after the progestin challenge but do bleed			
after estrogen/progestin and have normal or low FSH/LH			
Hypotha-			
lamic	- Possi	ble Causes of Hyp	othalamic Amenorrhea:
Ammen-	 medications (e.g. phenothiazines), 		
rrhea	- extre	mes of weight loss	·,
	- stress	s or exercise.	
	- A pitı	uitary or hypothala	imic tumor
Мх	HRT fo	or protection from	osteoporosis.

• Asherman's syndrome are intrauterine adhesions that occur after infections or D&C, if the basalis gets damaged think of Asherman's

• Anovulation: (Progesterone Lacking, no corpus luteum) - it can manifest in the early stages amenorrhea, but in the late stages menorrhagia, characterized by very thin and sticky cervical mucus

Infertility

Definition	 Inability to conceive after 1-2 y of regular UPSI (NICE) it is linked to the woman's age, if <35 y she can try for 2 y, if >35y then for 1 y and if >40 y then for only 6 m Brimary infortility: couple failed to conceive before
	• Secondary: been pregnant regardless the outcome
Conception	• 85% will conceive within 1 yr (if no Contra & <35 yo)
Chances	 ½ will conceive in 2nd year (cumulative 92%)
	 the probability of pregnancy for 1 regular cycle is 20%,
	• UPSI Frequency/timing:
	 every 2-3 d optimize chances, the more the better
	• BMI:
	 high BMI (>30) causes anovulation (long time)
Factors	 low BMI (<19) have irregular menstruation
Affecting	• Smoking: in both parents
Fertility	 Caffeinated beverages: no evidence
	 Alcohol: intoxication affect semen quality
	 Prescribed, OTC and recreational drug use
	Occupation
	• Tight underwear: \uparrow scrotal temperature & \downarrow quality
-	- 25% male, 25% female, 25% mixed, 25% unexplained
Causes	1) Sperm motility, morphology & concentration (count).
	2) Regular ovulation of a healthy ovum each cycle.
	3) Healthy fallopian tubes and receptive endometrium.
Desis Marde	• Carried out by the GPs and should be offered to:
Basic Work	1. Woman in reproductive age not conceived after 1
up	year of UPSI, in the absence of any known cause
	2. woman in reproductive age who is using artificial
	insemination to conceive after 6 failed trials.

- Consider earlier referral to infertility specialists in:
- the woman is aged 36 years or over.
- there is a known clinical cause or a Hx of infertility.
- treatment that may result in infertility (as cancer ttt).
- People concerned about their fertility & who known to have chronic viral infections such as hep B, C or HIV.

• Semen analysis (WHO 2010) – (Kruger's Criteria):

- we call it <u>SFA</u> (<u>seminal fluid analysis</u>) or sperm analysis the pt. abstain from sex for 3 days then take a sample

Semen Volume	1.5 ml or more
рН	7.2 or more
Sperm Concentration	≥ 15 million spermatozoa/ml
	≥ 39 million spermatozoa/ejaculation
Total Sperm count	Oligospermia: low count
	Azoospermia: absent count
	Aspermia: No ejaculation/retrograde
Total Mobility	≥ 40% or ≥ 32% with progressive
	motility (known as Grade A.)

 <u>Retrograde ejaculation</u> happens when the bladder neck fails to close, we Dx by telling the pt. to have sex and then we perform a urine analysis, we treat this case by treating the cause, to solve infertility we may do IVF, we obtain the sperms for IVF, by 2 options:

<u>We alkaline the urine of the pt</u>. 1-night b4 taking the specimen so that the sperms will survive in acidic urine.
 We do surgical sperm retrieval: take sperms directly from the testes (*TESA - testicular sperm aspiration*)

- What are the components of seminal fluid?

96% of the seminal fluid is *seminal plasma*, secretions from prostate & seminal vesicles, and about *3% sperms*

• Evidence of ovulation

(Day 2-3 gonadotropins, Day 21 progesterone):

- *Evidence*: we should have a regular cycle to do this:

a. Menstrual Hx of regular cycle

b. serum progesterone in the mid-luteal phase of their cycle MLP (mid luteal progesterone - day 21 of 28 day cycle – even if they have regular menstrual cycles)
c. Serum gonatropins (FSH/LH) on <u>Day 2-3</u>: at day 3 we test the level of hormones, (FSH, LH and E2) to check the ovarian reserve (# of eggs) especially in irregular periods

• Susceptibility of Rubella:

harmful to the fetus, it causes congenital rubella syndrome (triad: sensorineural deafness (58%), eye abnormalities 43%, congenital heart disease 50%)

- Cervical Smear Screening
- Screening for Chlamydia trachomatis
- Serum prolactin: only needed in irregular cycles
- TFT: only needed in irregular cycles

• Ovarian reserve:

more important in >35 yo, suspected ovarian failure and to detect response to ovulation induction
no evidence for: ovarian volume, ovarian blood flow, inhibin B, E2

We have three methods to asses ovarian reserve:
1) Early follicular phase (day 2-5): FSH it should be > 8.9
IU/L for a low response and < 4 IU/L for high response, but this is not too sensitive because of cyclic changes.

2) *AMH* (Anti-mullerian hormone): it prevent the effect of FSH, and decrease follicle recruiting each cycle to preserve eggs, and this test is more sensitive, you can order this test at any point during the menstrual cycle as it is not affected by the cyclic changes: \leq 5.4 pmol/L for a low response or \geq 25 pmol/L for a high response

3) *AFC* (Total antral follicle count): we count them using U/S and it is sensitive.

- HSG: tubal patency test, used to see the uterus shape.
- Hysterosalpingo-contrast-Sonography (Hy-Co-Sy)
- tubal patency test
- Consider before IUI
- Requires more expertise.
- Less invasive.

• Laparoscopy:

- *Invasive*, check for pelvic disease, endometriosis, and check for tubal patency by using a dye

- Therapeutic as in laparoscopic myomectomy, LOD, and tubal surgery

• Hysteroscopy:

- in case of repeated IVF cycles
- therapeutic as intra-uterine septum
- Counseling
- Мx
- Treat the cause
- Treat the cause
- Ovulation induction

• Artificial (Intrauterine) insemination (IUI):

- used for female infertility factor (not male): inject the male sample inside the uterus, according to the NICE, IUI is recommended *in only 3 cases*:

1) Inability of the couple to have sexual intercourse

2) HIV infected male

3) Couple of a same sex

- 50% of women will conceive after 6 cycles.
- ½ of unsuccessful, will conceive with further 6 cycles.
- Success rate varies between 8-12% per cycle.

- Do not offer IUI for people with unexplained infertility, mild endometriosis or mild male factor

• *IVF/ICSI*: intracytoplasmic sperm injection, where we inject the sperm inside the cytoplasm of the egg

o Treat the Cause:

Male factors:

A. Medical Mx:

- Men with hypogonadotrophic hypogonadism should be offered gonadotrophin drugs.

- Men with idiopathic semen abnormalities should not be offered anti-oestrogens, gonadotrophins, androgens, bromocriptine

B. Surgical:

- correct the epididymal block: obstructive azoospermia.
- No evidence for surgical ttt of varicocele in infertility.
- SSR (PESA, TESA and TESE) ...then ART

SSR: surgical sperm retrieval.

PESA: percutaneous episymal sperm aspiration.

TESA: testicular sperm aspiration through a needle.

TESE: testicular sperm extraction, where we open the testes and take a biopsy.

ART (form Wikipedia): assisted reproductive technology.

C. Management of ejaculatory failure:

Causes of azoospermia or oligospermia: it may be

1) higher center hypogonadotropic: give HRT

2) Testicular failure with high gonadotrophins.

3) Post testicular they mostly have obstruction, or retrograde ejaculation, they may also have bilateral absence of the vas like cystic fibrosis pts, and they always check for this in the west as CF is common

Female factors:

o Ovulation disorders:

The WHO classifies ovulation disorders into 3 groups:

 Group I: hypothalamic pituitary failure (hypothalamic amenorrhoea or hypogonadotrophic hypogonadism).
 Group II: hypothalamic-pituitary-ovarian dysfunction (Predominately PCOS).

3. Group III: ovarian failure

GROUP 1: - Weight gain if BMI less than 19. - pulsatile administration of GnRH or gonadotrophins with LH activity to induce ovulation.

• Hyperprolactinaemic amenorrhoea:

- Women with ovulatory disorders due to hyperprolactinaemia should be offered treatment with <u>dopamine agonists</u> such as <u>bromocriptine</u>.

o Tubal and uterine factors:

1. Tubal microsurgery and laparoscopic tubal surgery:

- May be more effective than no treatment.

- No strong evidence. (e.g.: fimbrial end dilatation)

2. Tubal catheterization or cannulation:

 With proximal tubal obstruction, selective salpingography + tubal catheterisation, or hysteroscopic tubal cannulation, may be ttt options.

- pregnancy probability 50%

3. Uterine surgery:

- Women with amenorrhea who are found to have intrauterine adhesions should be offered hysteroscopic adhesiolysis because this may restore menstruation and improve the chance of pregnancy.

4. Surgery for hydrosalpinges before IVF treatment:

- Laparoscopic salping ectomy or disconnection of both tubes improve IVF/ICSI success (\uparrow pregnancy rate 50%).

o Endometriosis:

1. Medical Mx:

- Ovarian suppression of minimal & mild endometriosis diagnosed as the cause of infertility in women does not enhance fertility and should not be offered.

2. Surgical ablation:

- In minimal or mild endometriosis; surgical ablation or resection of endometriosis + laparoscopic adhesiolysis improves the chance of spontaneous pregnancy.

- Laparoscopic resection of endometriomas may be beneficial, however recent RCTs suggest intervention only in endometriomas > 4cm.

	- In moderate or severe endometriosis; surgical
	treatment should be offered. (Debatable)
	 Post-operative medical treatment does not improve
	pregnancy rates.
	Unexplained infertility:
	 Ovarian stimulation should not be considered as does
	not improve pregnancy or birth rates.
	 Advise to try to conceive for 2 years of UPSI before
	other options (Fecundity is 3- 5%).
	- After 2 y (dr:5y) of failure to conceive consider IVF/ICSI
	IVF/ICSI
	the oocyte is fertilized by sperm outside the body (in
	vitro) & then gamete retransferred intrauterine
Definition	There is a criterion in IVF that we should only put back
	one embryo to avoid multiple pregnancies
	Unfortunately here in Jordan there is no regulation so
	• In women <40 who have not conceived after 2 y of
	regular UPSI or 12 cycles of artificial insemination
	(Where 6 or more are by intrauterine insemination),
	offer 3 full cycles of IVF, with or without ICSI. If the
	woman reaches 40 during ttt, complete the current full
Indications	cycle but do not offer further full cycles.
	• In women aged 40–42 who have not conceived after 2
	years of UPSI or 12 cycles of artificial insemination
	(where 6 or more are by intrauterine insemination),
	offer 1 full cycle of IVF, with or without ICSI.
	• IVF/ICSI cycle consists of:
	 IVF/ICSI cycle consists of: 1. Down-regulation of gonadotrophins: by GnRH
IVF/ICSI	 IVF/ICSI cycle consists of: 1. Down-regulation of gonadotrophins: by GnRH 2. Controlled ovarian stimulation: by FSH
IVF/ICSI Cvcle	 IVF/ICSI cycle consists of: 1. Down-regulation of gonadotrophins: by GnRH 2. Controlled ovarian stimulation: by FSH 3. Maturation of oocvtes.
IVF/ICSI Cycle	 IVF/ICSI cycle consists of: 1. Down-regulation of gonadotrophins: by GnRH 2. Controlled ovarian stimulation: by FSH 3. Maturation of oocytes. 4. Oocytes retrieval.

6. Embryo-transfer.

7. Luteal phase support: with progesterone (and cryopreservation choice offered if good quality embryos are available)

Down-regulation:

- shut down the pituitary giving a GnRH analogue, to suppress FSH & LH secretion

- to avoid premature LH surge & spontaneous ovulation
- either GnRH agonist or antagonist protocol
- always use GnRH antagonist protocol in women with

high risk of OHSS (Ovarian hyperstimulation syndrome)

rate: 40-60%/cycle

Success

cle Controlled ovarian Stimulation:

- then we inject controlled amount of FSH to induce multiple ovulation.
- By urinary or recombinant FSH and/or HMG.
- Dose depend: age, BMI, PCO presence, ovarian reserve
- Monitoring of folliculometry by USS and E2.

Triggering of ovulation:

- By urinary of recombinant HCG, 36 before oocyte retrieval.

Retrieval:

- under U/S guidance by needle connected to a test tube that we send to lab where they collect eggs.

- Fertilization and incubation
- Embryo-transfer: SET/DET

Luteal Phase support:

- Should be offer luteal phase support with progesterone till 8 weeks of gestation.

- Different form of progesterone with different routes of administration are available, RCT are taking place comparing efficacy of different forms.

Cryopreservation:

Cryopreservation of semen, oocytes or embryos should be offered to anyone who may undergo treatment that may affect his/her fertility. (e.g.: chemo for cancer).
For cancer-related fertility preservation, do not apply eligibility criteria used for conventional infertility ttt.
Do not use a lower age limit for cryopreservation for

fertility preservation in people diagnosed with cancer.

Hirsutism

Definition	 male pattern (terminal) hair growth in a female due to increased androgen production or sensitivity it is not a Dx but a manifestation 		
Hypertrichosis	generalized non-sexual (villus) hair growth (short thin), Causes: hereditary, medication, malignancy		
Virilism	 Hirsutism + other defeminization Sx: 2° Amenorrhea, male pattern baldness, Clitoromegaly, Voice deepening (irreversible) Causes: ovarian and adrenal tumors, CAH, Cushing's and acromegaly, latrogenic (mainly by danazol) 		
Causes of ↑ Androgen Level	 a. ↑ production of androgens: adrenal (Cushing's, CAH, tumors) ovarian (tumors, PCOS) b. ↑ free testosterone: ↓ SHBG, normal T, ↑ insulin due to insulin resistance (PCOS) c. Increase local activity of 5-α-reductase (5αR) d. latrogenic Hyperandrogenism causes: 75% PCOS, 15% idiopathic, 3% adrenal hyperplasia, 1% tumors ovarian, adrenal, medications, cushing's MCC of hirsutism is familial not hyperandrogenism Medications e.g.: androgens, danazol, anabolic steroids, minoxidil, phenytoin, valproate, diazoxide 		
Physiology of	 Adult hair is 2 types (terminal, villus) Hair growth is dynamic, (it goes by 3 phases): 		
hair growth	a. <u>Ana</u> gen (growing): mitosis, e.g. scalp hair, face b. <u>Cata</u> gen (ceasing) c. Telogen (resting)		

Testosterone	Normal women	Hirsutism	
level	1% Free	2% Free	
SHBG has only	19% Albumin	19% Albumin	
and only 1%			
increase in			
testosterone,	80% SHBG	79% SHBG	
but this actually		, 5 , 6 1 1 C	
means that the			
l level doubled			
	Name	Secreted by	
	Dehydroepiandrosterone	Adrenal Gland	
	(DHEA)		
	DHEAS	Adrenal Gland	
Female	Androstendione (A)	Adrenal Gland + Ovaries	
Androgens		(50/50)	
	Testosterone (T)	Adrenal Gland + Ovaries	
	produced from A conversion	Granulosa cells)	
	Dihydrotestosterone (DHT)	produced from T conversion by 5-	
		(T x 100 / DHT x 200)	
	a. Detailed Hx		
Clinical	b. Examination:		
Assessment	- Severity by Ferriman Gallw	vey Scoring System	
	- Acne, and Virilization signs	5,	
	- Acanthosis Nigricans		
	- Anarogens: Testosterone (concentration, FAI (free	
Invostigations	anurogen muex), DHEA	$\int for CAH (21, OH dof))$	
investigations	- Devamethasone sunnressi	on test/21 hr urinary free	
	cortisol (Cushing)		
	- Pelvic imaging (US. CT. MR	(1)	

Treatment	 I. Treat symptoms: i.e: remove excessive hair II. Treat the Cause: OCP: ↑ SHBG, ↓ androgen (levonorgestrel) Androgen Antagonists: 2nd line mono or + OCP in severe cases (Spironolactone, Cyproterone acetate, Flutamide, Finasteride) Cyproterone Acetate (CPA + ethinyl estradiol) Elfornithine (Vaniqa®): topical antiprotozoal, inhibit hair follicle ornithine decarboxylase enzyme, S.E (obstruction of sebaceous glands and hence acne worsening), it enhances effect of laser treatment Insulin sensitizing agents: metformin
	worsening), it enhances effect of laser treatment - Insulin sensitizing agents: metformin
	- GnRH agonists
	- Weight loss
	- Surgical

PCOS

Prevalence	5-10%, 25% of IVF patients, higher in south Asians,		
	higher in gestational diabetes, premature adrenarche		
Etiology	unknown (mostly familial)		
	ESHRE/ASRM (2 of the following + exclude other causes):		
	- Oligo and/or anovulation		
PCOS	- Hyperandrogenism (clinical) and/or		
Criteria	hyperandrogenemia (biochemical)		
	 PCO on U/S (≥12 follicles per ovary, 2-9mm and/or 		
	ovary volume (>10ml))		
	- Ovarian dysfunction: 个 androgen – by 个 LH by theca		
Pathophy-	- Hypothalamus dysfunction: 个 androgen – by 个 LH		
siology	- Insulin resistance: compensatory hyperinsulinemia, it		
	\uparrow androgen production, \downarrow SHBG in liver and \uparrow free T,		

	note : In patients with PCOS, there is selective tissue
	insulin sensitivity (skeletal muscle is resistant but ovary
	and adrenal are sensitive).
-	- Obesity (50%): central android
Manifest-	- Metabolic syndrome: DM II , HTN, IHD, Atherosclerosis,
ations	- Dermatological: Hirsutism, Acanthosis, alopecia, acne
	 Long-term: infertility (caused by anovulation),
	endometrium Ca, CVS, Miscarriages, PET, DM, HTN
	- TSH
	 Fasting Blood Sugar (FBS) and lipid profile
Investigate	- Prolactin (PRL): 个 in 40% 2 ^{ry} chronic estrogen
	- Free Androgen Index
	- FSH and Estradiol (to exclude POF / FSH >25 + E2 <30)
	A. Weight Reduction:
	 Life style, Bariatric surgery
	 Medications: Sibutramine (central), Orlistat (peripheral)
	B. Ovulation Induction: by this <u>chronological order</u> :
	 Wt. reduction to reach optimal BMI around 19-30.
	2) <i>Metformin</i> with 8% rate of success.
	3) <i>Clomiphene</i> for 6 cycles with 75% ovulation rate.
	4) Letrozole aromatase inhibitor.
Treatment	5) Either FSH & LH injections or Laparoscopic Ovarian
	Drilling (LOD – Success: 80% to ovulate, and 60% in
	pregnancy, it applies the Rule of 4: 4 punctures, 4 mm
	depth, 4 seconds on cautery and on 40 Watt)
	6) <i>IVF</i> (In vitro fertilization) as a last resort.
	N.B.:
	- GnRH should not be offered with ovulation induction
	for risk of OHSS, No evidence for the role of adjuvant GH.
WHO	1) Hypothalamic-pituitary-gonadal: The MICC of
Classify	nypothalamic is stress by diet and exercise.
	2) Hypothalamic-pituitary-ovarian (PCOS): cover 75%
(imp!!)	3) Gonadal Tallure: less than 5%.

	35
	Fibroids
	• Fibroids are leiomyomas (benign tumor of smooth muscles)
	Incidence rate 30%
	 Life-time risk 80% (by histopathology)
Info	 Estrogen – Dependent
	 Common age pf presentation: 30's
	 Growth rate: 1cm/yr if rapid consider sarcomatous changes
	(<0.2%)
	 Nulliparity (high estrogen)
	• Early menarche and late menopause (longer estrogen duration)
Risks	 Afro-Caribbean (black racial)
	• PCOS
	 Obesity (High estrogen due to peripheral conversion
	(aromatization) of androgen to estrogen
	 Multiparity: post-partum remodeling of the uterus leads to
Protecti	shedding off
ve	• Smoking
factors	 Late menarche, early menopause
	• OCP
	 According to <u>Site</u>:
	- Intramural (MC 70%)
	- <i>Subserosal</i> (most benign)
	 Cervical: difficult to treat, highly vascular, treated by
Classific	hysterectomy
ation of	- Broad ligament
fibroid	- Parasitic: avulsuion of subserosal
	- Submucosal (3 grades):
	Grade 0: 100% within the cavity
	Grade 1: 50% - 100%
	Grade 2: <50% (the remaining within the myometrium – these are
	the most difficult to resect)

	 Mostly asymptomatic (70%!) Most common presentation: AUB (menorrhagia: increase in
	amount, duration or both at regular interval, if irregular this is
C/P	menometrorraghia)
	• Other S/Sx: Pressure Sx (urinary, pelvic and back pain), Secondary
	infertility, recurrent miscarriages
	• This mainly depends on what? Site, Size of the fibroids
	• C/P
	• U/S
Dx	• MRI: site, size, number
	Hysteroscope
	• Gross
How	• Endometrial stretching (most acceptable, due to increase of the
does it	cavity size – increase surface area)
cause	 Increase vascularity (the tumor needs more blood to grow)
AUB	Uterine contractibility
	 Endometrial hyperplasia (due to hyper-estrogenic state)
	 Interfere with implantation (and if it occurred, it increase
How	miscarriage risk)
does it	Anatomical distortion (indication for surgical Mx)
cause	• Tubal obstruction (mechanical) (present sperm passage)
infertilit	Hypervascularity (sick endometrium + abnormal hormones)
У	• Intramural causes infertility if: > 4cm, and due to abnormal
	normonal changes
	 According to Age, size, site, number, SX, tertility wisnes for example read these scenarios;
	 IOI example read these Scenarios. 1. Pt young in reproductive age, sumptometics go for surgery.
How to	2. Pt premenongusal: modical till mononausa (hyposstrogonia
	ctate it will chrink on its own)
IVIX	3. Pt during waiting list for surgery: medical My
	A Pt young small fibroids with severe ALIR and don't want
	hormonal therapy: NSAIDs (20-50% decrease the bleeding)
A. Expectant:

- Asymptomatic, small, close to menopause
- Follow up fibroid growth every 3-6 months

B. Medical:

- Indications for medical Mx: close to menopause, unfit for surgery
- Progesterone (cyclic one)
- Mirena (the best choice)

• *NSAIDs* (decrease the menstrual loss 20-30%, used when the only Sx is menorrhagia, and in small fibroids

• GnRH Analogue (Decapeptyl):

a. when is it used?

- if you are close to menopause or pre-op (shrink the size by 50%)

Mx b. For how long is it given?

Options

 3 months (max shrinkage at 8-12 weeks, after that it will not shrink & due to the hypoestrogenic state (you will enter pseudomenopause > osteopenia > osteoporosis (risk after 9 m)
 c. How does GnRH act?

- it causes hypogonadotropic hypogonadism, which will shut down the pituitary, NO FSH, NO LH so no folliculogenesis and no estrogen so hypoestrogenic state will occur and this decrease the need of blood supply, so low vascularity leads to low size eventually decreasing the Sx (decrease intra-op bleeding, and decrease the decline in Hb which will provide time to correct the anemia) *d. What is the only bad effect of decapetyl?*

- it will obliterate the cleavage plate capsule, there is a fake capsule comes from the compression of the growing myoma on surrounding tissue, and when shrinkage occur adhesions occur in the cleavage plate capsule

- also note that this is a very expensive medication

C. Surgery:

- Myomectomy: indications:
- a. Young age b. Sx
- c. Fertility wishes d. Pt desire
- Hysterectomy: if completed family, why hysterectomy?
- a. High recurrence rate: 40%
- b. Risk of uterine rupture
- c. Risk of placenta previa
- Uterine artery embolization:
- risk of premature ovarian failure 5%
- indications: single, large fibroid, unfit for surgery
- Hyphe: Radiofrequency ablation > otolysis
- *Transcervical resection of fibroids* (*TCRF*) by hysteroscope through the cervix in submucosal fibroids

Endometriosis

Presence and growth of endometrial glands, stroma outside the endometrial cavityCan be found in any part, most commonly throughout the pelvis (ovarian, uterosacral ligament 65%)DefinitionOvarian endometrioma 5% of infertile women scheduled for IVF, Ovarian: bleeding and scarring will occur which will cause Chocolate cyst (accumulated altered blood), this is very harmful to the ovarian reserve as it destroys healthy tissue.Pathophys Theories• Coelomic metaplasis (the most acceptable one) • Implantation theory, • Embolization theory • Caucasian (5-10%) RFRF• Nulliparous • Higher socioeconomic classes• Pelvic pain (MC), Dysmenorrhea & Deep Dyspareunia • Subfertility (might cause infertility – 30-40%)Hx• Bowel habit alteration • Hematuria • Might be asymptomatic! • Abdominal: mass, ruptured cyst (acute abdomen)PE• Speculum: bluish discoloration of cervix or vagina		Endometriosis			
Outside the endometrial cavityCan be found in any part, most commonly throughout the pelvis (ovarian, uterosacral ligament 65%)DefinitionOvarian endometrioma 5% of infertile women scheduled for IVF, Ovarian: bleeding and scarring will occur which will cause Chocolate cyst (accumulated altered blood), this is very harmful to the ovarian reserve as it destroys healthy tissue.Pathophys• Coelomic metaplasis (the most acceptable one) • Implantation theory, • Embolization theory • Caucasian (5-10%) RFRF• Nulliparous • Higher socioeconomic classes• Pelvic pain (MC), Dysmenorrhea & Deep Dyspareunia • Subfertility (might cause infertility – 30-40%)Hx• Bowel habit alteration • Hematuria • Might be asymptomatic!• Abdominal: mass, ruptured cyst (acute abdomen) • Speculum: bluish discoloration of cervix or vagina		Presence and growth of endometrial glands, stroma			
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• Speculum: bluish discoloration of cervix or vagina		 Abdominal: mass, ruptured cyst (acute abdomen) 			
	PE	 Speculum: bluish discoloration of cervix or vagina 			
 Bimanual: pathognomonic sign: fixed retroverted 		 Bimanual: pathognomonic sign: fixed retroverted 			
uterus that can't be moved due to severe adhesions		uterus that can't be moved due to severe adhesions			
Adnexal: ovarian mass		• Adnexal: ovarian mass			

Infertility	 Pathophysiology (Causes): 1) Presence of abnormal macrophages (main cause) 2) Extensive adhesions & distortion of anatomy 3) Ovarian endometrioma leads to poor ovarian reserve (Ovulatory and endocrine abnormalities) 4) Impaired implantation 				
	 Mx of infertility in endometriosis: Medical Mx: no role (teratogenicity/delay conception) Mild-moderate: Surgical Mx 				
	- Severe: IVF				
Investigate	 Laparoscopy: gold standard for staging and Mx Classify: mild, moderate, severe, deep, superficial US: ovarian endometricma 				
	• MRI				
	 Depend on: age, fertility plans, Sx, site 				
	 Conservative: simple analgesics, avoid hormonal Rx 				
	• Medical:				
Мх	 Aim is to achieve amenorrhea atrophy & stop growth COCP, progestogen, GnRH agonist 				
	- all these medications help in reducing pain				
	- high recurrence rate after Rx cessation				
	 Surgical: drainage & stripping, peel & scrape the cyst Laparoscopic ablation and excision (effective for pain – high improvement rate) Laparoscopy: Dee-roofed (striping & is indispensable) and excision TAH + BSO (total abdominal hysterectomy + bilateral salpingo-copherectomy) 				

Adenomyosis

Adenomyosis		
Definition	 Presence and growth of endometrial glands and stroma 	
	within the myometrium (cystic changes)	
RF	• High parity (>5)	
	 Curettage of the uterus (Hx of D/C & E/C) 	
	• HMB	
Hx	 Progressive dysmenorrhea 	
	Deep dyspareunia	
PE	 Symmetrically enlarged uterus that may be tender 	
Dx	• Histologically after hysterectomy (only way for definitive Dx)	
	MRI maybe helpful	
Mx	 Hormonal therapy: limited response 	
	Hysterectomy: often required	



Endometrial Cancer

	Estrogen dependent disease				
Info	 Normally progesterone balances the estrogen effect 				
	• Endometrium: the part that responds to hormones				
	 MC type: adeno Ca "unopposed estrogen" 				
	 Diagnosed early: because it occurs after menopause 				
	mainly, and suddenly they have bleeding				
	 Mean age: 61 years (75% post-menopausal) 				
	 50% Complex with Atypia! highly progress to cancer 				
Etiology	↑ Estrogen (theca tumors, estrogen use, unopposed)				
	个 Estrogen - 个 growth – hyperplasia simple (normal				
Sequence	cells) – Complex hyperplasia (abnormal) – Atypia –				
	Malignancy (if crossed basement membrane)				
	o Type of patient:				
	 Nullipara/low parity 				
	 middle or upper social classes 				
	• 个 BMI				
	 Early menarche and late menopause 				
	White people				
	o Associated factors:				
RF	• DM				
	• HTN				
	• Fibroids				
	• PCOS				
	• Tamoxifen use				
	- Tamoxifen: estrogen antagonist on breast, agonist on uterus				
	 Infertility, arthritis, thyroid disease 				
	 Pelvic irradiation 				
	 FHx of breast, ovarian, colon cancer 				
	(Lynch syndrome – HNPCC)				

Protective	 Smoking (as Crohn's) 		
Factors	 Oral Contraceptives (mainly progesterone) 		
	 Progesterone use 		
	 Endometrioid Adenocarcinoma 50% 		
	 95% adenocarcinoma, 5% SCC are well differentiated 		
	than anaplastic		
	 Associated: pyometra (uterine infection) or hematometra 		
Types	 there are 2 histopathological types: 		
	Type 1: due to unopposed estrogen:		
	- good prognosis		
	- e.g. adenocarcinoma		
	Type 2: not related to estrogen		
	- bad prognosis, 5YS: <50%		
	- e.g.: serous, clear cells		
Spread	 MC Route: Direct invasion 		
	 Types: Direct, LN, Blood (least common) 		
	 Postmenopausal bleeding (R/O Endometrium Ca) 		
	• MC Sx is AUB! (20%):		
Hx	 Perimenopausal menstrual irregularities 		
	 Blood stained vaginal discharge 		
	 Heavy and irregular vaginal bleeding 		
PE	 mostly entirely normal 		
	 check surrounding structures (METS, extension) 		
	 US: for thickness, invasion and LN staging, thickness: 		
	thin (≤ 4mm) reassure her / if thick (>4mm): biopsy		
	• CBC, LFT, RFT		
	• CXR		
Investigate	 Cytology brush (to analyze cells) 		
	 Endometrial sampling: 		
	a. Sample for histology: Piplle		
	b. Examination under anesthesia and D/C		
	c. Hysteroscopy and biopsy		

	• Suspicion of METS: proctoscopy, sigmoidoscopy			
	cystoscopy, bone scan			
Staging	 Staging laparotomy for Endometrium Cancer: Hysterectomy + Bilateral salpingo-opherectomy (BSO) + Abdomen fluid sample (Peritoneal wash cytology) + LN (pelvic sampling – not always) Prognostic factors included in surgical staging: histologic type, differentiation, stage, depth, result of peritoneal wash, LN METS, adnexal METS, others, Ploidy and GF, age and body morphology 			
Degree of Differentia- tion	 it is based on the degree of abnormality of glandular architecture and the degree of nuclear atypia G1= 5% or less of non-squamous or non-morular solid growth pattern (well differentiated, glandular pattern similar to normal endo glands) G2 = 6-50% of a non-squamous or non-morular solid pattern growth (has glandular structures mixed with papillary, occasionally solid areas) G3 = >50% (glandular structures has become permanently solid with a relative Paucity of endometrial glands) 			
	A T confined to uterus no or < ½ myometrial			
	Ι		invasion (mc!)	
Carcinoma	$\mathbf{B} > \frac{1}{2}$			
of the	II cervical stromal invasion, not beyond uterus			
Endometri-	A T invades serosa, or adnexia			
um (FIGO)	III B invades vaginal &/or parametrial involvement			
Classification	C1 pelvic node			
		C2	Para-aortic node	
	IV	Α	bladder and/or bowel mucosa	
		В	Distant METS	

• In General: TAH + BSO + PW + LNB

o Information:

• anything beside 1A well differentiated (surgery only), you must give radiotherapy & chemo for stages 3, 4

• laparotomy with a lower midline abdominal incision for better exposure for staging

• pelvic and para-aortic lymphadenectomy are indicated in high RF cases (grade, vessel/myometrial invasion, cervical/adnexal involvement) or if its enlarged LN, or if its serous/clear Ca

• pelvic and para-aortic lymphadenectomy are indicated where high RF (grade, invasion, enlarged LN, serous or clear Ca) are present

- usually external radio followed by intracavitary radio
- Role of chemotherapy is limited

o Post-operative radiotherapy indications:

- Moderate, poor differentiation (G2,3)
- other types than adenocarcinoma
- Invasion of myometrium
- (+) peritoneal wash
- (+) LN

Mx

o Radiotherapy may be used as:

- adjuvant to surgery: stage I
- radical treatment: stage ILIII
- palliative treatment: stage IV

o Adjuvants to hormonal therapy:

- Medroxyprogesterone acetate
- GnRH analogues

Others	 o Synchronous ovarian and endometrial cancer: found in 5% of women with endometrial carcinoma & 10% of women with ovarian cancer o Inoperable patients: For women with presumed stage I disease who are unfit or unwilling to have surgery, primary radiation therapy may be acceptable. o Fertility preservation: Women with stage I, grade 1 endometrial carcinoma who wish to preserve fertility may be candidates for treatment with progestin therapy, megestrol acetates evaluation prior to medical therapy (eg, dilation and curettage, imaging studies) is necessary to try to confirm that the locion is low grade, low stage disease
	confirm that the lesion is low grade, low stage disease
	Oterine Sarcomas
Information	 tissue elements within the endometrium. Compared to common endometrial carcinomas it behave more aggressively and has poorer prognosis. Dx by histology after myomectomy/hysterectomy
RF	 Race (African-American – x2) Tamoxifen use (long-term) Pelvic Radiation Hereditary conditions (HLRCC)
Tupos 9	 Homologous: the tissue that is malignant is normall present in uterus (e.g. endometrial stroma, muscle) Hotorologous: tissue is not normally present (hono)
Types & Classification	 Malignant Mixed Mesodermal T (Carcinosarcoma): should be managed as grade III endometrial Ca 50% have distant METS at the time of Dx

• Endostromal Sarcomas and Leiomyosarcomas: are the MC pure uterine sarcomas

o Leiomyosarcoma:

- MC 55 yo, poor prognosis
- Sx: pain, bleeding, mass
- most cases not diagnosed pre-op
- Treatment: TAH+BSO (low response to chemo)
- Arises:
- denovo from uterine muscles
- rarely from previous benign leiomyoma (fibroid <1%)
- you can differentiate between malignant/benign by:
- mitotic count (>10 per 10 high power field)
- presence or absence of atypia
- presence or absence of coagulation necrosis

o Endometrial stromal tumors: (3 types)

- I. Nodule: benign, rare, hysterectomy(curative)
- II. Sarcoma: low grade > TAH+BSO
- III. Undifferentiated: pre-menopause > TAH+BSO

o Adeno-Sarcomas:

- low grade, post-menopausal
- benign epithelial components + malignant mesenchymal components (low grade endo-sarcoma)

Ovarian Pathology

Cyst events	 Rupture, hemorrhage, torsion, infection 			
	 With adnexal pathology, there may be referred pai 			
	down the cutaneous distribution of the obturator nerve			
	(inner side of thigh dow	n to the knee).		
	Ovarian cancer is the	2 nd most common gyne		
	malignancy after uterin	e and 5 th in women		
	• Majority of ovarian Ts	are epithelial in origin (originate		
	from fallopian tubes)			
Notes	 1ry ovarian neoplasm 	s are commonly in: 40-60s		
	 Teratomas & Sex cord 	l T mostly before puberty		
	 Overall 5YS is 35% 			
	 Silent killer: asymptor 	matic & Dx in advanced age-75%		
	 MCC of death from G 	yne malignancy		
	o In Children:			
	 Cysts, teratomas MC benign/ Germ cells MC malignant 			
	Torsion MC complication 33%			
	Pregnancy test			
	US (torsion: enlarged ovary/mass, free fluid)			
	Urinalysis and culture			
Investigate	• FBC, urea, electrolytes			
	• LFT, coagulation screen			
	• CA125 (only if you suspect malignancy)			
	 Swabs for infection (PID suspicion) 			
	• Doppler (torsion susp			
22	• Ectopic (mc)	• Diverticular disease		
DDx	Appendicitis			
	• PID	Renal colic/urinary calculi		
	Pelvic abscess	Fibroid degeneration		
M X	• Expectantly with analgesia and observation, rescan			
	atter 6 weeks			

	 Laparoscopy if: hemodynamic compromise, uncertain Dx, or likelihood torsion, no Sx relief within 48 hr COCPs for cyst formers Ovarian cyst in pregnancy intervention indications: Sx relief, malignancy suspicion Dermoid cysts – 50% cystadenomas (mostly benign) Conservative MX is appropriate
	 o Classification A (Nature) Functional: follicle, corpus luteum, theca lutein Inflammatory: tubo-ovarian abscess Benign tumors: Fibroma, brenner T, benign teratoma Malignant: Malignant teratoma, cystadenoma /cystadenocarcinoma (>50% for serous, 5% mucinous)
Types of Cysts/ Tumors	 o Classification B (WHO classification – origin): o Epithelial Tumors: Serous Mucinous Endometroid (Endometriosis??) Clear cell (mesonephroid) Transitional cells (Brenner) Mixed Undifferentiated/unclassified o Germ cell tumors: Dysgerminoma (MC malignant) Endodermal Sinus Tumors (Yolk sac tumors) Choriocarcinoma Teratomas Gonadoblastoma Sex cord Stromal Tumors: Granulosa-Stromal T (Granulosa, thecoma-fibroma) Anroblastomas: Sertoli-leydig cell tumors Fibromas: ascites + Hydrothorax = meigs syndrome

	• 2ry tumor with gastric origin (signet ring cells)					
Krukenberg	• mucinous METS					
	 mainly bilateral (other mucinous Ts are unilateral) Abdominal GL Urinary Constitutional Sy + SOB 					
Ovarian Ca	 Abdominal, GI, Urinary 	 Abdominal, GI, Urinary, Constitutional Sx, + SOB 				
Sx	Granulosa cell T often	present early, more acutely				
	• Sporadic, unknown etiology					
	 BRCA1/BRCA2 & HNPCC groups most significant 					
	 10% familial: 3 familial 	syndromes:				
	- familial breast-ovarian	cancer syndrome (BRCA1/2)				
	- site-specific ovarian car	ncer (BRCA1/2)				
RF	- cancer family syndrom	e (Lynch type II)				
	 Age: peak > 60 yr 					
	Reproductive Hx: men	arche, nulli,				
	 Fertility drugs 					
	Personal breast cancer Hx					
	Talcum powder (Baby powder)					
Protective	 Multiparity 	 Hysterectomy 				
Factors	Oral contraceptives	Lactation				
	Tubal ligation	Bilateral oopherectomy				
	• History, PE (ask about other systems for METS)					
	• US: TA/TV (limitations:	poor PPV, normal ovary size)				
	Iumor markers: CA 125: poor specifity, sensitivity					
	Color-flow Doppler CT (MAD)					
	• CT/MRI					
Diagnostic	 CBC, urea, electrolytes, LFT 					
tools						
	Risk of malignancy index (RMI): components:					
	$\mathbf{RMI} = \mathbf{U} \times \mathbf{M} \times \mathbf{CA125}$					
	- U: ultrasound findings (1 point for each):					
	* Multi-locular cyst * Bilateral					
	* Solid areas * METS * Ascites					
	- M: menopausal status (post = 3 / pre = 1)					
	- RMI <25 low risk, 25-200 moderate, >200 high					

	Serous	CA 125	Endodermal sinus	α-FP & AT		
Tumor	Mucinous	CA 19-9	Choriocarcinoma	B - HCG		
markers	Granulosa	Inhibin	Dysgerminoma	LDH. Alkaline		
			- /-0	phosphatase		
US Findings	Benign T	umors	Malignant	Tumors		
	 Unilatera 	I	 Bilateral 			
	 Unilocula 	r	 Multilocular 			
	• Thin-wall		 Thick-wall 			
	 No papilla 	ae	 Present papillae 			
	 No solid a 	areas	 Mixed echogenic 	ity (solid areas)		
	 vascularit 	ty	Greater vasculari	ty, angiogenesis		
Spread	•Seeding, I	ymphatics	s, blood (sarcoma, te	eratoma), direct		
	o Surgery:	for accura	te staging (surgico-	pathological)		
	• TAH+BSO					
	 TAHBSO+ 	 infracolic 	olic omentectomy+peritoneal cytology			
	 Unilatera 	l salpingo	-oherectomy (to res	erve fertility)		
	 Cytoreductive/debulking 					
	 Peritonea 	al METS re	duction			
	 Second Ic 	ook laparo	tomy			
	 Laparosco 	opic surge	ry			
	Conserva	tive surge	ry			
Мх	• Fertility c	onserving	surgery			
	o Chemo : (Dvarian ca	ncer is a chemo-sen	sitive solid T		
	Adiuvant					
	• Combina	tion				
	• Neo-adiu	vant				
	• Agents:					
	- Alkylating: Cisplatin, Carboplatin					
	- Plant alkaloids: Pacilitaxel (MC!)					
	- Anti-canc	er antibio	tics			
	- Antimetal	bolites				

	 Life-after treatment: follow up for reassurance, and recurrence, done by CA125, MRI, CT (as palliative) Chemo SE: N/V, fatigue, sore throat, ototoxicity (cisplatin), peripheral neuritis, nephrotoxicity, myelosuspression with infection risk, pulmonary toxicity (bleomycin)
	o Radio
	o Surgery/Plat/Taxol remains standard care for ovarian cancer since 1990s
Ovarian germ cell T (GCTs)	 Benign/malignant MC ovarian cancer in the 1st two decades of life Racial: ↑ in African, South & East Asian & Hispanic RF for MOGCTs: gonadal dysgenesis, sexual immaturity & presence of abnormal karyotype With multimodality ttt: excellent prognosis and preservation of fertility MOGCT Radio: Dysgerminomas are radiosensitive Chemo + toxicities + radio are no longer forms a part of routine treatment
1 ^{ry} fallopian tube Ca (FTC)	 BRCA-1 and BRCA-2 90% of FTCs serous papillary adenocarcinoma 40–60 years (median age 55 years) Sx are vague and non-specific but similar to EOC Latzko's triad of Sx – intermittent profuse serosanguinous vaginal discharge, colicky pain relieved by discharge and abdominal or pelvic mass Rx as EOC

SE	Carboplatin	Paclitaxel
Thrombocytopenia	\checkmark	X
Neurotoxicity	X	✓
Alopecia	X	✓
Nephrotoxicity	\checkmark	✓
Neutropenia	\checkmark	\checkmark
N/V	\checkmark	\checkmark
Hypersensitivity	X	\checkmark
Arthralgia	X	\checkmark
Myalgia	X	\checkmark
Cardiac SE	X	X
Diarrhea	X	×

	FIGO ovarian cancer staging
Note	From stage I c: is considered advanced ovarian cancer
<u> </u>	Tumor confined to ovaries
II	One or both ovaries + pelvic extension (below pelvic brim) or
	primary peritoneal cancer
	One or both ovaries with cytologically or histologically
III	confirmed spread to the peritonium outside the pelvis and/or
	metastasis to the retroperitoneal lymph nodes
IV	Distant METS excluding peritoneal METS
Gra	ding by "FIGO" for ovarian cancer – Epithelial T sub-classified
Gx	Grade cannot be assessed
G1	Well differentiated
G2	Moderately differentiated
G3	Poorly differentiated

Cervical Cancer

Info	 It doesn't occur suddenly, it starts as dysplasia then cancer (takes time maybe years unlike endometrial Ca) 50% of cases are diagnosed btw ages 35-55, 20% >65 y The cancer depends on the type of the patient, stage low incidence in Muslims, Jews 	
Types	 SCC: 80-90% outside of the cervix into the vagina, likely to be invasive Adeno Ca: 10-20% inside the cervical canal (columnar epithelium) arise from <i>glandular</i> epithelium (glandular tumors) are not detectable by screening and associated with skip lesions & require radical surgery Mixed 	
Sx	 MC is vaginal bleeding and abdominal pain abnormal discharge coital pain, bleeding after intercourse or pelvic exam menses is longer, heavier 	
RF	 HPV (Main RF! - 16,18 high risk / 6,11 low risk) Sexual behavior, Diet Smoking, Low socioeconomic status HIV, Chlamydia Pregnancy, OCP Diethylstilbestrol (DES) 	
Prevent-	Avoiding the RF	
ion	 Pap test: 3 y after 1st intercourse or by age 21 <u>annually</u> 	
Dx	 History: many women are symptomatic, with abnormal cx smear complain of abnormal vaginal bleeding (postmenopausal, perimenopausal, post coital) blood stain vaginal discharge 	

• Examination:			
 always exam the cervix (histology, biopsy - cuscu's 			
speculum), nothing is found in early stages			
 PV/PR to determine stage 			
- don't see but suspect then do <i>pap smear</i> , more			
suspicion <i>colposcopy</i>			
• Tools:			
 <u>Coloposcopy</u>: to examine the cervix 			
- <u>Cerv</u>	 <u>Cervical biopsies</u>: colposcopic, endocervical curettage, 		
cone	liopsy		
0	In situ		
1	Invaded cervix, no spread, 5YS: 80%		
A1	Confined to the cervix, Dx by microscopy wi	th	
	invasion of <3 mm in depth + lateral spread <7	mm	
A2	Confined to the cervix, Dx with microscopy w	vith	
	invasion of >3 mm & <5 mm + lateral spread <	7mm	
B1 Clinically visible lesion or greater than A2, < 4 cm		cm in	
	greatest dimension		
B2	Clinically visible, > 4 cm in greatest dimension	on	
2	Spread nearby within pelvic, 5YS: 50-60%		
A1	Involvement of the upper 2/3 of vagina, with	out	
	parametrial invasion, <4 cm in greatest dimen	sion	
A2	> 4 cm in greatest dimension		
B	With parametrial involvement		
3	Spread to the lower part of the vagina, 5YS: 30	-40%	
A/B	Unchanged		
A/B 4	Spread to nearby organs, METS, 5YS: 4%		
A/B 4 A/B	Spread to nearby organs, METS, 5YS: 4% Unchanged		
A/B 4 A/B	Unchanged Spread to nearby organs, METS, 5YS: 4% Unchanged Unchanged irect Lymphatic Dissemination (late)	
	 Exan alway specul PV/P don't suspici Tools Colop <u>Colop</u> <u>Colop</u> <u>Colop</u> <u>Colop</u> <u>Colop</u> <u>Colop</u> <u>Colop</u> <u>Colop</u> <u>Colop</u> <u>Tools</u> <u>Tools</u>	 Examination: always exam the cervix (histology, biopsy - cuscu's speculum), nothing is found in early stages PV/PR to determine stage don't see but suspect then do <i>pap smear</i>, more suspicion <i>colposcopy</i> Tools: <i>Coloposcopy</i>: to examine the cervix <i>Cervical biopsies</i>: colposcopic, endocervical curettag cone biopsy Invaded cervix, no spread, 5YS: 80% A1 Confined to the cervix, Dx by microscopy wi invasion of <3 mm in depth + lateral spread <7 A2 Confined to the cervix, Dx with microscopy wi invasion of >3 mm & <5 mm + lateral spread <7 B1 Clinically visible lesion or greater than A2, < 4 con greatest dimension B2 Clinically visible, > 4 cm in greatest dimension B2 Spread nearby within pelvic, 5YS: 50-60%	

• Mx Options:

- <u>Surgery</u>:

a. pre-invasive: cryosurgery, laser, conization

b. invasive: simple hysterectomy, radical hysterectomy + pelvic LN

- <u>Radiation</u>

- <u>Chemotherapy</u>

• Surgery advantages:

- ovary presentation (radiotherapy will destroy them)

- chance to save sex function (radio: vaginal stenosis 85%)
- Psychological feeling of removing the disease
- more accurate staging and prognosis

• Surgical complications:

Мх

- Hemorrhage: primary or secondary

- injury to bladder, ureters
- bladder dysfunction
- fistula, lymphocele
- vaginal shortening

• P/O XRT Indications after Wertheim's hysterectomy (Stage I. IIa):

- positive pelvic LN

- tumor close to resection margins and/or parametrial extension

• Radiotherapy:

- Stage IIb,III

- Radical radiotherapy, external radiation (teletherapy), intracavitary radiation (brachytherapy)

 in some cases of stage IIa/b radio + chemo to be given then simple hysterectomy

	Recurrent disease Mx:		
	I. Local: radiation (if not used), pelvic exenturation		
	II. Distant: chemo		
	 On completion of treatment all patients are given a 		
	vaginal dilator to use until vaginal mucosa healed, this		
	prevents vaginal stenosis		
	 Premenopausal patients commenced on HRT: 		
	 post hysterectomy-Extraderm skin patches 50 mg 2/w 		
Follow up	- No hysterectomy- Cycloprogyn 1mg daily.		
	 <u>The patient to be seen 1/12 post-treatment</u>. 		
	3 monthly for 2 years. 4 monthly for 3rd year. 6 monthly		
	until 5years. Then yearly all her life.		
	 Patients with stage I and II disease treated with radical 		
	radiotherapy will be assessed by EUA approximately 3		
	months after completing ttt.		
What	 HPV test and vaccine, radical trachelectomy, other 		

new in ttt clinical trials



Pap Smear • SCC Age peak 35 – 55 Cervical RF: HPV (16/18/31/33/45), smoking, HIV, Chlamydia, diet, OCP, Multiple pregnancies, low socio-economic status, FHx Cancer • S/Sx: AUB, Vaginal discharge Good prognosis • Preventable Prevention Primary: lower the RF, HPV, folate, vitamens • Secondary: Pap smear Info Can be done in the clinic Best taken across transformation zone (squamocolumnar junction) **Disadvantag** • False negative: 50% Sensitivity for CIN detection: 50%, after 3 years it become: 87% es Depends on the technique • start at age of 25 Screening • do it every 3 years until 49 UK Guidelines • then every 5 years until 65 at 65 if there was 3 consequence negative smears, you can stop it Categories Normal Inflammatory of cells Infection Dysplasia or cancer Avoid intercourse, douching, or using any vaginal medicines or spermicidal foams, creams or jellies for two days before having a Pap smear, as these may wash away or obscure abnormal cells. How to prepare for Try not to schedule a Pap smear during your menstrual period. It's best a pap smear to avoid this time of your cycle, if possible. Why? Menstrual blood can obscure the visibility of the **cervical** cells collected in the sample, which can lead to inaccurate results Gloves, Speculum, Lubricant • Collecting device: - Spatula (wooden, plastic): rotate 360° Tools - Endocervical brush: rotate 90° - 180°

- Cervical broom: rotate 360° x 5
- Sterile labeled container for the sample/slide, fixative material

A. Conventional pap smear:



B. Liquid-based thin layer cytology:

Cytology Methods

- collecting device is placed into a liquid fixative solution and rotated in the solution .When the liquid is processed by the cytology lab, loose cells are trapped onto a filter then plated in a monolayer onto a glass slide.



					60
	Conventional Pap Sme	ar	ThinPrep	Pap Test	
Which is	Majority of cells not ca Non-representative tra	aptured	• Virtuall	y all of sample is	
	Clumping and overlap	ping	 Randor transfer 	mized, representat	ive
	 Obscuring material 		• Even d	istribution	
			• Minimiz	tes obscuring mate	erial
	Score Negative	interpretatio	on De infectio	ons	
	Atypical squamous cells • ASC		JS (undet	ermined)	
Cytology	(ASC)	• ASC-H	l (cannot	exclude HSIL)	
(Bethesda	Low-grade squamou	us CIN 1			
System)	intraepithelial lesion (I	LSIC)			
	High-grade squamous CIN 2,3, CIS intraepithelial lesion (HSIC)				
	Cancer	, Histolog	gy shows	invasive cance	er
	Histology	CIN 1	CIN 2	CIN 3	
Histology	Normal	Very mild dysplasia Mild dysplasia	Moderate dysplasia	Severe dysplasia Cancer in situ	
(Cervical intra- epithelial					
Neoplasia (CIN))					

Low-Grade SIL

Cytology

High-Grade SIL

-	- used in ASC-US		
-	$_{-}$ repeat pap at 4-6 month intervals until there are 2 consecutive negative		
	- repeat pap at 4-6 month intervals until there are 2 consecutive negative		
	paps If a repeat pap is again ASC-US or worse then do colposcopy		
Mx of			
abnormal	HPV DNA typing:		
PAP smear	 also used for ASC-US & age >25 year 		
-	 - if negative then follow up, If also ASCUS then colposcopy 		
	Colposcopy evaluation and biopsy:		
-	- LSIL & Age >25 year		
-	 used for uncertain patient/abnormal pap smears 		
	 Better visualization and magnification 		
Cervical	 You can take a biopsy if indicated 		
Colposcopy	• We apply acetic acid: acetowhite change (it improves visualization of		
1	the abnormal area's, it makes vascular patterns more visible)		
	 Satisfactory or not based on if you can see the whole T-zone 		



FIGURE 38-3 Algorithm for evaluation of patients with an abnormal Papanicolaou smear and a grossly normal-appearing cervix. ECC, Endocervical curettage; HSIL, high-grade squamous intraepithelial lesion; LLETZ, large loop excision of the transformation zone; LSIL, low-grade squamous intraepithelial lesion.

	Endocervical curettage (ECC):		
	 for all nonpregnant ladies to R/O endocervical lesions 		
	Ectocervical bionsy		
	 Cone Biopsy (Conization of the cervix): 		
After	- Indications:		
colposcopy	a) Pap smear worse than the histology (some sites are not biopsied)		
b) Abnormal ECC histology			
c) Lesion entering the endocervical canal			
	d) Biopsy showing microinvasive carcinoma of the cervix		
	- Risks:		
	a) Deep biopsies result incompetent cervix		
	b) Cervical stenosis		
	• HPV vaccine (Gardasil):		
Prevention	- for females in between 8-26 yo (mainly 11,12)		
by Vaccine	- 3 doses are given at 0,2,6 months		
	- costly		
	 not recommended for pregnant, lactating or immunosuppressed 		

Lower Urinary Tract Infections

o Urine: Storage and Voiding:

• Normally you don't feel the process of filling the bladder until you reach a certain level, 200ml this is called the first sensation, and until it reaches 400ml that's when you feel that you have to empty it, though you can hold it until 600ml, where you really have to go

	 Urinary incontinence: involuntary leakage
	 Urgency: the detrusor muscle suddenly contracts without
	leakage, if there was leakage , then its urgency incontinence
	 Increased daytime frequency
	 Nocturia: more times to void at night
	 Nocturnal enuresis: loss of urine occurring during sleep
Storage	 Urge urinary incontinence (UUI): involuntary leakage
Sx	preceded by urgency
	• Stress urinary incontinence (SUI): leakage with effort (sudden
	increase in the intra-abdominal pressure)
	 Mixed urinary incontinence (MUI): leakage + urgency + effort
	 Continuous urinary incontinence: continuous leakage (fistula)
	 Sexual intercourse incontinence
	Giggle incontinence
Voiding	Slow-stream
Sx	 Intermittent stream (intermittency)
	Hesitancy
	 Straining to void: muscle effort to void
Post-	 Feeling of incomplete emptying
mictur.	Post-micturition dribble

o Urinary incontinence, epidemiology: they all affect the UT anatomy:

• Age • Race • Pregnancy • Childbirth • Menopause

o Urinary incontinence, Causes:

- Urinary Stress Incontinence (USI)**
- Detrusor over activity (DO)
- Fistula (vesicovaginal, urethrovaginal)
- Congenital (ectopic ureter)
- Urethral diverticulum
- Functional (immobility), Other (UTI, Fecal impaction, medication)

o Assessment of LUTS:

1) Clinical evaluation:

• Abdominal and pelvic examination: any mass can increase IAP or a prolapse that squeeze the bladder

- Neurological exam
- patients mobility and mental state
- incontinence associated dermatitis
- vulval and vaginal atrophy
- 2) Imaging
- 3) Investigations: focus on urodynamic studies
- Basic:
- Urine test (for infections)

- <u>Bladder diary</u> (voiding, frequency & volume): objective information on voids, fluid intake, volume, incontinence episodes, Rx changes

- *Pad test*: objective, non-invasive, 1hr,4hr,12hr,24hr,48hr:

** The 1 Hour Standardized Test:

o Pre-weigh pad

- o Drink 500mls
- o Rest 15 mins
- o Moderate exercise 30 mins

o 15 mins provocative exercises (laugh, jump, cough),

then you weight the pad and if:

o Positive test >2g increase

o Severe incontinence >10g increase

• Advanced:

- conventional subtracted cytometry
- videocystourethrography
- ambulatory urodynamics monitoring
- urethral pressure profilometry
- imaging studies: bladder wall thickness (BWT), MRI, voiding cystourethrography, upper renal tracts
- cystourethroscopy

o The urodynamic studies (UDS):

- Definition: studies of LUT function and dysfunction
- Why urodynamics? Bladder is a poor witness, for correct Dx, Mx
- UDS: free flow study, filling cytometry, voiding cytometry

o Uroflowmetry:

- simple, non-invasive, voided volume and flow rate
- normal study:
- peak flow rate: >15ml/sec
- voided volume: >150ml
- post void residual volume (PVRV): <100 ml

o Filling Cystometry:

- Retrograde filling of the bladder
- Filling medium is usually Fluid
- Filling rate: 100ml/min
- Performed in: supine, sitting or standing
- Pressures measured via microtip or external transducers
- By a three way catheter, you fill it with normal saline at a rate of 100ml/min , then you record the first sensation at what rate , the rate at which the patient has the desire to urinate for bladder capacity..etc

o Voiding Cytometry (Pressure flow studies):

- the relationship between detrusor pressure and the flow rate
- Obstruction: high detrusor pressure (>50cmH₂O) Poor flow (<15ml/s)
- Under active detrusor function: low detrusor pressure (<20) poor flow

o Urodynamic Dx:

• Detrusor overactivity (DO): involuntary detrusor contractions during the filling phase which may be spontaneous or provoked

- Detrusor overactivity incontinence (DOI): DO + urine leak
- Urodynamic stress incontinence (USI): leak due to \uparrow IAP in the absence of detrusor contraction

• Mixed urodynamic incontinence: DO and/or DIO + USI

o Cystourethroscopy:

- Rigid/flexible
- Confirmation of anatomy
- Visualization: calculi, tumor, diverticula
- Biopsy of urothelium to assess for chronic inflammation, cancer

o Treatment of urinary incontinence:

- Lifestyle modifications: fluid intake, drinks (caffeine, tea, coke), weight reduction (over weight on the bladder), smoking
- PFMT: strengthen the pelvic floor muscle not correcting the prolapse

o Stress urinary incontinence (SUI) Mx:

A. Conservative:

- effective, few complications
- does not compromise further injury
- useful in women who: unfit for surgery, have not completed their family, breast feeding, <6m post-partum

• Conservative measures include: PFMT (Pelvic floor muscle training), biofeedback, electrical stimulation, vaginal cones, urethral devices

B. Pharmacological Mx:

- Duloxetine:
- Potent serotonin-noradrenaline reuptake inhibitor
- \uparrow urethral striated sphincter activity by a centrally mediated pathway
- Efficacy and safety:
- Significant decrease in incontinence episodes
- Optimal effect: after 4 weeks of therapy
- Nausea: 25 %
- Useful in women awaiting surgery
- Synergistic effect with PFMT

C. Surgical Mx:

- >200 procedures
- the 1st procedure offers the best chance of cure
- Correcting only the prolapse has had a high FR, so we don't use it
- the mid urethral theory or the integral theory concept:
- Maximal urethral closure pressure is at mid-urethra
- Damage to pubourethral ligaments impairs mid-urethral support
- Mid-urethral procedures have largely replaced Colposuspension

• <u>Colposuspension</u>: it's a two stage, where you left the bladder "and thus the urethra" and the vagina by attaching them to pelvic ligaments

• <u>Sub-urethral tapes</u> (TVT – transvaginal /TOT - transopturator): tension free tape, you put it under the urethra to support it like hammock (GS!)

• Urethral bulking agents:

 - useful in: elderly, Women who have undergone previous operations and have a fixed, scarred fibrosed urethra and Women who have not completed their families Outcome (long-term follow-up (> two years))
 - Objective cure rate: 50% / Subjective improvement: 70%

o SUI NICE guidelines:

- <u>PFMT</u> of at least 3m should be offered as 1st-line Mx to all women with SUI or MUI
- *<u>Retropubic tapes</u>*: recommended where conservative Mx failed
- <u>Colposuspension</u>: recommended alternatives
- Bulking agents: considered for the Mx of SUI if conservative Mx failed

•<u>Anterior repair</u>, needle suspension procedures, paravaginal defect repair and the MMK procedure are NOT recommended

o SUI: Key points:

- Conservative Mx should be offered prior to surgery
- Duloxetine may be used in conjunction with PFMT
- Mid-urethral tape: operation of choice in primary continence surgery
- TVT and TOT procedures have similar success rates
- Urethral bulking agents offer an alternative to continence surgery

o Overactive Bladder (OAB, urge syndrome, urgencyfrequency, hyperactive or detrusor instability):

o **OAB Rx**:

A. Conservative Mx:

- Advice regarding fluid intake (1 -1.5 L / day)
- Reduce caffeine and alcohol intake
- Bladder retraining (BT): 1st you void hourly then every 2 hr then after
- 4 hr.. Etc. as a way to 'train' your bladder / Cure rate: 44 90 %
- PFMT
- B. Pharmacological Rx:
- no specific drugs act on the bladder & urethra without systemic effect (anti-muscarinic. Because it's the muscle that its overactive)
- Anti-muscarinic
- SE: dry mouth, constipation, blurred vision, insomnia
- Mirabigron: B3 agonist (not anti-muscarinic), daily dose, 50 mg, 25mg in hepatic, renal insufficiency

• DO: intravesical therapy botulinum toxin (inject it to cause relaxation)

	Antimuscareni	cs
	Advantages	Disadvantages
Oxybutynin IR	Flexible dosing, rapid onset of action, cheap	Persistence limited by dry mouth
Oxybutynin ER	Flexible dosing	Cognitive impairment
Oxybutynin TDS	Placebo rate of SE	15-20% rate of pruritus
Tolterodine ER	Well tolerated	Single dose
Solifenacin	Superior efficacy to Tolterodine ER	High rate of dry mouth at 10mg dose
Darifenacin	Low rate of cognitive impairment	High rate of constipation
Trospium	Does not cross BBB	
Propiverine	Well tolerated	Efficacious for frequency
Fesoterodine	Flexible dosing	Limited experience

OAB/DO: Neuromodulation

Outcome of neuromodulation: > 50 % reduction in Sx & 46% completely cured

o Sacral neuromodulation:

- Stimulation of the dorsal sacral nerve root in the S3 sacral foramen
- Sacral nerves contain autonomic and somatic fibers to pelvic floor muscles
- Invasive and expensive
- A useful alternative to medical and surgical therapies in patients with severe, intractable OAB prior to reconstructive surgery

OAB/DO: Neuromodulation

o Peripheral neuromodulation (PTNS)

• Posterior Tibial Nerve (PTN) originates from the same spinal cord segments as the innervation to the bladder and pelvic floor

OAB/DO: Surgical management

- 10 % remain refractory to medical and behavioural therapy
- Different surgical techniques
- Augmentation to increase bladder capacity:
- Clam cystoplasty
- Auto-augmentation (Detrusor Myomectomy)
- Urinary Diversion

OAB/DO: NICE guidelines

- Bladder retraining (BT) for a minimum of 6 wks
- If no satisfactory benefit from BT: antimuscarinics
- First line drug treatment: Immediate-release oxybutynin
- If not tolerated: darifenacin, solifenacin, tolterodine, trospium or an extended-release or trans-dermal oxybutynin
- Women should be counselled regarding the SE of antimuscarinics

OAB/DO: NICE Guidelines

- Systemic HRT should not be recommended
- Intra-vaginal estrogens are recommended for OAB in postmenopausal women with urogenital atrophy
- " In old women to nourish the genital tissue might improve the Sx " $\,$

Overflow Incontinence & VOIDING DIFFICULTIES, causes:

Neurological

- MS, Spinal injuries, CVA, brain tumors
- Prolapsed intervertebral disc, cauda equina syndrome, herpes zoster

Myogenic

- Ischemia due to acute retention, e.g. after epidural block

latrogenic

- Postoperative retention associated with long operations, epidural, PCA, high dose opiates, large volumes of IVF

- Obstructive outflow procedures as continence procedures

o Causes:

Obstructive

- Extrinsic: pregnancy, large fibroid
- Intrinsic: urethral stricture or foreign body

• Inflammatory:

- Vulval abscess
- Acute herpetic infections

Diagnosis

- Clinical suspicion
- U/S or catheterization

Management

• Immediate catheterization, catheter left in for 2 days then trial w/o catheter under strict supervision

- If retention then SPC for 2-6 wks
- Bethanechol 25mg tds
- Surgery, Rx cause
- CISC

Pelvic Organ Prolapse

-	• Cyctocele (two types: distension and replacement),
Types	• Rectocele,
	• Enterocele,
	Uterine Prolapse
	 Parity is the strongest RF
	 Maximum birth weight
	 Age, menopause (conflicting)
	 Constipation, and straining
RF	 Heavy lifting
	• Obesity
	 Chronic pulmonary disease (个 abdominal pressure)
	Hysterectomy
	 Colposuspension (enterocele)
	 Sacrospinous Fixation (anterior compartment prolapse)
	General Sx:
	 Bulge, heaviness, dragging
	- Backache
	 Vaginal dryness or irritation
	 need to push the prolapse after straining (defecation)
	- Sexual activity embarrassing or painful
Sx	• Urinary Sx:
	- Stress urinary incontinence
	- Bladder neck hyper mobility
	- Urinary frequency and urgency
	- Occult stress incontinence
	- Voiding dysfunction
	- Recurrent UTI
	- Ureters
	Rectocolo Sv:
------------	---
	- incomplete howel emptying
	- obstructed defecation
	constinution
	- consupation
	food incontinence if rostal prolance
	• For cystocele:
	-Renal US
I	- mid stream urine (urinalysis , culture)
investiga.	- cystoscopy/urethroscopy
	- urodynamic studies (Cytometry)
	• For rectocele:
	- anoscopy/sigmoidoscopy
	- BA enema
	Based on the hymen (reference point), if proximal to it
Stages	stage 1, on the same level is stage 2, & if below it stages 3
Ŭ	Stage 4: procidentia
	Cystocele, Urethrocele:
	- Urethral diverticula's
	- Skene gland abscess
	• Rectocele
DDx	- Obstructive lesion of colon & rectum (linomas, sarcomas
	fibromas)
	• Uterine prolapses:
	- Cervical elongation
	- Prolapsed cervical polyp or cervical
	- Lower uterine segment fibroids
	Pelvic floor exercise (if mild)
Мх	 HRT: for post-menopausal women
	• •

- Surgical treatment: vaginal, abdominal, laparoscopic:
- <u>Cystocele</u>: anterior colporraphy
- <u>Rectocele</u>: posterior colporraphy
- <u>Uterine prolapse</u>: vaginal hysterectomy

POP-Q System:

Aa	Ва	С
"point A of the	"point B of the anterior	"cervix"
anterior wall"	wall"	normally: 7 cm above
3 cm above hymen	6 cm above hymen	the hymen ring
gh	Pb	Tvl
"genital hiatus"	"perineal body"	"total vaginal length"
- normally: 3 - 4.5	- normally: 2 – 3.5	- normally: 8-10
- <3 narrow vagina	- <2 deficient perineum	- Short < 8
- >4.5 wide vagina		- Long > 10
Ар	Вр	D
"point B of the	"point B of the	"posterior fornix"
posterior wall"	posterior wall"	
• for anterior wall pro	olapse: look at 1 st row (Aa	<i>,</i> Ba)
 for posterior wall pr 	rolapse: look at the last ro	ow (Aa, Bp)
• for uterine prolapse	: "C" value	
 for the anterior/posterior wall: 1st degree: (-3) - (-1) 2nd degree: (-1) - (+1) 3rd degree: > (+1) 		
 for uterine prolapse: (I'm not sure about this one tbh) 1st degree: (- 6) - (-1) 2nd degree, and 3rd degree as rectocele and cystocele 		

Contraception

- FR (Effectiveness) expressed as failure rate per 100 WY
- 1 WY (Woman Years) = 13 Cycles

Me

- Pregnancy & abnormal bleeding are CI for all contraception methods

Natural Family Planning (NFP)

	o Cycle or rhythm method (Calendar method):
	- Safe method, At least 3-6 months must monitor her
	cycle, it must be a regular cycle, choose the longest cycle
	& Count fertile days & avoid intercourse in these days.
	- <u><i>E.g.</i></u> Cycle is in range of 26-28 days 26-20 = 6 , 28-10= 18
	so fertile days are from day 6 to day 18 of the cycle
	Ova is viable for 24-36 hours, sperm is viable for 3-7 days
	(3 days in vagina bcz vagina is acidic , 7 days above vagina)
	O Temperature method:
thede	- remperature increases due to progesterone, when it
ethods	increases it means a sign of ovulation so no intercourse
	o Cervical mucus method (Billing's method):
	- Mucus is thin "watery" to increase mobility of sperm. it
	becomes thick" viscous" to prevent further entry of sperm
	o Cervical palpation method:
	- Cervix is more accessible in 1 st phase of cycle, higher and
	more posterior in 2 nd all changes is to encourage
	pregnancy.
	O Minor clinical indicators of fertility:
	- signs of pre-ovulation like breast tenderness.

	o Personal fertility monitors:		
	• Monitor most fertile days in cycle and confirm ovulation		
 o Lactational amenorrhea method (LAM): Unknown mechanism Inhibition of normal pulsatile LH – anovulation >98% effective if fully, <6/12, Amenorrhoeic no medical conditions where LAM is restricted Alternative contraception when: * reduce frequency of breast-feeding * stop night feed/baby sleep through the night * separation from the baby * introducing supplements * Anxiety, stress 			
N	10A	Effectiveness	
Awa	reness	Combined methods are more effective	
Adva	intages	Disadvantages	
- might be o	nly option	- high FR	
- not medica	al (no clínics)	- rely that conception days are known	
- aware wor	nen	- long periods of abstinence (no sex)	
- enhance communication - No STI protection			
	B	arrier Methods	
- Shouldn't k	be used with oil	-based creams (use <u>water-based</u> creams)	
- Condoms p	prevent STI & HI	IV transmission so use it when on OCP	
	o Male Condo	oms:	
	- FR 3-23/100	WY	
	o Female Con	doms (Femidom):	
Types:	- FR 5-21/100	WY - STI/HIV prevention (used + OCP)	
	o Occlusive Ca	aps: Diaphragms, Cervical Caps	
	o Vaginal Spo	nges	
	o Spermicides		
	o Spermicides		

 - Client choice - Medical reasons to exclude hormonal - Intermittent/infrequent intercourse - while a new method is taking effect - protect against STI 		
	Advantages	Disadvantages
- Male c	ondoms widely available	- High FR
- Protect	t against STI	- Not acceptable in some relations
- No syst	tomic SE	- Diaphrams need clinic nitting
- No lact	ration effect	change + 4kg
- Spermi	cides give lubrication	
- ↓ risk	of cervical cancer	
	Combined Hormon	al Contraception (CHC)
Info	 Low FR if used correctly Variable dose means it changes in 1st 7 d, 2nd 7 d, 3rd 7 d tablets (try to mimic natural cycle) There is a problem of spotting so ↑ pills to ↓ spotting Because of risk of thrombosis, dose of estrogen in new tablets is 20µg. but ↓ the dose ↑ risk of other problems <u>Gestodene</u> and <u>Cyproterone acetate</u> are antiandrogenic, good for those who have hirsutism and acne (think of PCOS!) <u>Drospirenone</u> is called ושיחצי in the market. COCPs don't prevent STI so use condoms too. The worst SE is VTE (mainly in 1st year after that the risk 1/) 	
Types	o Pills (mono/bi/tri-phasic) o Patches o Vaginal Ring	
MOA	o inhibit Ovulation o Alter vaginal & cervica o Atrophic endometrium	l mucus & inhibit sperm transport n non-receptive

Advantages		Disadvantages	
- Reliable		- Minor SE (nausea, fluid retention)	
- Reversible		- 个 VTE risk (worst SE!)	
- Indepe	indent of IC	- 1 Arterial disease	
- Non-co	ontraceptive benefits	- Drug interactions	
		- Loss of efficacy by diarrhea,	
		vomiting, missed pills	
	o Menstrual cycle: Imme	ediately after the menses (up to 5	
	days), to prevent pregna	псу	
	o Amenorrhoeic : Given	immediately after you exclude	
	pregnancy and stop brea	ast feeding, to confirm pregnancy:	
When	3 weeks without intercourse and make pregnancy test if it's		
to	negative then start pills.		
start			
СНС	o Postpartum : 6 weeks after delivery, why 6 w? Because of		
	thrombosis (pregnancy is a thrombogenic status) so wait		
	until 6 w or 3 w after delivery give only progesterone		
	o Miscarriage: 7 days an	ter miscarriage	
	Combined Oral Co	ontraceptives (COCP)	
FR	0.2-8/100WY (Very low)		
Types	- Monophasic (fixed dos	e): Estrogen + Progesterone	
	- Variable dose (phasic)		
	- Majority 21 Tablets/7d	PFI (pill free interval) (1 st 7 to inhibit	
	ovulation (most important!), 14 maintain)		
Consti-	- Estrogen (<i>ethinyloestra</i>	idiol)	

tuents - Progesterone: *Levonorgestrel* (LNG)/ *Norgestrel* (NG)

Non- o It Decreases:

Contra
captive - menstrual disorders (menorrhagia, irregular bleeding: 50%,
Benefit dysmenorrhea: 40%, Premenstrual Syndrome (PMS))

 Functional <u>ovarian cysts</u> (not to manage the cyst but to prevent further cysts/ note: <u>POP have ovarian cysts as a SE</u>) Benign <u>ovarian tumors</u> Benign <u>breast disease</u> 50% <u>endometrial</u>, <u>ovarian cancer</u> (15 year after stopping), <u>but Cervical cancer risk slightly increase</u> <u>Colorectal</u> cancer Protective against RA, thyroid disease, duodenal ulcer VTE Major SE Migraine Cancer (breast, cervical, liver) <u>Estrogen SE</u> Progesterone SE Acne 		(not to manage the cyst but to e: <u>POP have ovarian cysts as a SE</u>) n cancer (15 year after stopping), <u>ahtly increase</u> yroid disease, duodenal ulcer iver) <u>Progesterone SE</u> - Acne - Greasy skin/hair
- Weigh	' ⁶ t gain	- Hirsutism
- Nause	a	- Depression
- Non-in	fective vaginal discharge	- Loss of libido
- Heada	che	- Vaginal dryness
- Chloas	ma (Melasma): dark skin	vaginar aryness
discolor	ation	
- Photos	ensitivity	
1110100	Absolute Cl	Relative Cl
- Past or	r present CVD	- FHx of VTE
- VTE Hy	(- BP (140-159/90-94)
- Thrombogenic mutations		- BMI 30-35
- Familial hypercholesterolemia		- Focal migraine + aura >5y ago
- IDDM (Insulin dependent DM)		- Malabsorption
- BP (>160/95)		- Drug interactions
- Smokers (>35 y, >15cig/day)		- Gallbladder disease
- BMI ≥ 40		
- Focal r	nigraine with aura	
- Stroke		

 Major surgery with prolonged immobilization Liver disease Porphyria Medical condition affected by sex steroids (chorea) undiagnosed GI bleeding Estrogen dependent T (breast) 		 MENOMIC OF SOME OF CI: ABCD A >> ACUTE LIVER DISEASE. B >> BREAST cancer C >> COAGULATION PROBLEMS D >> DVT AND ABNORMAL BLEEDING
Missed Pills	 ed GI bleeding ependent T (breast) EC: emergency contraception UPSI: unprotected sexual intercourse PFI: pill free interval When missing a pill you need to check the efficacy of contraception & if she needs Emergency contraception. o 1 pill missed: only take the missed pill when remembered and proceed, and no EC is needed o 2 or more: take the most recent pill ASAP, and the remaining pills as usual, also use condoms or abstinence for 7 days, for the EC: If missed Pills 1-7 (Week 1): EC is required if UPSI in the PFI or 1st week because 1st 7 to inhibit ovulation. If missed pills 15-21 (Week 3): she could have minimal spotting because of not taking the pills. Omitting the PFI because in free interval (7 day after 21 days) cycle effect of contraception is determined by correct taking pills before and after PFI thus interruption in 3rd week should be followed by a new pack 	
Follow up	3 months after 1 st prescription and check BP, RF, assess	

	- Cycle > finish > bleeding > cycle (intermenstrual bleed).		
	IT bleeding occur while on pills it's called breakthrough		
	bleeding. DDx of breakthrough bleeding:		
	1. If patient is new to pills reassure, because bleeding is		
- •	expected in <u>1st 3 months</u> , however, examine the patient		
Break	2. Did she miss a pill? Default (<u>2-3 d after missed pill</u>)		
through	3. Infections such as STI and chlamydia		
Bleeding	4. Cervical causes		
(BTB)	5. R/O pregnancy		
	6. Ask about medications (liver enzyme inducers)		
	7. Diarrhea and vomiting (D&V)		
	8. Disturbance of absorption		
	9. Low dose pills (estrogen $20\mu g$): change to higher dose		
	""IN COCPS"" But In HRI if she has BIB we 1		
	progesterone dose, not the estrogen because estrogen		
	Is narmful if 1' in HRT.		
	Progesterone only contraception (POC)		
Tupos	- Progesterone only pill (POP)		
Types	Implants		
	- Implants		
	Progestoropo only Pills (POP)		
	- POPs tab contain 28 nill (for 28 days) and all of them		
	are progesterone and active not $21+7$		
Notes	- Can be given to smokers		
Notes	- Usually well tolerated		
	- For them to be effective must be taken the same exact		
	time (by hour and minutes), because they work for 22 hr		
	- 3 hour window period: if a patient forgets to take a pill		
	she has till 3 hours to remember taking it. if she didn't		
	she continues with backup method (condoms for 24		
	hours) > alarming, (Cirazette has a 12 hour period)		
MOA	Same as CHC		
	•		

FR	0.3-0.8/100WY, age > 40 (\downarrow FR), weight (no evidence)	
	- Change in bleeding pattern	
	(2/10 amenorrhoeic, 4/10 regular, and 4/10 irregular)	
	- Mood changes	
SE	- Ovarian cysts	
	- Some claim that it may affect tubal motility so might be	
	RF for ectopic pregnancy.	
	- Increase weight by 2 kg.	
	 Age is not a CI, neither Is migraine (+/- Aura) 	
Advantage	- <u>No effect on breast feeding</u>	
	 Not associated with VTE, MI, stroke, breast cancer 	
	Long-Acting Reversible Contraception (LARC)	
	- Non-hormonal: IUCD or Cu-IUD (Intra-uterine cupper)	
	- Hormonal: LNG-IUS (Mirena), POIC, POI)	
_		
Types	- IUS: MIRENA	
 POIC: progesterone only injectable POI: progesterone only implants 		
	- If LNG-IUS (MIRENA) it can stay for 5 years.	
IUCD &	- Standard T shaped IUCD copper 10 years	
LNG-IUS	- Other Cu-IUDs and LNG-IUS 5 years	
	- FR: 0.2-2 HWY	
	- INNIBIT TERTIFIZATION BY <u>airect toxicity</u>	
	- <u>Anti-Implantation</u> Inflammatory reaction endometrium	
MOA	- Copper in CX mucus inhibits sperm penetration	
	- LING-IUS mainly on endometrium and Cx mucus	
	O STI FISK assessment (sexual HX):	
Insertion &	- Sexual fix, multiples partners is a risk for STD so the pt	
Removal	must be screened for Uniamydia, managed and then sne	

o Screening

o Prophylactic Abx:

it's a request from cardiologist if the pt has mechanical heart valves. Or for Chlamydia if it risk.

o Timing:

*Immediately after bleeding (menses), eg: if a lady has a period of 4 days, she puts it the fifth day, because cervix will be slightly dilated and to rule out pregnancy. (Not inserted in first day of menses because of uterine cramps increase risk of expulsion)

* If a patient is **breastfeeding**, **Amenorrhoeic**, and you are sure she's not pregnant and no intercourse she can insert it at anytime

**Miscarriage*: after 1 week

* Delivered: 4-6 weeks post-delivery

*If she want to be **pregnant**, she can remove it anytime. * if she wants to remove it because she's **not comfortable** with it, but wants to continue with her contraception, we don't remove it if there was intercourse over the last 3 days. (important).because sperm will survive and she can become pregnant.

*if she *wants to switch* to another IUCD , we can remove it regardless if there was intercourse or not.
(but those should be copper not MIRENA)
*In MIRENA tell the pt no intercourse before removal if she doesn't want to become pregnant.

o Follow up:

after 4-6 weeks of insertion to check it is still in. do U.S or by speculum can see thread, X-ray for perforation

- Expulsion
- PID:

only in the *first 3 weeks of IUCD insertion*, after that IUCD wearers and non-wearers have the same chance of getting PID.

• Perforation:

mostly at the time of insertion, if it occurs pull it out, observe and cover with antibiotics. Do laparoscopy if <u>after 3 days</u> patient has abdominal pain or if she's unstable and bleeding.

• Ectopic pregnancy: the overall risk of ectopic pregnancy is not increased with IUCD, however, if pregnancy occurs while having IUCD in uterus then rule out ectopic.

• Bleeding pattern and pain:

Risks

menorrhagia and dysmenorrhea *For those who have menorrhagia it is better to use for them mirena*

• Vasovagal syncope:

only at time of insertion, and the patient can take ibuprofen before 30 minutes of insertion as a pain killer.

- Pregnancy (failure of procedure)
- Lost threads:

If we can't see the thread by speculum, we do U/S, & we see a <u>white line of copper IUCD</u>, in MIRENA its not a line, it appears as two dots and needs experience

o DDx of missed thread:

- Expulsion
- Perforation
- <u>Short thread</u>
- <u>Pregnancy</u>: uterus enlarges, goes up and becomes abdominal organ so can't see the device.

	** If the thread is not seen with U/S, we do plain abdomen, because copper is radio-opaque. MIRENA is not seen on x-ray, so we do CT scan, if still not seen think of perforation, if pt is coming 6 weeks after insertion & clinically stable, we do elective laparoscopy and remove the IUCD, because it's a foreign body and might cause adhesions. But if perforation is at time of procedure and pt is unstable we do ER laparoscopy.
	 o Pregnancy with IUCD: - R/O ectopic - Remove <<u>12 w</u> if visible thread - Increased risk of 2nd trimester <i>miscarriage</i>, Pre-term Delivery (<i>PTD</i>), <i>infection</i> if left inside - Small risk of miscarriage with removal
	** If it's intra uterine pregnancy, remove the IUCD in the first 12 weeks of pregnancy, but tell the pt we have to remove the IUCD, bec if we keep it in, there's a chance of infection and sepsis. If we remove it, there's a chance of miscarriage, if you can't see IUCD leave the IUCD in, removed with placenta at delivery
Info	 Q: all of the following are hormonal methods of LARC, you should differentiate btw hormonal & non hormonal Mirena inhibits ovulation. 20µg /day of progesterone is released from mirena Direct toxicity of copper IUCD inhibits fertilization MIRENA: causes atrophic endometrium and changes in the cervical mucus. MIRENA releases levonorgestrel.
Hormonal SE	 Weight Change Bleeding pattern/blackish discharge Headache

	- Acne		
	- Mood changes		
	- Breast tenderness		
	- Change in libido		
	- Ovarian cyst-functional: if using progesterone		
	preparation. if functional cyst found with combined pills		
	the cyst will be suspicious because combined pills		
	prevent ovulation		
	- LNG-IUS \downarrow blood loss and pain		
NCB	- Endometrial protection		
	- Mx of endometriosis		
Pro	gesterone only Injectable Contraception (POIC)		
	 <u>Depot medroxy progesterone acetate</u> (DMPA): 12 		
	weekly: given every 3 months (12 weeks). If she forgets		
Names	a week or 10 days (comes at week 13 instead of 12) its		
	ok because it's duration of action is 13 weeks and 5 days		
	- <u>Norethisterone enanthate</u> (NET-EN) 8 weekly		
MOA	Same as POP and CHC		
	- DMPA FR : <4/1000 over 2 years		
FR	- <u>Delay in return of fertility up to 18 m!</u> (the others		
	fertility return once removed/stopped)		
	o Bleeding problems:		
	- amenorrhea (up to 6 m), spotting, infrequent or		
	prolonged amenorrhea 1/3 at 3 months & 70% by 1 year		
SE	- R/O STIs, pregnancy		
	- Estrogen is given if irregular bleeding occurs (bleeding		
	is because of low estrogen)		
	o Weight gain:		
	$-2-6$ kg, more in women with BMI ≥ 30		
DMPA	- CVD: safe when estrogen is contraindicated		
Concerns	- Bone mineral density: small loss but recovers when		
	D/C (discontinued) <18 yrs (DMPA is not recommended		

	in those who are <18 years old and perimenopausal)		
NCB	Improve dysmenorrhea and endometriosis		
	Progestogen Only Impant (POI)		
	Implanon: Estonogestrel (ENG)/3 yrs		
Name	- Placed in left arm, shouldn't keep touching it because it		
	release progesterone continuously		
MOA	Same as POIC, POP, CHC		
	 Bleeding problems, weight gain (not significant) 		
SE	 removal complications (occur only if insertion is wrong) 		
	 Side effects of all progesterone's is the same. 		
Concerns	 Enzyme inducing drugs reduce efficacy 		
	 No evidence it decreases bone mineral density 		
	Emergency Contraception		
	• Progestogen only EC: Levonorgestrel levonelle:		
	- ASAP after UPSI – 72 nr ($\underline{73-120 \text{ nr limited efficacy}}$)		
- can be used more than once in a cycle (advantage)			
	- Double the dose if taking liver enzyme-inducing drugs		
Hormonal	- MOA is the same		
method	MOA is the same.		
method	o Illinristal acetate-ellaOne:		
	- selective progesterone receptor modulator		
	- is one of the up to date treatments for <i>endometriosis</i>		
	- 72-120 hrs of UPSI		
	- effective as levonelle		
	- repeat dose is not advised in the same cycle		
	- Cu-IUCD inserted up to 5 days of 1 st episode of UPSI		
	and up to 5 days of the estimated day of ovulation		
IUCD	- Effective immediately		
	- Long-term		
	- FR: 1% (the least!)		

	 Screening for STIs, HIV, Chlamydia/Prophylactic Abx C/I same as IUCD the problem is progesterone is not easily found so pt use combined pills with high dose estrogen increasing risk of thrombosis SE of hormonal IUCD: vomiting: if it occurs within 2 hr must repeat the dose Ectopic Pregnancy: If failure of contraception, you must R/O ectopic
	- Timing of next menses
	- No evidence of teratogenicity
Types	- Female tubal occlusion (FR 1/200) - Male Sterilization-Vasectomy (FR 1/2000)
Notes	 if pt wants to reverse with reopening of the tube, it's an abnormal tube so chances of ectopic increases. Mirena is as good as sterilization In those with multi pregnancy sterilization reduces obstetric complications Tubal occlusion can be done by laparoscopy or laparotomy. laparoscopy: cut or cautery laparotomy:, band or clip with General anesthesia Can the pt get pregnant after tubal sterilization? Yes, most failures occurs early, if the pt is pregnant, so before doing a sterilization make sure she's not pregnant, by preventing UPSI. Male sterilization is better, because of less FR, & reversibility can be done & this is a procedure under LA. Early failure of male sterilization is due to: wrong structure cut, or due to congenital abnormality. Male sterilization: must confirm azoospermia before stopping the contraceptive method they're using (this

usually needs <u>12 weeks</u>), We confirm it by a sample 6 w after the procedure, and repeat the azoospermia sample			
	again another 6 w after.		
	- Cause of failure in tu clipping the round liga	bal ligation in female is accidently ament rather than the tube itself	
	Contracepti	ion after 40	
- No co	ntraceptive method is CI by a	age alone	
- Comb	bined is used up to menopaus	se provided no Cl	
001118	UMKEC	Definition	
	1	No restriction	
	2	Advantages outweigh	
	3	Risks outweigh	
4 Unacceptable		Unacceptable	
Notes	 es - Smoking if >35 years old it is UKMEC 4, if <35 years old - Smokes more than 15 cigarettes/day it is also UKMEC 4 - BMI: 35 is UKMEC 4, If controlled HTN then its UKMEC 2 		
	PC)P	
3 (All PO methods)		4	
 Pregnancy Past breast cancer, clear for 5 yr Active liver disease Abnormal HCG due to GTD 		 Breast disease (current cancer) Undiagnosed vaginal bleeding 	
	IUO	CD	
4	 Pregnancy Unexplained vaginal blee GTD Cervical Cancer - Endore Current PID (chlamydia, 	eding netrial Cancer - Ovarian Cancer gonorrhea.)	

	LNG-IUS		
	- same as IUCD +		
4	- Puerperal Sepsis		
	- Post-septic Abortion		
	- Breast Cancer (Current)		
POIC			
	- CVD - HTN – vascular disease		
3	- Current and Hx of IHD and Stroke		
(DMPA)	- Unexplained vaginal bleeding		
	- Past Breast Cancer - Diabetes – end organ damage		
	- Cirrhosis - Liver Tumors - SLE		
4	- Current Breast Cancer		
	POI		
4	- Current Breast Cancer		

Obstetrics

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A MAR MORE P

General Concepts

Gravida	 Number of pregnancies despite the outcome and despite 	
	the gestational age (any pregnancy what so ever)	
	 The number of pregnancies >20 weeks (duration varies 	
	from region to region, 20 - 28 weeks, depending upon age of	
Para (Parity)	viability).	
	 P (Pregnancy >24 weeks + Pregnancy <24 weeks) 	
Expected date	• EDD = LMP – 3 months + 7 days	
of delivery		
Lie	• Relationship of long. axis of fetus to long axis of uterus e.g	
	longitudinal, transverse, oblique	
Presentation	Presenting part of fetus occupying the lower pole	
	of uterus i.e cephalic (vertex), breech, face, brow or shoulder	
Position	 Relation of denominator (occiput/ sacrum) of presenting 	
	part to the quadrants of pelvis e.g LOA, LOP	
Engagement	 Widest diameter of head below the pelvic brim 	
Station	 Position of presenting part in cm in relation to ischial spine 	
Descent	 Passage of the presenting part of the fetus through the 	
	birth canal, this occurs as a result of the active labor forces.	

Physiologic Changes in Pregnancy

	↓ tone
	\downarrow Systemic vascular resistance SVR
	\downarrow Peripheral vascular resistance
	\downarrow Systolic pressure, $\downarrow \downarrow$ Diastolic pressure
	↓ MAP
CVS	个 CO, SV, HR (20% of the CO for the placenta!! PPH!)
	\uparrow Ventricular distension (LVH, pericardial effusion)
	个 Dysrhythmias, physiologic hypokalemia
	 Murmurs 96%, mainly systolic
	 ECG – ST Changes: non-specific
	 Venous pressure doesn't change
	$ m \uparrow$ Blood volume (dilutional $ m \uparrow$ in blood volume)
Blood	个 RBC (relative, physiologic anemia), 个 WBC
	个 Coagulation factors, fibrinogen (risk: thromboembolic)
	\downarrow Platelets, Factor XI, XIII (mild thrombocytopenia)
	 Pelvic organ till 12 weeks (not-palpable – not affected),
	 if a pt said her abdomen is full b4 12, this is gases because
	progesterone causes relaxation of the smooth muscle
	 week 20-22: umbilicus, afterwards 1 w = 1 cm
Uterus	 Supine hypotensive syndrome: because the uterus
	compress veins and IVC, in the supine position, and \downarrow VR,
	CO, so \downarrow BP & Hypotension Sx, it is relived by laying on left,
	also the compression might lead to varicose, hemorrhoids
	• Poseiro effect : the uterus compress on aorta/branches, \downarrow
	pressure in the femoral artery compared to brachial, also
	contractions may \uparrow the compression causing fetal distress
	(in supine position & the femoral pulse isn't palpable)

	o Auscultation:	
	• S3 Gallop	
	 Systolic ejection murmur 	
	o CXR:	
S/Sx of	 Change in heart position and size 	
pregn-	 increase vascular markings 	
ancy	o EKG:	
mimic	 non-specific ST-T wave changes 	
Heart	Axis deviation	
disease	• LVH	
	o Signs:	
	Peripheral edema	
	• JVD	
	o <mark>Sx:</mark>	
	 Reduced exercise tolerance 	
	• Dyspnea	
	个 Renal blood flow	
	\uparrow Water retention (Renin-stimulated by progesterone)	
	个 GFR	
Kidney	↑ Ureteral dilation/Hydroureter (progesterone SM relax)	
	个 Kidney Size	
	↓ Albumin (Oncotic colloid pressure)	
	\downarrow Afferent, efferent arteriolar resistance (due to vaso-	
	relaxation induced by: relaxin, endothelin, NO)	
	 Constant rate, IRV 	
	 Compensated respiratory alkalosis (due hyperventilation) 	
	个 minute ventilation (TV x RR)	
Lungs	个 tidal volume	
	\downarrow FRC (due to enlarged uterus, so less negative pressure)	
	\downarrow ERV, RV	
	 Constant VC, because it doesn't affect the diaphragm or 	
	thoracic muscle motion	

	\downarrow GI motility (constipation)
GI	 Relaxation of LES: GERD
	 N/V: proportional to HCG
	 Liver/gallbladder: stasis, more stones
	 Pancreas: Insulin resistance
Endo-	• Thyroid: \uparrow TIBG, T4, T3, unchanged-free, \downarrow TSH (by HCG)
crine	• Adrenal: 个 cortisol, by CRH (个 ACTH)
	\downarrow plasma Ca+2 conc. (due to \downarrow in albumin conc.)
	 must accept the allograft
Immu-	 IgG crosses placenta (protect baby by mother immunity)
nology	个 hormonal/innate immunity
	个 susceptible to CMV, HSV, Varicella, Malaria
	\downarrow autoimmune disorders Sx
	\downarrow NK cells
	 Relaxin hormone, secreted by placenta: causes relaxation
	in cartilaginous joints, for more flexibility, to make the birth
Other	process easier (widening: symphysis pubis, CVA)
	 Altered gait, center of gravity
	 Fatigue, somnolence
	 Spider angiomata, palmar erythema
Integu-	 Hair grow (pregnancy hormones as steroids enhance hair)
menta-	 Mucosal hyperemia
ry	 Striae gravidarum
	 Hyperpigmentation (especially linea nigra)
	 Rashes, acne (common)

Cardiac Diseases in Pregnancy

	Ca	rdiac Diseases	
	 Most common Diseases decreating 	n cause of maternal deat eased at first then re-include beart diseases	h during pregnancy reased (still increasing)
aue to <u>Acquired</u> field to diseases			couired congenital
	- Congenital: Pu	<i>Imonary HTN</i> (the mcc o	f death)
	- Acquired: <u>MI</u> (mc), SADS, Aortic dissect	ion, Cardiomyopathy
	Postpartum de	eath is more common th	an antenatally
	 Time of great 	est risks (when the CO is	high or changing
	rapidly): early pregnancy, 2 nd stage & immediately postpartum		
	 O2 consumption increase (demand) 		
	CVS changes:		rasistanaa
	$- \sqrt{2}$ SVR, alterit	ad, pulmonary vascular	$\frac{1}{2}$
	of the total incre	ease occur by 8 weeks of	² gestation)
Physiologic	- BP full in the 2	nd trimester, rising slight	v in late pregnancy
Changes	- COP & SV peak	by week 16	, 10,
during	Physiologic EC	G changes:	
pregnancy	- Atrial, ventricular ectopic		
	- left QRS axis shift		
	- small Q wave a	and inverted T wave in le	ad III
	- ST segment de	pression and T wave inve	ersion in the interior
	* FCG in pregna	» ncy is useful in detecting	arrythmias rather than
	structural abnormalities		
	Low <1%	Intermediate 5-15%	High 25-50%
	- ASD	- Mitral stenosis +	- Pulmonary HTN
Maternal	- VSD	A.Fib	- Eisemenger's synd.
Mortality Risk	- Minimal	- Uncorrected TOF	- Peripartum
	mitral stenosis	- Marfan with normal	Cardiomyopathy
	- Corrected	aortic root diameter	(PPCMP)
Antonatal My	• Multidiscipling	- ALUILLIAI VAIVE	
of Cardiac	Fnsure rest st	on smoking nrevent and	emia
disease	• Mx the respiratory infections		
		ı	

	 Aim: vaginal delivery term 			
Labor Mx of	 Give antibiotics, analgesia 			
Cardiac	 Avoid aortocaval compression 			
disease	 Shorten 2nd stage by using forceps or vacuum 			
	Ergometrine is best avoided			
Rł	neumatic Disease (RD) & Mitral Stenosis (MS)			
	 Incidence decreased (available Rx) 			
Info	 Rheumatic endocarditis cause 75% of MS 			
	 MS: Diastolic murmur at apex 			
	 risk of pulmonary edema 			
	• β-blockers: ↓ HR			
	• \downarrow physical activity: short active 2 nd stage or elective forceps			
	Avoid anemia, good analgesia			
Mx of MS	 treat and anticoagulated arrythmias (specially A.fib) 			
	• Avoid Syntometrine (might lead to pulmonary edema)			
	• Avoid fluid overload: diuretics may have a role, keep her on			
	the dry side (80 ml/hr)			
	Aortic Stenosis (AS)			
	Late Sx, sudden death may occur			
Info	• PE: Loud (>3/6) harsh systolic murmur			
	• if severe might lead to acute left ventricular failure, and they			
	don't tolerate hypotension or tachycardia			
AS Mx	Avoid hypotension, fluid depletion			
	 Apply lateral tilt if pregnant (or semi-sitting position) 			
	Valve Problems (Regurgitant Valves)			
	 Regurgitated valves are well tolerated in pregnancy 			
Info	 During pregnancy, thrombotic risk increase (29%, 2.9% of 			
	maternal morality – need of effective anticoagulation (warfarin,			
	unfractionated heparin (UFH) or LMWH)			
Valve	 Prosthetic artificial valves: lifelong anticoagulation/warfarin 			
Replacements	 Prosthetic tissue valves: deteriorate with time 			
Eisenmenger Syndrome				
	Long-standing left to right shunt caused by congenital defect			
Info	 Maternal morality: 20-40% 			
	 Fetal outcome (Poor): Cyanosis, Low O2 sat, Polycythemia 			
Mx	Most maternal death occurs in puerperium			
	 Risk of death remain high (7%) with pregnancy termination 			

	Ischemic Heart Disease (IHD)		
Info	 Incidence is increase It occurs suddenly, unexpectantly 20% with acute MI die in pregnancy or within 1 w of delivery MI in 2nd trimester has lower mortality rate than the 3rd 		
IHD RF	 Multigravida, smoking, DM, HTN, obesity, hyper-cholesterol 		
	Cardiomyopathy (CMP)		
Peripartum CMP (PPCAMP)	 Presents near term or 1st few weeks postpartum (up to 5 m) Common in older, black, multiparous, obese or HTN pt Perform Echo in: unexplained SOB, tachycardia, edema or SVT we check after delivery (up to 5 m) if it was unresolved then we tell the pt that pregnancy is contraindicated (due to high recurrence in future pregnancies) 		
	Heart Failure (HF)		
Info	 Maternal health takes priority ACEI is contraindicated (teratogenic, cause fetal renal failure) ACEI can be used during breast feeding <i>6-blockers</i>: ↓ HR <i>Furosemide</i> can be used <i>Digoxin</i>: can be used (it crosses the placenta but at therapeutic levels it doesn't harm the fetus) Consider prophylactic <i>anticoagulation</i> 		
	Arrythmias		
Info	Ectopic beats, palpitations are common		
Investigations	 12 lead ECG (ideally when an episode occurs) TFT, Hemoglobin, 24 hr ECG Echocardiography and cardiology review 		
	General Points for Cardiac Diseases		
 Risk of fetal congenital heart disease (CHD) for a mother with a CHD is 4% (50% it is the same case) Fetal echo is ordered if 1 of the parents have CHD Prophylaxis against bacterial endocarditis: NICE guidelines (do not give), while the AHA (give prophylaxis – the Dr supports giving) 			

Venous Thromboembolism (VTE)

	Venous Thromboembolism (VTE)
Causes of pro- thrombotic state in pregnancy	 Stasis: a) Compression of iliac veins (gravid uterus, right iliac artery over left iliac vein – hence most DVT's are left in pregnancy) b) Hormonally mediated vein dilation c) Immobilization Vascular Damage: a) Vascular compression at delivery b) Assisted or operative delivery Hypercoagulable blood: (= ↑ Thrombin ↓ clot dissolution a) ↑ Procoagulant factors: ↑ Fibrinogen, ↑ V, IX, C and VIII b) ↓ Anticoagulant activity: ↑ Protein C resistance, ↓ Protein S
	c) \downarrow Fibrinolytic activity: \uparrow PAI1 & 2 activity, \downarrow tPA activity
Prophylaxis	 Risk assessment ≥ 4 current RF (beside VTE Hx or thrombophilia): give prophylactic LMWH during antenatally & for 6 w postnatally ≥ 3 current RF: give LMWH from 28 w & 6 w postnatally ≥ 2 current RF: give LMWH for atleast 10 days postnatally Women with previous VTE only (no other RF): RCOG: offer anti-coagulation for 6 weeks postpartum, British Society: prophylaxis the time pregnancy is confirmed
	Thrombophilia's
Definition	 Predisposition to thrombosis, 2ry to hypercoagulable state it might be inherited or acquired
	Antiphospholipid Syndrome
Thrombosis	• ≥ 1 clinical episodes or arterial, venous thrombosis
Pregnancy Morbidity	 1) ≥ 1 unexplained death of morphologically normal fetus ≥10 w 2) ≥ 1 Preterm birth (PTB) of a normal neonate <34 w due to: (i) eclampsia or severe PET or (ii) placental insufficiency 3) ≥ 3 consecutive miscarriages <10 w
Laboratory Criteria	 Lupus Anticoagulant (<i>LAC</i>): ≥ 2 occasions at least 12 w apart Anticardiolipin Antibodies (<i>aCL</i> – IgG &/or IgM): present in medium or high titre on ≥ 2 occasions at least 12 w apart Anti Anti-82-glycoprotein I antibody (IgG and/or IgM),

present on \geq 2 occasions at least 12 w apart

Pregnancy	 Pregnancy loss 	, <u>PET</u> , <u>Fetal growth</u> restri	<u>ction</u> , <u>PTD</u> (iatrogenic
Risks	or spontaneous)	, <u>Thrombosis</u> (DVT, PE, Ce	erebral infarct)
	V	TE Prophylaxis	
	 All VTE risk ass 	essment should be indivi	dualized (not all
Info	thrombophilia's	carry the same risk for ex	(ample)
	 Hereditary thro 	ombophilia screens are n	ot done in pregnancy.
	- Protein C, Prote	ein S, Antithrombin III are	altered
Inherited 1	Thrombophilia	Antenatal Prophylaxis	Postnatal Prophylaxis
Anti-thron	nbin deficiency	Yes - LMWH	Yes - LMWH
Protein C deficiency		Yes - LMWH	Yes - LMWH
Homozygous factor V Leiden		Yes - LMWH	Yes - LMWH
Homozygous prothrombin gene		Yes - LMWH	Yes - LMWH
Protein S deficiency		No – ? Aspirin	? – LMWH
Heterozygous factor V leiden		No – ? Aspirin	? – LMWH
Heterozygous prothrombin gene		No – ? Aspirin	? – LMWH
Other-thrombophilic defects:		Yes - LMWH	Yes - LMWH
e.g. Anti-phos	pholipid syndrome		
	1) <i>LMWH</i> :		
 <u>Agent of choice</u> for AN/PN, weight safe in breastfeed 			in breastfeed
	• <u>1st line anticoa</u>	<u>gulant</u>	
	Ricks		

- RISKS:
- Heparin induced thrombocytopenia (*HIT*): v. rare in pregnancy
- Osteopenia: rare
- Allergic reaction: pruritis, ulceration: try histamine, steroid

2) **UFH**

Which Agents

To Use

3) Aspirin: not recommended (unless heparin is not used)

4) Warfarin:

• Restricted in pregnancy to few situations where heparin is unsuitable (e.g. mechanical heart valves)

• Contraindicated in the 1st trimester (*Teratogenicity 5% risk*)

• *In 2nd*, 3rd trimester: cause retroplacental and intracerebral fetal bleeding leading to poor growth & CNS abnormalities

 Long term anticoagulation can be converted from LMWH to warfarin postpartum when the risk of hemorrhage is reduced, mostly 5-7 d after delivery (reserved for the postpartum period) - Warfarin is safe in breast feeding

	DVT
	Maybe asymptomatic
	 Sx: swelling, pain, hotness, erythema (redness)
Info	 80% DVT's in pregnancy are left sided
	 70% DVT's in pregnancy are iliofemoral (above knee) as
	compared to a non-pregnant rate of 9%
	 When you suspect DVT start treatment immediately!
	 Compression duplex US: reliable and sensitive for proximal
	DVT but not for calf DVT
	 If US is negative & low suspicion: discontinue the treatment
Dx	 If US is negative and high suspicion: stop the anticoagulant
	and repeat the US on days 3 and 7
	 D-dimer: not used because it increase in pregnancy
	 CBC, Coagulation screen, Urea & Electrolytes, LFT
	Thrombophilia screen: not recommended
	 Elevate leg & elastic compression stocking: to reduce edema,
	used up to 2 years post thrombotic syndrome
Mx	Anticoagulation
	 Mx with LMWH during the remainder of pregnancy and 6
	weeks postnatally and until atleast 3 months of treatment has
	been given in total
	Pulmonary Embolism (PE)
	 Leading cause of maternal death
Info	 Difficult to clinically diagnose it: 2-20% of pregnant patient
	with clinically suspected PE prove to have PE
	 Dyspnea, Chest pain, Cough, Hemoptysis, Pyrexia,
C/P	Tachycardia, Tachypnea, Cyanosis, Raised JVP, Pleural rub,
	Pleural effusion, EVF
	 ABG: hypoxemia, hypocapnia (respiratory alkalosis)
	• <i>ECG</i> :
	- Inverted T wave and atrial arrythmias
	- Right axis deviation, T wave inversion (lead III), Q wave (lead
Investigations	III), are normal findings in pregnancy
	• CXR: doesn't harm the fetus (minimal radiation amount)
	Ventilation perfusion scintigraphy:
	- used when PE suspicion, leg doppler are (-)
	- low radiation (increase risk for childhood malignancy)

	- negative predictive value of a normal VQ scan in pregnancy is
	excellent, making this a useful first line investigation
	- if low probability but there is a high suspicion consider further
	imaging types
	 if medium-high probability then anticoagulation is continued CT Pulmonary Angiography (CTPA):
	- less radiation than VQ but expose maternal breast tissue
	- used in high suspicion only and V/Q is equivocal
	- gold standard for Dx, but CI in pregnancy (invasive)
	• Compression US – leg doppler:
	 - if there is symptoms of DVT – indirect confirmation (can be
	confirmatory), if it's positive no further investigations needed
	- Pelvic DVT is easily missed
	 Venous stasis and reduced venous return can also result in
	false positive in pregnancy (mainly at 20 weeks)
	 if asymptomatic the sensitivity is poor
	 D-Dimer: not useful in pregnancy
	• Call for help, multidisplinary team, high dependency unit
	 High flow oxygen
	• +/- ventilation
	• LMWH (175 IU/kg)
Mx	 +/- Thrombolysis (Streptokinase – controversial)
	• IVC filter in:
	 recurrent VTE despite therapy
	 can not tolerate anticoagulant or contraindicated
	- extensive VTE at ≥36 weeks of pregnancy

Liver & GI in Pregnancy

• Physiologic Changes in Pregnancy:

- \downarrow lower oesophageal sphincter pressure
- \downarrow gastric peristalsis
- delayed gastric emptying
- \uparrow small and large bowel transit times.
- Thus, gastric reflux and constipation are common in pregnancy.

• Liver function tests (LFTs) are altered:

- ALP \uparrow with gestation due to placental production (2–4x by term)
- ALT, AST and GGT are reduced

- albumin \downarrow by 20–40%; this is partially dilutional due to increased total blood volume (contributes to most of the \downarrow in total serum protein)

- fibrinogen, caeruloplasmin, transferrin, & many binding proteins \uparrow
- bilirubin levels do not change.
- S&S of normal Pregnancy: they mimic GI and liver disease,,
- Nausea and vomiting
- Dyspepsia and heartburn, gastric reflux
- Constipation, Abdominal pain
- Spider naevi and palmer erythema (due to high estrogen lvls)

Hyperemesis Gravidarum (HG)
 Intractable vomiting
 >5% weight loss
 Dehydration
• Ketosis
Electrolyte imbalance
Always 1 st trimester!

Causes	Unknown, there are contributing fact	ors
	Maternal	Fetal
	 Hyponatremia 	 Small for GA
	 Hypokalemia 	• IUGR
Complica-	 vit B1 (Thiamine - Wernicke's 	• PTB
tions	encephalopathy Korsakoff psychosis)	
	• vit B12, B6	
(Sx)	 Metabolic hyperchloremic Alkalosis 	j
	 Mallory-Weis 	
	 Central pontine myelinolysis (rare) 	
	 Abnormal liver function 	
	 Biochemical thyrotoxicosis 	
	 Admission: rehydration, R/O molar, 	UTI
	 Anti-emetics 	
Mx	Vitamins	
	 Steroids (Corticosteroids) 	
	 Thrombo-prophylaxis (risk: dehydra 	ation + pregnancy)
	Constipation	
Frequency	40%	
Мх	Reassurance, hydration, fibers, laxati	ves
	Gastric Reflux	
	 Antacids 	
Мх	 Avoid food/fluid 	
	• H ₂ receptor blockers (Cimetidine, R	anitidine)
	Omeprazole	
	Pancreatitis	
Sx	 Epigastric pain 	
	• N/V	
	• Alcohol	
RF	Hypertriglyceridemia (triglycerides	↑ 3x in pregnancy)
	 1^{ry} Hyperparathyroidism 	
Investigate	 Serum amylase > 1000 	

	• IV fluid
	Analgesia
	• NBM
Мx	Intubation
	• ERCP
	 Stent drainage
	Gallbladder/stone removal
	Peptic Ulcer Disease (PUD)
Info	Rare, Pre-existing disease tend to improve in pregnancy
	(PG and E2 induced by pregnancy protect the mucosa)
Sx	Epigastric pain
	Complications: Hemorrhage, perforation are rare
	H2 Antagonists, PPI, Antacids
Mx	Avoid misoprostol
	• H.pylori eradication after delivery
	• Upper GI endoscopy (if significant Sx, hematemesis)
	Obstetric Cholestasis
Info	 Unique to pregnancy, 3rd trimester, recurrence 90%
Sx	• Pruritis
	 Excoriations (scratching) without rash
Investigate	• LFT abnormal • Dx of exclusion
	 Malabsorption
	• PPH
Complica-	• PTD
tions	• IUFD
	Fetal distress
	 Fetal ICH (intracranial H, stroke)
	Delivery ASAP
	• vit. K for both
Мх	Anti-histamine
	 UDCA (ursodeoxycholic acid)

1	n	6
-	v	v

	Acute Fatty Liver of Pregnancy (AFLP)
Note	 Rare, fatal, 3rd trimester
	 Risk of fulminant hepatic failure, encephalopahy
Sx	 Severe vomiting
	Abdominal pain
DDx	 HELLP Syndrome (hemolysis, elevated liver enzymes,
	low platelets)
Investigate	• Liver dysfunction: hypoglycemia, hyperuricemia, renal
	impairment, coagulopathy

Neurological

Epilepsy in Pregnancy			
Info	- MC neuro	ological problem in pregn	ancy - Subfertility
	- <u>Pregnanc</u>	<u>y effects on epilepsy</u> : 30%	~个, 10% ↓, 60% NC,
	risk of seiz	ures is highest peripartun	n
Effects	- <u>Epilepsy e</u>	e <u>ffects on pregnancy</u> : feta	Il loss (miscarriage),
	fetal grow	th restriction, fetal malfor	rmations (4-10% -
	general 2-3	3% - we give <mark>sodium valp</mark> ı	roate)
Precon	ception	During	Postparum
- Drug withd	rawal/ dose	- Booking US scan –	- Encourage
change if inc	licated	anencephaly can be	breastfeeding
Monotherap	у	detected at 11 weeks	
- preconception folic acid		- Review seizure	 Review anticonvulsant
5 mg daily		frequency & AEDs	therapy and
- discussion of potential		- Detailed anomaly & fetal	contraception
maternal and perinatal		cardiac US scans	
risks		- Clinical assessment of	- Advise the woman on
- advice about the most		fetal growth	strategies to minimise
appropriate		- Vit K last 4wks of	harm to her and her
contraception.		pregnancy	baby
		- Baby should receive Vit K	
		1 mg at birth.	

Thyroid Diseases

	Hyperthyrodism
Causes	Graves (90%), toxic nodule, toxic multinodular goiter,
	hydatiform mole, HG
	- PET
Outcomes if	- neonatal <i>hypo</i> thyroidism
uncontrolled	 thyroid storm and thyrotoxic heart failure
for both	 fetal growth restriction
mother and	- prematurity
baby	- stillbirths
	- fetal or neonatal thyrotoxicosis
	- <u>Medications</u> : carbimazole (CBZ), methiamazole
	(MMI) or propylthiouracil (PTU), cross the placenta,
	but PTU less so than CBZ and MMI (because it crosses
	the placenta in lesser amounts).
Мх	 <u>Surgery</u> is rarely performed in pregnancy, possibly if
	drug resistance or serious side effects with antithyroid
	drugs, e.g. agranulocytosis.
	 Radioactive iodine treatment is contraindicated
	during pregnancy and should be avoided for at least 6
	months after treatment.
Hy	ypothyroidism – Overt hypothyroidism (OH)
	OH complicates 2–10/1000 pregnancies & is due to:
	 Hashimoto's thyroiditis
Causes	 previous radioiodine therapy/thyroid surgery
	 previous postpartum thyroiditis
	 hypopituitarism
	iodine deficiency.
Outcomes if	 higher risks of spontaneous miscarriage, PET, PIH, PPH
uncontrolled	& low birth weight.

for both	 There is a risk of a slight reduction in IQ in the fetus
	but no increased risk of congenital malformations.
Mother	 Pregnancy itself probably has no effect on
and baby	hypothyroidism although approximately 25% of
	women will require an increase in their thyroxine dose
	in pregnancy
	well-documented syndrome of growth restriction,
Congenital	deafness and neuropsychological impairment, resulting
Cretinism	from severe iodine deficiency or untreated congenital
	hypothyroidism
Diabetes in Pregnancy

Types	pre-existing (type I,II), or Gestat	ional (pre-existing, TRUE)
	 effect of pregnancy on pre-existing: 	
Pre-	- \uparrow insulin requirement	
existing	- nephro, neuro, retino-pathies	deterioration
	- Hypoglycemia, DKA	
	Maternal	Fetal
		- congenital
Effects	 increase risk of (miscarriage, 	abnormalities, mortality,
	PET, infection, lower segment	late stillbirth,
	caesarian section (LSCS))	hypoglycemia,
		polycythemia, jaundice
	a. <u>Diet</u> : low carbs, high fibers (o	rlistat + metphormin),
	avoid starvation, & frequent sna	acks might be needed
	** note: hypoglycemia after birt	th is caused by Λ in insulin
	 b. <u>Insulin</u>: 3 pre-meal short acting intermediate acting (protophan flexibility 	ng insuln (actrapid) +/- e – 3 m) as it allows max
	 target glucose level: fasting (< 	āmmol/L), 2hr (<7mmol/L)
Мх	c. <u>Oral hypoglycemic agents</u> : - risk of congenital abnormality	
	- for DMII, we stop oral hypogly	cemic & change to insulin
	• <u>Biguanides</u> (<u>Metformin</u>):	toratogonic) catagory P
	- can be used in pregnancy (not	leratugenic, category b & reproductive function
	- $\downarrow 1^{st}$ trimester miscarriage, 10	$x \downarrow GD$
	 <u>Sulfonylureas</u>: category C 	

	- 1 st generation (个 risk of neonatal hypoglycemia),
	 2nd generation (Glyburide, no such effects)
	- 4-20% pt fail to achieve glucose control with max dose,
	- \uparrow risk of pre-eclampsia and need phototherapy
	d. <i>Insulin Analogues</i> :
	 <u>Rapid acting</u> (<u>Lispro</u>):
	- cat B, teratogenic concerns, antibody formation, growth
	promoting properties, majority of evidence showed that
	doesn't cross the placenta and has no maternal/fetal S.E
	 Long acting (Glargine):
	- cat C, not well studies
	 control sugar during delivery, timing and mode,
	intrapartum insulin infusion with glucose monitoring, no
	<u>CI for breast feeding at all</u>
	o Pre-conception Counselling:
Delivery	 to optimize control prior conception
	 If needed, proliferative retinopathy treated with
	photocoagulation before conception
	 <u>the only CI to pregnancy are</u>: ischemic heart disease,
	untreated proliferative retinopathy, severe renal
	impairment (creatinine >250)
	Gestational Diabetes (GD)
	carbohydrate intolerance first recognized during the
Definition	present pregnancy (includes pre-existing unrecognized,
	30% identified as GDM in fact are pre-existing)
	 the test is performed between 24-28 weeks (because
	the diabetogenic effect can be manipulated at this time),
	in 1 st trimester check for blood sugar, at 24-28 weeks (en
Screening	of 2 nd) check glucose intolerance test if there is risk, if hig
Screening	of 2 nd) check glucose intolerance test if there is risk, if hig risk then at week 10
Screening	of 2 nd) check glucose intolerance test if there is risk, if hig risk then at week 10 - Screening by fasting, random glucose, glucose challenge

RF	 Age >25y BMI > 25 previous GDM FHx of DM in 1st degree relative previous macrosomic baby (≥ 4 polyhydramnios large for date baby in current p previous un explained stillbirth 	e 4Kg) pregnancy	
Dx	- Glucose challenge test (75gm/100gm)		
	- fasting glucose 75gm glucose		
	Fetal	Maternal	
	- Macrosomia (>4kg)	- \uparrow risk of hypertension	
	- increase C-section,	- \uparrow risk of c –section,	
	instrumental deliveries, birth	instrumental deliveries	
Complic.	trauma (brachial plexus	- 个 risk (40-60%) of	
	injuries, clavicular fractures)	developing DMII within	
	- 个 in neonatal hypoglycemia	20-15 y (hence woman	
	(24%), hyperbilirubinemia,	should be screened	
	polycythemia	annually)	
	- \uparrow risk of DMII, obesity in life		
Мх	- similar to pre-existing, monitor	r glucose, diet control,	

Pregnancy Category	Description
Α	No risk in controlled human studies
В	No risk in other studies
С	Risk not ruled out
D	+ evidence of risk
X	CI in pregnancy
N	FDA has not classified it

Hypertension in Pregnancy

	- BP \geq 140/90 Hg for 1 st time during pregnancy
Gestational	- no proteinuria
	- BP appear after 20 w GA & return in <12 w Postpar.
	- Final Dx only made postpartum
PET	- same as gestational HTN + proteinuria (≥ 300mg/23
	hr or 1+ dipstick)
	 same features as PET + tonic clonic seizures
Eclampsia	- Seizures that cannot be attributed to other causes in
	a PET
	- increase in BP either before pregnancy or before 20
Chronic HTN	w of gestation or 1 st dx after 20 w of gestation &
	doesn't disappear 12 w postpartum
PET	 chronic HTN + worsening of BP + worsening of
superimposed	proteinuria
on chronic	
	 life-threatening pregnancy complication usually
	considered to be a variant of PET. Both conditions
	usually occur during the later stages of pregnancy, or
HELLP	after childbirth.
	H (hemolysis, which is the breaking down of RBCs)
	EL (elevated liver enzymes)
	LP (low platelet count)
	• FHx
	Obesity
	 1st Pregnancy of a couple
RF	 Age extremities (<20, >40)
	 Medical: antiphospholipid, DM, renal disease,
	 Obstetric: multiple pregnancies, previous PET,
	hydrops fetalis, triploidy, hydatiform mole

	 Asymptomatic (most commonly)
	 flashing lights, photophobia, headache, visual field
Sx	loss, epigastric pain, vomiting
	** Remember: BP decreases in pregnancy then
	increase but stays within normal range
	Mammogram
Screening	 Abnormalities in maternal uterine artery Doppler
	waveform (notching) between 18-24 w gestation may
	identify increased risk

• Potential 2^{ry} effects in pre-eclampsia:

- *Placenta*: infarction, retroplacental bleeding, abruption (Pre-mature separation of the placenta due to increased BP (normally placenta separates from uterus during delivery)

- *Fetus*: impaired uteroplacental circulation might lead to growth retardation, hypoxemia and intrauterine death

	Pre-Eclampsia (PET)
Mild form	- B/P 140-159/90-109, proteinuria 300-5K mg/24 hr - no end organ dmg
	 conservative ttt & follow up (to prevent progression) delivery at term (37w)
Severe form	 signs of end organ damage B/P >160/>110 on 2 occasions at least 6 hrs apart proteinuria ≥ 5g/24 hr Oliguria <500 cc/24 hr cerebral or visual Sx, epigastric or RUQ pain pulmonary edema or cyanosis low PLt IUGR ↑↑↑ liver enzymes

	- Delivery of the fetus even if preterm (to avoid compl.)
Mx	-If fetus <24 w then terminate pregnancy
	->32w GA there is no cause not to deliver the fetus!
	 Term pregnancy with mild or severe PET
Indications	 Severe PET regardless of the GA
for	 Warning signs: headache , visual disturbance,
Termination	epigastric pain, oliguria
	 Eclampsia: the Pt must be stabilized & delivered
	- Preterm mild PET: Assess fetal by NST, BPP, Doppler
Methods of	 IOL with PG followed by IV oxytocin
Termination	- Elective CS: Severe PET with unfavorable Cx
	- Mild: no benefit
	 Severe: used to prevent maternal stroke (complica.)
	- Methyldopa
Anti-HTN	 Nifidipine or labetalol may be added to Methyldopa
therapy	- <u>Avoid: diuretics, atenolol, ACE's, ARBS</u>
	- If BP >170/110 mmHG:
	Labetalol or Hydralazine, or Nifedipine
	- Check BP every 15 m until stable
	• MgSO4:
Prevention	 is given in SEVERE PET to prevent eclampsia
& Control of	- MgSO4 is Ca ⁺² CB
Convulsions	 If MgSO4 isn't available, use Phenytoin or Diazepam
	 - MgSO₄ Toxicity: BURP (blood pressure ↓, urine
	output \downarrow , RR <12, patellar reflex absent)
	 Total fluid should be limited to 80ml/hr or 1 ml/kg/hr
Fluid Mx	 Historically Pulmonary edema was a cause of death
	- Fluid should be maintained (if low: renal failure, if high
	then pulmonary edema)
Fetal	o CTG
Assessment	o Further assessment tests include;
	- US measurement of fetal size

	- Umbilical artery Doppler
	- Assessment of fluid volume
	o Women in labor with severe PET should have
	continuous CTG
Prevention	 Low dose aspirin: before 2nd pregnancy
of PET	- Ca ⁺² Supplements: for prevention but doesn't \downarrow risk
	Chronic HTN
	- Booking tests
To check for	- CXR (Cardiomegaly)
HTN Comp.	- ECG (Left ventricular hypertrophy)
	- \uparrow serum creatinine, \downarrow creatinine clearance &
	proteinuria (5-10%, might be before pregnancy)
	- Superimposed PET in 1/3 of Pt
	- 个 risk of abruption placentae
Maternal	Renal function:
Compli-	 If renal function is well creatinine < 1.5 mg/dl
cations	- pregnancy doesn't change the course of renal disease
	- If function is affected prior to pregnancy,
	deterioration occur more rapidly
Eatal	- Promoturity 25-20%
Complica	
complica.	Stillbirth & fotal distross
	- mild CH HPT (140-179/90-109) only monitor no Mx
	 Pt with severe CH HTN should have their BP controlled
	before pregnancy & continue Rx in pregnancy
	- α Methyle Dopa
Mx	- Calcium channel blockers
	- β blockers can be used but > IUGR
	- Labetalol
	 Serial U/S for fetal growth. BPP, NST > 34wk
	- Follow up every 2 wks till 30 then weekly

• Investigations:

- Renal function test, uric acid , calcium ,LFT, 24hrs urine for creatinine clearance & protein, CBC, Urinalysis, ECG.GTT

- Early U/S for dating of pregnancy
- Not allowed to continue past 40wks, IOL at 40 wks
- Regular diet no salt restriction
- IOL > for superimposed PET,IUGR, fetal distress, worsening renal function

Twins

- the rate of monozygotic twins is constant (3-5/1000)
- Prevalence: 1% JOR
- only 4 per year are monoamniotic, monochorionic in JOR
- Chorion (sac), Amniotic (Placenta), Zygosity (Zygote)
- dizygotic twins have separate placenta but can be fused (DCDA)
- Monochorionic Monoamniotic (MCMA): no separating membrane
- MCDA
- DCDA

o **Monozygotic (identical twins) Twins**: if division of inner cell mass occur after the amniotic cavity occurs (8 days) the twin will be MCMA (1%), 70% of are MCDA, 29% DCDA

Complications	
Maternal	Fetal
Miscarriage	 Prematurity (MC!)
• PTL	• PROM
• PET	 Congenital anomalies
• GD	 Umbilical cord: velamentous,
 Abruption 	prolapse, vasa previa
• Anemia	 Discordance – unequal weights
• UTI	 TTTS (all MC carry risk)
 Anomalies 	 Antepartum death of 1 twin
 Polyhydramnios 	• IUGR
 Malpresentation 	 unequal placenta surface
• CS	 genetic syndrome
• PPH	 Cerebral palsy risk



	• US (every 4 w): discordance, AFV, umbilical
	• Doppler: umbilical artery flow velocity (mainly in MC)
	 Serum screening: risk of Down
	 Nuchal translucency
Antenatal	 CVS or amniocentesis
Care	 Fetal reduction or termination even
	 Nutritional advice
	 more antenatal visits
	• Twins grow should be followed by the singleton growth
	curve until 32-25 w (b4 32w – normal, >32 w, less space)
	• IUGR
Discorda-	• TTTS
nce cause	• Aneuploidy
	• Anomaly
	Viral infection
	 Higher risk of TTTS, Death of one twin (it leads to
MC twins	hypotension, and give the 2 nd baby thromboplastic death
	or nourclogical damage)

• 15% of MC

• Vascular communication (artery-vein, anemia for one, polycythemia for other)

o Grades:

- Bigger bladder
- more amniotic fluid
- gets bigger size

TTTS - bigger baby die from heart failure

Twin

То

- Fetal hydrops is pre-terminal sign
- Recipient twin: cardiomegaly, CHF, polyhydramnios, die
- Donor twin: IUGR, HF if anemia (severe), hydrops, oligo-

• Stuck twin: sonographic appearance of extreme TTTS

Transfuse Syndrome

PTL

Twin

o Treatment:

- Amnioreduction (by amniocentesis)
- Laser ablation of vascular anastomoses
- PTD

o Death of one twin:

- most feared sequalae is neurological damage (for the survivor – thrombotic arterial occlusion)

- Mother: DIC

- biggest RF for mortality, morbidity
- Problems of PTL: corticosteroids help!
- Lungs: RDS
 - GI: NE
 - Neuro: intra-cerebral hemorrhage
 - Infection: sepsis

Post- partum Mx	 IV Syntocinon (prophylactic)
	** Breech of 2 nd twin is not a CI of Vaginal Delivery
	- Other major obstetric RF
	- 1 st twin non Vertex
	- Discordant twins
Мх	- MA twins
partum	o Elective C/S if:
Intra-	
	 Epidural anesthesia might be useful
	• US
	 Continuous electronic fetal monitoring (EFM)
	• CBC, blood group
	 neither has fetal compromise requiring CS
	- Twin 1 is cephalic
	- Diamniotic twins
	 Vaginal birth indications:
	- vertex/transverse: 7%
	- breech/breech: 9%
	- vertex/breech: 30%
	 Frequency of fetal presentation: vertex/vertex: 40%
	 37 w is the best to deliver twins
	presentation (determined by US)
	 most important element in deciding the NIOD is feta

Assessment of Small for Gestational Age Fetus (SGA)

IUGR ≠ SGA ≠ LBW

SGA: newborns who are smaller in size than their normal for the same GA, weight is <10th percentile for appropriate GA IUGR (Intra-uterine growth restriction): presence of a pathological process in-utero that inhibits the fetal growth, birth weight is below 10th percentile for a given gestational age

• LBW (Low birth weight): birth weight below 2500

IUGR	SGA
Fetus growth is restricted or	Size of the fetus is SGA
retarded while in the uterus	
Appear malnourished	Small, not always malnourished
- US	- US
- Doppler flow	 Measure fundus to pubic bone
 Measure fundus to pubic bone 	
Measure is based on the change	Measure is based on a one-time
in growth over time	measurement that falls below a statistical value
Always slower than normal	Might be normal
Might be normal	Always lower than normal
Always pathological	Not always pathological, mostly
	due to Genetics
SGA	
• GA (Gestational Age), Growth	Curves, Centile
• Ovum lives for 48 hours after of	ovulation
- You can know it by two method	ds:
1) Calculation:	_
- We calculate GA depending on	LMP or EDD
- To use LMP: must be regular, k	nown date, no OCP or lactation
- EDD = LMP + 7 days $-$ 3 month	s + 1 vear
- Duration of pregnancy = $266 da$, avs (from conception till birth) +
14 days = 280 days (from LMP) =	= 40 weeks (could be up to 42)
LMP GA	 EDD (280 days in total)
	IUGR Fetus growth is restricted or retarded while in the uterus Appear malnourished - US - US - Doppler flow - Measure fundus to pubic bone Measure is based on the change in growth over time Always slower than normal Might be normal Always pathological SGA SGA • GA (Gestational Age), Growth • Ovum lives for 48 hours after of • You can know it by two method 1) Calculation: • We calculate GA depending on • To use LMP: must be regular, k • EDD = LMP + 7 days – 3 month • Duration of pregnancy = 266 day 14 days = 280 days (from LMP) = LMP GA

<u>How to calculate</u>?

a) Calculate EDD, LMP

b) Check todays date, and which one is closer to it?

- if it's closer to LMP: then GA = todays date LMP
- if it's closer to EDD: then X = todays date EDD / GA = 280 X
- *Example 1*:
- LMP = 1/5/2019
- Today = 3/9/2019
- EDD = 8/2/2020
- so LMP is closer: 2 days difference + 4 months (16 weeks)
- GA = 16 week + 2 days (or count the days)

• <u>Example 2</u>:

- LMP = 22/2/2019
- Today = 3/9/2019
- EDD = 29/11/2020
- so EDD is closer: 26 days difference + 2 months (total 86 days)
- 86 days / 7 = 12 weeks and 3 days
- GA = 40 (12 weeks + 3 days) = 27 weeks and 4 days

2) <u>US</u>:

- the earlier the more accurate, for example:

if you do US in	Error days	
8/9 week	+/- 3 days	you can't
20 week	+/- 10 days	know it
35 week	+/- 21 days	exactly

	• Graphical representation of how a particular quantity (weight,
	height,) increases over time
Growth	 there is a normal curve for every variant (bell shaped curve)
Curves	you must be within this curve, beside that there is a problem
	 Term used to describe the degree of the fetal growth based
	on the normal curve
Centile	 50 Percentile: is the mean where most babies exist in
"Percentile"	 > 95 Percentile: small % of babies that are more than average
	• < 5 Percentile: small % of babies that are less than average
	and if a shill we include the states TO as while lives the transmission

e.g.: if a child weight is at the 50 centile line, that means out
 of 100 children at the same age 50 will be bigger and 50 smaller

	• We measure head circumferent	ce (HC), abdominal
	circumference (AC), femur lengtr	1 (FL)
How to	• Then we put them in a certain e	equation to measure the
measure SGA	estimated fetal weight (EFW), we	e put this weight in the growth
	curve depending on the fetal GA,	compare it to the normal to
	see at what percentile is this fetu	S
	IUGR	
	 40% of unexplained stillbirths 	
IUGR	 30% of sudden infant death syr 	ndrome (SIDS)
Complications	 8 fold increase risk of infant module 	ortality
	 Operative delivery 	
"Important-	Hypoxic Ischemic Encephalopat	hy (HIE), Mental retardation
ance to	 Prematurity 	
differentiate	 Meconium Aspiration 	
IUGR/SGA"	• Asphyxia, Hypoxia, Polycythem	ia, Hypoglycemia, Acidemia
	• Risk of adult onset conditions: I	DM, HTN, Atherosclerosis,
	Strokes, Adult metabolic syndron	ne (Barkers hypothesis)
	• <10 th centile for GA	
Threshold	- high sensitivity, low specificity,	might be SGA
 <5th centile for GA (worse outcome) 		ome)
	Maternal	Placental
	- Poor nutrition	- Placental insuffiency
	- Smoking	- Essential HTN
	- Alcoholism	- Obesity (leptin resistance)
	- Alcoholism - Drug abuse	- Obesity (leptin resistance) - CKD
	- Alcoholism - Drug abuse - Early CVS disease	- Obesity (leptin resistance) - CKD - Pregnancy induced HTN
Causes	 Alcoholism Drug abuse Early CVS disease Cyanotic heart disease 	 Obesity (leptin resistance) CKD Pregnancy induced HTN Velamentous cord insertion
Causes	 Alcoholism Drug abuse Early CVS disease Cyanotic heart disease HTN, DM 	 Obesity (leptin resistance) CKD Pregnancy induced HTN Velamentous cord insertion
Causes	 Alcoholism Drug abuse Early CVS disease Cyanotic heart disease HTN, DM Obesity (Leptin resistance) 	 Obesity (leptin resistance) CKD Pregnancy induced HTN Velamentous cord insertion
Causes	 Alcoholism Drug abuse Early CVS disease Cyanotic heart disease HTN, DM Obesity (Leptin resistance) Pulmonary insufficiency 	 Obesity (leptin resistance) CKD Pregnancy induced HTN Velamentous cord insertion
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Causes IUGR RF	 Alcoholism Drug abuse Early CVS disease Cyanotic heart disease HTN, DM Obesity (Leptin resistance) Pulmonary insufficiency Anti-phospholipid syndrome Hereditary thrombophilia's Previous Hx HTN, DM Twins Smoking, Alcohol, drugs 	 Obesity (leptin resistance) CKD Pregnancy induced HTN Velamentous cord insertion Fetal IU infections: TORCH Congenital anomalies
Causes IUGR RF	 Alcoholism Drug abuse Early CVS disease Cyanotic heart disease HTN, DM Obesity (Leptin resistance) Pulmonary insufficiency Anti-phospholipid syndrome Hereditary thrombophilia's Previous Hx HTN, DM Twins Smoking, Alcohol, drugs Maternal anemia (Hgb < 10) 	 Obesity (leptin resistance) CKD Pregnancy induced HTN Velamentous cord insertion Fetal IU infections: TORCH Congenital anomalies

	 1-16 weeks: mostly cellular hyperplasia
Phases of	 16-32 weeks: both hyperplasia and hypertrophy
fetal growth	 >32 weeks: mostly hypertrophy
	 Hence early IUGR affect number and have a global
	(symmetrical) effect, while later IUGR size will be affected
	 High altitudes (smaller)
Factors	 Multiple gestation
influencing	• Race, Gender
Intrauterine	 Smoking, Alcohol
growth	 Socioeconomic status
	 Pathologies: Maternal, Fetal, Placental
	• Can occur in:
	- Multiple gestations
	 Chromosomal abnormalities: Triploidy and Tri 18: very early
Early IUGR	and severe, Tri 13: less severe, Tri 21: no IUGR but short femur
	and humorous
	- Cardiac malformations
	 Early pregnancy infections: rubella, CMV, toxoplasmosis
	• 1/3 of all cases
Symmetric	 Fetus is small (HC, AC, FL), normal HC:AC ratio
IUGR	 Growth rate is decreased
	 Diagnosed early: mostly due to IU infections or anomalies
	 Early insult affecting the cell number
	• 2/3 of all cases
	• Nutritional (DM, HTN,)
	• Found late at 2 nd , 3 rd trimester
	Placental insuffiency
	 Slow AC growth, normal HC and FL; the bran is prefentially
	spared at the expense of abdominal viscera (HC > AC)
Asymmetric	- Why AC slow? Because not enough O2 supply reach the fetus,
IUGR	so the blood goes to the vital organ (heart, CVS) and less supply
	reach the kidneys and intestines (hence the small AC)
	- Pancreas and liver are the one mainly affected
	- Glycogen utilization by liver (failure of storage), liver shrink,
	decreased AC; preferential shunting to brain (maintained HC)
	- Due to the liver changes: the fetus is at risk of obesity, DM
	later in life

	GA measurement must be correct
	 Serial Ultrasound Measurements (more than 2 w interval):
	- it detects 50-90% of IUGR
	1) Biparietal diameter (BPD)
	2) Head circumference (HC)
	3) Abdominal Circumference (AC): most effective parameter,
	because it is affected in both symmetric/asymmetric
	4) Femoral length (FL)
	- HC:AC ratio, FL:AC ratio (to know is it symmetrical or not)
IUGR Dx	5) Amniotic fluid volume: Oligohydramnios in IUGR
	6) Calculated fetal weight, Estimated fetal weight (EFW)
	7) Umbilical and uterine artery Doppler
	8) Transcerebellar distance
	9) Cheek-cheek diameter
	- Amniotic fluid source: <12 weeks (shedding of cells) > 12
	weeks (fetal urine), so when the blood supply to the kidneys
	decrease due to IUGR, there is less urine output so less fluid
	- As pregnancy advances the HC remains > AC, until 34 weeks
	the ratio is nearly 1, After 34 weeks the normal pregnancy is
	associated with AC > HC
	• <u>Pre-pregnancy</u> :
	 improve nutrition, avoid smoking
	 check the presence of maternal causes
	 women with antiphospholipid syndrome and a previous IUGR:
	give low-dose aspirin (81mg/d) in early pregnancy
	 for women with hereditary thrombophilia's: give low-dose
IUGR Mx	heparin (5000 U twice daily), with/out low-dose aspirin
	** so aspirin and heparin are used as prophylaxis
	• <u>Antepartum</u> : with an IUGR case:
	 Avoid maternal preventable causes (better nutrition, smoking)
	 Work leave (if work fatigue), hospitalization (will increase the
	uterine flow hence better nutrition)
	 Aim is to deliver before fetal compromise but after lung
	maturation, so you need monitoring by NST, AFI,

	 Based on the fetus: a) US normal growth, Fetal monitor normal – no intervention b) US: Strongly suggest IUGR: consider delivery at 34 weeks if at risk of fetal death, also pulmonary maturation should be documented by amniocentesis c) US: equivocal for IUGR: bed rest, serial US (3 week interval), fetal surveillance, assessment of fetal movements (kick counts), Doppler-derived umbilical artery systolic-diastolic ratios are abnormal in IUGR fetuses
	 Labor & Delivery: IUGR per se is not a CI for IOL C/S if fetal distress: in labor these high risk patients should be monitored to detect earliest fetal distress evidence Combined obstetric-neonatal team are needed in fear of fetal asphyxia
	 <u>After Delivery</u>: Examine infant to R/O congenital anomalies, chronic infection Monitor fetal blood glucose (due to no adequate hepatic glycogen stores): hypoglycemia is common Hypothermia is uncommon RDS is more common in presence of fetal distress due to fetal acidosis (which reduces surfactant synthesis and release)
When to Deliver	 Fetal lung maturity achieved Absence or reverse end diastolic flow velocity of umbilical artery wave-form In PT IUGR: it should be based on maternal health, fetal function tests, biochemical tests of fetal lung maturity
IUGR Prognosis	• should be monitored, short-term outlook is good, but there is higher risk of co-morbidities in the adulthood
FIUSIOSIS	

Early Pregnancy Complications & Miscarriages

- **Definition**: <12 w of gestation
- Investigations: Routine (urine analysis, CBC) + US + β HCG
- Gestational Sac (GS): 1.3 mm/day, seen around 4th w TVUS
- Yolk sac: appear at 5 w, disappears at 10 w
- CRL: crown-rump-length: to estimate GA

o Human chorionic gonadotrophin βHCG: most useful test

- double every 48 hr till 8 week (plateau, max: 100K)
- increase <66% over 48 hr is associated with EP, miscarriage
- Discriminatory zone: 1000-2000 TVUS, >4500 TAUS

	Miscarriage
Definition	- Vaginal bleeding w/o loss of pregnancy b4 24 w
	- Early ≤ 12 w, Late ≥ 12 w
Terms	• Clinically detected: US & detect heart beat at the 6 th w
_	 Biochemically detected: pregnancy test +
Causes	• 1 st Trimester: Genetically 50% (chromosome abnormal)
_	 2nd: inf, unexplained (20%), uterine abn., Cx weakness
Types	
Threatened	 any vaginal bleeding, painless/painful b4 24 w
	- Dx: US, Rh, Speculum, βHCG
	- RPOC (retained product of conception) become infected
Septic	 Sx: offensive vaginal discharge
	- Mx: admission, IV antibiotics, Surgical: evacuation
	 embryo has died or has not been developed normally
Missed	- Dx: US (GS ≥ 25mm) with no embryo or no FHB when
	CRL ≥ 7mm
	- Sx: asymptomatic

	 some RPOC passed and some remained
Incomplete	- Dx: clinical: tissue seen passing by US, HMB because
	cervix cant contract due to RPOC
Complete	 Endpoint miscarriages: all product of conception passed
	- Dx clinical: no pregnancy Sx, US: empty uterus
	o Expectant:
	 90% will miscarry within 3 w, rescan and contact early
	pregnancy unit
	- Complications: infection, hemorrhage
	o Medical:
Мх	 Uterotonic agent: misoprostol (PG)
	- CI: mitral stenosis, HTN, hemoglobinopathy, anti-
	coagulation, asthma
	o Surgical:
	• Vacuum, E/C, D/C
	o <mark>Anti-D</mark> : >12 w
	Pregnancy of unknown location (PUL)
	 + pregnancy test, but no IU/EU pregnancy on TVS
Definition	• 50% failing pregnancy, 27% very early IU pregnancy, 9%
	complete, 14% early ectopic
Mx	HCG repeat in 48 hr
	• repeat TVS when HCG >1000
	• follow up
	Recurrent Pregnancy Loss (RPL)
Definition	2 or more consecutive pregnancy loss <24 w gestation
	- unexplained, age, uterine abnormality
Causes	- abnormal parental karyotype
	 anti-phospholipid antibody syndrome
	- Genetic thrombophilia
	·

 Karyotyping HSG +/- hysteroscopy pelvic US Antiphospholipid syndrome screening: anti-cardiolipin Abs lupus anti-coagulant DRVVT (Dilute Russell's viper venom time) Parental chromosomal abnormality: counseling, pre- implantation genetic diagnosis (PGD), pre-implantation genetic screening (PGS) Mx Anti-phospholipid: LMWT heparin, low dose aspirin Congenital uterine anomalies: Hysteroscopic resection of seprum Unexplained Definition is the implantation of a fertilized ova outside the endometrial cavity, it results because of a problem during the journey of the ova from the tubes to the cavity F Tubal damage (from a previous surgical infection) Hx of previous EP (25% more risk) Smoking Altered tubal motility Hx of infertility Hx of fulfiple sexual partners PID Contraception: all contraception methods will cause a decrease in risk of ectopic pregnancies as they decrease the risk of pregnancy in general, except for Mirena IUD because it alters the motility of the tubes Massive hemorrhage Infertility Death 		
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 Massive hemorrhage Complicate Infertility Death 	Sx	Triad: pain, amenorrhea, vaginal bleeding
Complicate • Infertility • Death		Massive hemorrhage
• Death	Complicate	• Infertility
	••••••	• Death

Dx	 Clinically by: HCG, US (empty uterus, adnexal mass,
	fluid collection), then laparoscopy (GS!!)
	• Expectant: if HCG <1000
	 Medical (MTX): Methotrexate (chemical agent, anti-
Мх	metabolite) if stable pt, and HCG <5K, and sac <3.5 cm
	 Surgical: laparotomy or laparoscopy with
	salpingectomy, or salpingostomy

Uterine anomalies are caused by any dysfunction in the development of the Mullerian ducts, any error in the formation, descend, fusion, separation or resorption of the separating septum will end up by a uterine anomaly.



Class II: caused by failure of formation or descend by one of the Mullerian ducts, which will cause a small uterus leading to RPL or preterm delivery here we can't do anything except expectant Mx, & after a few failed pregnancies the uterus will expand in size and we may have a normal pregnancy.

Class III: this is the worst uterine anomaly but has the best obstetric outcome, caused by (complete) failure of fusion after normal descend, here the pt. will end up with two different cervixes and uterine.

Class IV: here we have (incomplete) failure of fusion, only in the upper part of the uterus, we have one cervix and one mid cavity but two upper parts.

Class V: normal descend and fusion, at the site of the fusion we have a separating septum, this septum should normally resorb but here it doesn't resulting in mildest uterine anomaly that is associated with with the worst obstetric outcome, because the septum is an avascular fibrous tissue, so unhealthy for implantation.

Class VII: in the past a drug called Diethylstilbestrol that was used for pregnancy support, it was stopped because it leads to congenital anomalies in female babies, it results in a small room T-shaped uterus with abnormal tubes.

Labor & Delivery

• Labor: is a physiological process during which the products of conception are expelled outside the uterus, it's a clinical Dx

• Labor: the onset of regular, painful contractions with progressive cervical effacement and dilatation accompanied by the descent of the presenting part

what initiate labor? We don't know exactly (theories – read them)

• in primigravida the dilation is slow, thus if she admitted because she could sense contractions we leave her a little bit to check if there is dilation progression if present then its labor, if not then send her home

• Factors that influence the progress of labour:

o Power:

a. uterus contractions:

- inorder for contractions to be efficient 3 things need to apply:

- 1. Frequency: 3-5/10 min
- 2. Intensity: strong (>50 mmHg) measured by CTG
- 3. Duration 40-60 seconds
- b. mother pushing (additional force)
- o Passenger (baby)
- o Passage (Pelvis)

• Zero station: on ischial spines

• **Moulding**: happens because the sutures are not closed: it is the change in the shape of the fetal head from external compression leading to reduction in its diameter

o Ranking:

0	No molding
+1	Sutures opposed
+2	Overlapped but reducible
+3	Overlapped but not reducible (mostly lead to obstructed labor)

• Braxton Hicks Contractions:

- mild, irregular, non-progressive contractions that may occur from 30 weeks gestation (mc after 36), it does not induce labor (don't apply the rules of labor contractions, also called false labor)

• Cervical changes during labor:

o Dilation and Effacement:

- Dilatation: examined by PV examination

 Effacement: measured by either % > effacement cm > dilation, we try to feel the distance between the internal and external orifices (os – length of the cervix because in pregnancy it shortens)

- Normally the cervix length is 2.5-3.5 cm during pregnancy so if you measured it as 2 cm then its 20% effacement

- In primi effacement always occur before the dilation, while in the multipara, it might occur after or together

- these changes are a result of profound alteration in biochemical properties of cervical tissue and include:

- a. reduction in collagen concentrations
- b. increase water content
- c. change in proteoglycan/glycosaminoglycan composition
- d. Rearrangement and realignment of collagen

• Rupture of fetal membranes (ROM):

- vital part of normal labor, might be done mechanically
- 90% remain intact until after the onset of labour
- 10% of women rupture prior to the onset of labour (PROM)

True Labor	False Labor	
Contraction occur at regular	Irregular	
intervals & become more frequent		
Duration of each contraction	Remain unchanged (long/short)	
gradually increases		
Intensity becomes stronger	Remain unchanged	
Cervix progressively dilates	Doesn't dilate	
Don't respond to simple analgesia	Responds	

Mechanism of Labour (LOA Position) the baby is responsible for the 7 cardinal movements

- 1. Head floating before engagement
- 2. Engagement, Descent, Flexion
- 3. Internal Rotation
- 4. Complete rotation, beginning extension
- 5. Complete Extension
- 7. Expulsion

(Engagement, Descent, flexion, internal rotation, extension, external rotation, expulsion)

** External Rotation (**Restitution**): This is the spontaneous realignment of the head with the shoulders.

** Expulsion. This is anterior and then posterior shoulders, followed by trunk and lower extremities in rapid succession

**** Shoulder dystocia** occurs when a baby's head passes through the birth canal and their **shoulders** become stuck during labor. ER

	Stages of Labor
	 Latent first stage of labour: period of time (hours even days!), not necessarily continuous when: there are painful contractions & there is some cervical change, including cervical effacement and dilatation up to 4cm
1 st Stage	 Established first stage of labour (Active Phase): when there are regular painful contractions & characterized by progressive cervical dilatation from 4cm. In Primi: 1cm/hr (1.2 to be exact) In Multi: 2cm/hr (1.5 cm to be exact)
	- <u>Dysfunctional labor patterns</u> : lack of progress in any phase of cervical dilatation
	- NICE : active phase in primi lasts avg 8 hrs (unlikely up to 18 hr), multi lasts 5 hrs (unlikely up to 12 hr)
	- Listen to HR: every 15 min after a contraction & for 1 min
2 nd Stage	 Starts with fully dilation includes propulsive and expulsive phases usually they wait an hr before they tell her to start pushing unless they are afraid for the baby then they wait a full 2 hrs Listen to HR: every 5 min or after every contraction NICE: nulliparous, birth is expected within 3 hr of the start of the active 2nd stage of labour, for multi women within 2 hr
	o Features of expulsive: - mothers irresistible desire to push - perineum distension - anus dilation

	o Management of 2 nd Stage:				
	- maternal position				
	- bearing down				
	- observation				
	- fetal monitoring				
	- uterine contraction				
	- descent				
	o Consequences of unduly prolonged second stage of labour:				
	• <i>Fetus</i> : acidosis, hypoxia				
	 Mother: urinary tract dmg, vesicovaginal fistula formation 				
	- delivery of the placenta (<i>within 5 m</i>), cord & membranes				
	- includes separation and expulsive phases				
	- prolonged if not completed within 30 min of the birth with				
	active Mx and 60 min with physiological Mx (NICE)				
	o Signs of placental separation:				
	- uterus become globular and firm				
3 rd	- gush of blood (placental detachment from the uterus wall)				
Stage	- the uterus rises because the placenta passes sown into the				
0	lower uterine segment and vagina				
	- the umbilical cord protrudes farther out of the vagina				
	o Active Mx of 3 rd stage:				
	- Syntometrine (oxytocin) + anterior shoulder delivery				
	- Placenta delivery by controlled cord traction				
	- Early clamping and cutting of the cord				
	, , , , , , , , , , , , , , , , , , , ,				
4 th	- The 1-2 hr post-delivery when the tone of the uterus is				
Stage	established and the uterus contracts down again, because				
	most complications after delivery happen at this stage				

- **Partogram**: (v. important, u should know about it!):
- represent the changes that occur in labor (to identify deviations.
- Using a partogram leads to \downarrow operative births and \downarrow use of oxytocin

Monitoring in labour (recorded on the partogram):

- Intermittent (for low-risk) /continuous (for high-risk) FHR monitoring
- Contractions should be assessed every 30min.
- PR should be checked hourly.
- BP & temperature should be checked 4-hourly.
- VE every 4h to assess progress.
- Urine 4-hr (or when she go to WC or when passed for ketones/protein)
- Regular bladder emptying should be encouraged



• Mx of Labor:

o on Admission: Check AN record, define risk, G/E pallor, edema, BMI, V/S, Heart & lungs, Urine dipstick, Abdominal exam, Vaginal exam, Position in labour, Monitoring progress in labour, Pain relief

o Causes of Labour pain:

- Hypoxia of contracted myometrium
- Compression of nerve ganglia in the cervix and lower uterus
- Stretching of the cervix during dilatation
- Stretching of the peritoneum overlying the uterus

o Analgesic methods during Labour:

- Psychological Counteract "fear-tension": 30-40% don't need analgesia
- Narcotics: pethidine
- Inhalational: Entonox

- Regional analgesia: <u>epidural</u> (continuous), <u>spinal</u> (no catheter, fast, lasts 2 hr, not continuous), <u>CSE</u> (combined spinal and epidural)

Narcotics in Labour				
Advantages	Disadvantages	Contraindications		
• Ease of	 Inadequate analgesia 	 Previous Allergic 		
administration	 Nausea & vomiting 	reactions		
 rapid analgesia 	 Psychic disturbance 	 Current mono- 		
 Low incidence of 	 Delayed gastric emp. 	amine oxidase		
serious side effects	 Neonatal Resp. Depr. 	inhibitors		
 Antagonists 				
available				

o Transcutaneous electrical nerve stimulation (TENS):

- Low grade electronic waves to nerves supplying the uterus via skin electrode. Provides good pain relief to 25% of pt. and it Carries no risk

o Epidural Analgesia:

- will provide analgesia in up to 90% , 16–20-gauge needle in L2–L5.

- Isobaric bupivacaine solution or with fentanyl.
- Continuous fetal monitoring Foley's catheter

- <u>CI for spinal/epidural</u>: maternal refusal, hypovolemia, severe back deformities, local infection, coagulation disorders

Maternal Hazards of Epidural Analgesia			
Immediate	Delayed		
 <u>Dural tap</u>: leads to "spinal" headache 	- <u>Severe spinal</u>		
 <u>Total spinal</u>: loss of sensory and motor 	<u>headache</u> ; due to spinal		
function, unconsciousness, hypotension,	tap, Keep the patient		
apnea, results from subarachnoid injection	lying down, Slow IV		
- <u>Hypotension</u> : avoided by nursing the pt on	injection of 1-1.5 L		
her side and IV infusion of Ringer lactate	saline over 24 hrs		
/lower risk due to low-dose local agents	- Urinary retention		
- <u>Motor paralysis</u> : reduces maternal	- <u>Sepsis;</u> unlikely if		
expulsive effort, tends to prevent fetal	bacterial filter used		
head rotation, and makes instrumental	- Diminished sensation		
delivery more likely	of dermatomes;		
 <u>Toxic reactions</u>: to local anesthetic agents 	temporary effect		

o Performing an Episiotomy:

- Infiltrate perineum with local anesthetic agent
- we do it after crowning because at this stage the muscles are very stretched & have the least vascularity to allow minimal loss of blood
- Episiotomy is considered a 2nd stage laceration.
- note: it's important that absorbable sutures be used for closure
- each layer is sutured separately (vaginal mucosa continuous suturing
- / muscle layer interrupted / Skin interrupted or subcuticular)

o Lacerations:

First degree: Involve the *fourchette, perineal skin, and vaginal mucosa,* Second degree: as 1st stage *and muscles of the perineal body* Third degree: as 2nd stage *and involve anal sphincter* Fourth degree: as 3rd stage and through *rectal mucosa*

o Apgar Scoring System:

- The condition of the baby is assessed at 1, 5, and 10min using the Apgar scoring system, if all is well, baby is handed to the mother ASAP

o Care immediately after delivery:

- Most complications occur in the first 2h after delivery, including; PPH/uterine inversion/hematoma
- Usually women are kept in LW to observe
- Where there is an increased risk of PPH an oxytocin infusion should be given prophylactically for 3–4h.
- Encourage skin-to-skin contact ASAP (don't separate for the 1st hr).
- Support should be provided for breast-feeding (1st hr)

• If no complications during these 2h, the mother may then be transferred to the postnatal ward: some women may then go home after a further 3–4h of observation.

o Poor labor progress Types of delay in progress:

- Latent phase delay is difficult to define/ can last 2–3 days or stop
- <u>Delay in the first stage</u> consists of dilatation of <0.5–1 cm/h or crossing the 4-h partogram line
- Delay in the second stage

o Obstruction is suspected if:

- Arrest of cervical dilatation & descent of the presenting part.
- Large caput
- Excessive moulding,

- Edematous cervix and vulva
- Maternal or fetal distress.
- Ballooning of LUS, formation of a
- retraction ring

o Amniotomy: Artificial rupture of the membranes (ARM):

- often shortens labor length if the woman is contracting regularly

o Diagnosis of Normal Labor:

Spontaneous onset	Spontaneous expulsion	Singleton
Presented by vertex	Within a reasonable time (not <3h	Alive
	and not > 18hrs)	
Through birth canal	Without complications for both	Term

Obstructed Labor (Dystocia)

	despite strong uterine d	contractions		
	Passenger		Passage	
	Head.		Contracted (due to malautrition)	
	Large retainead (oig for that pervis) Hydrocenhalus (brain surrounded by fluid, which m	akes the skull swell)	Deformed (due to trauma polio)	
	Presentation and position		Soft tissue	
Etiology	Brow face, shoulder		Tumour in the pelvis	
	Persistent malposition		 Viral infection in the uterus or abdome 	
	Twin pregnancy.	Scars (
	Locked twins (locked at the neck)			
	Conjoined twins (fused together with some shared organs)			
	 Partogram 			
	• General, Abdominal,	Vaginal Examinati	ions	
	 History: prolonged labor, labor become more severe and 			
	frequent, bearing down			
	Abdominal Exam:			
	• Abuommu Exum.			
	- General: maternal distress, painful state, scanty urine, foul			
	smelling meconium from vagina			
Dx	 Inspection: tonically contracted uterus, distended full 			
	bladder Badnl's ring (in multi) uterine inertia (in primi)			
	nalpation: utorus is to	n: uterus is tender liquor all drained fetal heart		
	- <i>palpation</i> : uterus is tender, liquor all drained, fetal neart			
	sounds absent			
	• Vaginal Exam:			
	 Edematous vulva, dry hot vaginal mucosa, cervix poorly 			
	applied to presenting part, cervix loosely handing/partially			
	dilated meconium draining caput on presenting part			
	unated, meconium draining, caput on presenting part,			
	molding, if uterus is rup	otured the fetal pa	art will be palpable	
	Fetal Complication	s:		
	 Neonatal sepsis 	 Intracania 	l hemorrhage	
Complication	• Facial injury	 Moulding 	C C	
•	• Sovere asphyvia			
	• Severe aspriyxia • Death			

	• Fail to empty the bladder (trauma/	blood)	
	Pressure necrosis to the bladder/u	rethra	
	• Cut to the blood supply	6	
	• Fistula (due to compression of the soft tissue, between the		
	vagina and the bladder/rectum/urethra/ureter)		
	• PPH	• Sepsis	
	Hypovolemic snock/death	Paralytic lieus	
	Annual detachment of the cervix	Oterine rupture	
revention	Antenatal care		
	• Intranatal: partogram		
	• <u>Immediate</u> :		
	- IV and catheterization		
	- Blood grouping and cross matching		
	- Correct maternal dehydration		
	- Adequate analgesia		
	- Broad spectrum antibiotics	idocic	
Mv	- Sodium bicarbonate infusion for acidosis		
	Agents used to delay premature ut	s erine activity include	
	- Magnesium sulphate		
	- Reta-mimetics (Terbutaline)		
	- Oxytocin Antagonist		
	- CCB (Nifedinine)		
	- Adrenergic beta-recentor agonists (Fenoterol)		
	- NSAIDs (Indomethacin)		
	• Symphysiotomy (rarely used now)		
	• Episiotomy		
Deliverv	 C/S or Vaginal (if vaginal delivery n 	ot risky)	
1	• Empty the bladder to avoid the fist	ula risk!	

Induction of Labor (IOL)

	Artificial stimulation of uterine contraction before the onset				
IOL	of spontaneous labour				
	 Done after 24 weeks GA (Termination is done < 24 weeks!) IOL is always innation[]] 				
	 IOL is always inpatient!!! 				
Augmentatio	 stimulation by spontaneous contractions that are considered 				
n	inadequate (most common obstetric intervention)				
	 Prolonged pregnancy (beyond 41 weeks – most common!) 				
	• PROM				
	 Fetal: IUGR, Oligohydromnios, Isoimmunization, IUFD 				
	 Note: IUFD is fetal death after 24 weeks so it's induction and 				
Indications of	not termination				
IOL	 Suspected macrosomia 				
	Social factors				
	 Maternal request 				
	 Maternal diseases (DM, HTN, SLE) 				
	 Pregnancy complications 				
Predictors for	 GA at induction (the advanced GA the better) 				
successful IOL	 Parity (In multi, the induction is faster and quicker) 				
	Modified bishops score				
	 Any CI for vaginal delivery is a CI for IOL! 				
	• Placenta previa/vasa previa				
	transverse lie				
CI for IOL	prolapsed umbilical cord				
	active genital herpes				
	 previous classical uterine incision/2LUSC/myomectomy 				
	 maternal/fetal anatomical abnormalities that CI VD 				

		Modifie	d Bishop Score		
Cervical Fea	ature		Pelvic	Score	
		0	1	2	3
Dilatation	(cm)	<1	1-2	2-4	>4
Length of cerv	vix (cm)	>4	2-4	1-2	<1
Station		-3	-2	-1/0	+1/+2
Consister	ncy	Firm	Avg	Soft	
Positio	า	Posterior	Mid/Anterior		
Th	e cervix i	s favorable v	when modified b	ishop score >8	3
		A score <4 is	s unfavorable cer	rvix	
	- Where	e do we indu	ce? In Antenatal	ward	
	the labo - Alway check ir the pati	or ward s remember ndications fo ent and mak	: before starting r induction, chec ke sure the baby	induction, cor k bishops sco is cephalic, ba	nfirm the GA, re, examine aseline CTG
Methods of IOL	 check indications for induction, check bishops score, examine the patient and make sure the baby is cephalic, baseline CTG PG (Dinoprostone), Oxytocin, Misoprostol a) PG (PGE2) different formulas: tablets, gel, slow releasing pessary 24 hr give 3 doses, examine every 6 hours with tablets/gel continuously monitor the CTG in first hour after inserting the first pessary if you are happy with the CTG and it's reactive then the patient can mobilize and walking if we induce by PG we should continue with oxytocin (once the cervix is favorable), but we don't start oxytocin until 6 hours after the last dose of PG PG very important to be used specially in unfavorable cervix Asthmatics are not suitable to take PG 				

b) Oxytocin (syntocinon)

forms: syringe, infusion pumps (pump advantage is the you can adjust the dose & change it according to the contractions)
still you can increase the syntocinon till you have adequate moderate contractions

- We don't start it with intact membrane

What is the advantage of oxytocin over PG? we can stop it, it has a short duration (PG only if it's a pessary we remove it)
we should not start oxytocin before 6 hours after last dose of PG. why? Risk of hyperstimulation and ruptured uterus
c) *Misoprostol* (*Cytotec*):

- not available and we don't use it

- unlicensed for IOL, used in termination, IUFD, clinical trials

Mechanical methods:

a) <u>Stripping of the membranes</u> (<u>Stretch</u> & <u>Sweep</u>):

- do vaginal exam, but to be able to do that, cervix should be dilated atleast admit 1 finger (if closed, you cannot do it)

- Technique: admit 1 or 2 fingers and move between cervix and membranes: this will lead to release of PG

- 3% will go in spontaneous labor in 7 days

- we offer this for ladies by 40 weeks

- midwife usually do that

b) <u>Catheter</u> or <u>Laminaria tents</u>:

we use foley's catheter with 2 balloons one endo one ecto cervix & we inflate both to compress the cervix and shorten it
Laminaria is a type of herbs, you put it in the cervix and it absorbs water and enlarge which will dilate the cervix

- Adv.: Simple, low cost, less SE, no need to keep inpatient

- Dis.: difficulty of insertion, risk of infection

c) <u>Caster oil</u>:

- might be used for better sexual intercourse

- it enhances cervical ripening
| | - it also stimulate the bowel movement (which is the worst |
|--------------|---|
| | thing to do for a pregnant lady) |
| | Previously, they were using enema to make clean |
| | environment for delivery |
| | d) Surgical ARM (Amniotomy): |
| | - we initiate or augment labor |
| | - other indications: apply fetal scalp electrode |
| | - fetal blood sampling |
| | - risks: cord prolapse, chorioamnionitis, rupture of vasa previa |
| | Hyperstimulation: Tachysystole, Hypertonus |
| | - Mx: reduce/stop oxytocin, consider tocolytics (terbutaline) |
| | • Fetal distress: |
| | - if not due to hyperstimulation, do immediate C/S |
| | Failed induction (we try 3 doses of PG): |
| | - What to do (Options): |
| Complication | a) repeat attempt at later GA |
| of IOL | b) Wait for the labor to start spontaneously |
| | c) Schedule a C/S |
| | d) Consider use of alternative cervical ripening strategies |
| | • <u>C/S</u> |
| | <u>Ruptured uterus</u>: Uterine Dehiscence (Disruption of the |
| | uterine muscle with intact uterine serosa) |
| | • <u>SE of drugs used</u> |
| Mx of labor | Antenatal ward |
| after | • LW – high risk |
| induction | Oxytocin + continuous FHR & uterine monitoring |
| | lady 41 weeks, previous C/S. Can we induce her?! |
| | In Fact, we have small randomized clinical trials (Inadequate |
| | data), but we may offer Stretch and sweep or catheter for |
| Cases | induction. PG some units Allow to be used in such a case. But, |
| | In General NO we cannot use. |
| | 2) Grand multipara? = para 5 and more |
| | Be aware In Both cases , the Risk of rupture uterus is high !! |
| | |

		Post-Date	9	
Info	• > 42 weeks (>294 d) • Incidence 5-10%			5-10%
	• 30% recurrence		it is not a p	oathological condition
	Materi	nal		Fetal
	Increase operation	ative	• Post-ma	turity syndrome
	delivery, hemorrhage and		More mortality/morbidity risk	
Risks	infection		• More sti	ll-birth risk
	 Psychological 	morbidity	• Intrapar	tum fetal hypoxia
			More bi	rth trauma and
	" more commor	n in primi "	shoulder o	lystocia risk
			Macrosc	omia
	 IOL or CS (mai 	nly at 41 we	eks)	
	 if mother refu 	ses IOL do tl	ne following	g:
	- Expectant Mx			
	 a) Fetal surveillance: CTG/US twice weekly 			
	b) Biophysical profile			
		BIOPHYS	ICAL PROFILI	
	fetal breathing movements	≥1 episodes in 30 min e	ach lasting <a>30 sec	absent or no episode >30 sec in 30 min
IVIX	gross body movements	three or more discrete body or limb movements in 30 min (episodes of active continuous movement = a single movement)		less than 3 episodes of body or limb movements in 30 min
	fetal tone	≥1 episodes of active extension with return to flexion of fetal limb(s) or trunk; opening and closing of hand is considered normal tone		slow extension w/return to flexion, moveme of limb in full extension, or fetal movement absent
	reactive fetal heart rate	≥2 episodes of accelerations (≥ 15 beats/min) in 20 min, each lasting ≥ 15 sec and associated with fetal movement		< 2 episodes of accelerations or acceleration of < 15 beats/min in 20 min
	qualitative amniotic fluid volume	>1 pockets of fluid measuring > 1 cm in 2 perpendicular planes		pockets absent or pocket < 1 cm in 2 perpendicular planes
		score		notes
	normal equivocal	8 – 10 (it amniotic fluid i 6	ndex is adequate)	CNS is functional & fetus is not hypoxemic
	abnormal	< 4 Image Cred	it: wordpress.com	along w/oligohydramnio→ labor induction
	• reduces rates	of IOL for po	ost-term, we	e determine the GA b
Dating Scan	a) Crown rump:	measurem	ent from 10	weeks 0 days to 13
Dating Scan	· · · ·	/	time a vula aa	nuchal transluconov
Dating Stan	weeks 6 days, it	s the same	time we go	

PTL (Pre-term Labor)

Definition	Between 24 – 37 (≥ 37 is term)			
	Previous PTL (strongest indicator)			
	 Infections: Intra-uterine (ascending - bacterial vaginosis), Extra- 			
	uterine (pyelonephritis)			
	 Cervical: dilation, surgery, trauma 			
RF	 Fetal: congenital or chromosomal abnormality 			
	 Uterine: abnormality, overdistension (multi, twins, 			
	polyhydramnios)			
	 Social: extremes of age, BMI <19, short pregnancy interval, 			
	poverty, black, smoking, alcohol, drugs, stress, domestic violence			
	 Other: APH, PET, uteroplacental insufficiency 			
	 Abdominal pain (MC), colicky 			
Hx	 Vaginal Discharge 			
	• Gush of fluid			
	 Pressure (presenting part descended) 			
	• SFH			
	Lie, presentation			
PE	• Tenderness (infection)			
	Pooling of fluid in vagina			
	Digital: only if regular contraction already			
	• UTI			
	Placental abruption			
DDx	• Gastroenteritis			
	• Constipation			
	• Fibroids			
Investigate	• Fetal fibronectin (should not be seen 24-34 weeks)			
	• Cervical Length TVS			
	0 Bed rest			
	o Maternal steroids : to mature the lungs (RDS, between 24-34			
	w/up to 39w in elective C/S): Betamethasone, Dexamethasone			
IVIX	(admit b4 giving if the pt is diabetic)			
	o iocolytics (anti-contraction): opposite of uterotonics:			
	• p-agonists: MC			

	- S.E: hypotension, tachycardia, headaches, hyperglycemia,
	nypokalemia, the most serious is
	pulmonary edema & in rare cases maternal death
	- C.I: symptomatic cardiac disease, uncontrolled DM,
	hyperthyroidism, multiple pregnancies
	Oxytocin antagonist (Atosiban):
	- less S.E, similar effectiveness, but more expensive
	 Calcium channel inhibitors (Nifedipine):
	- easy to administer (orally).
	- S.E: headache, flushing, dizziness
	 Magnesium sulfate (MgSO4):
	 used for neurologic protection (not used as tocolytic)
	 S.E: flushing, headache, fatigue, diplopia, at toxic lvl of
	Mg (>10) respiratory depression, hypoxia and cardiac
	arrest, the DTR are depressed and lost at <10mg/dl,
	also, pulmonary edema might occur
	 Prostaglandin inhibitors (indomethacin – NSAIDS):
	 we do not use it after 32 weeks because it causes premature
	closure of ductus arteriosus, but the
	- it also causes oligohydramnios and thus we may use it in some
	cases of polyhydramnios, but it increases risk of
	necrotizing enterocolitis & intraventricular hemorrhage
	o Antibiotics: erythromycin, in complicated PTL (PROM)
	o In-Utero-transfer
	o Fetal Assess: US, NST (non-stress test (similar to CTG))
Mode of	- if breech C/S
Delivery	- Case-related
	 No Vacuum before GA 36, and Forceps before GA 34
	o Maternal: tocolytics risk, depression, underlying cause
Outcomes	o Neonatal: mortality, morbidity (neural), RDS, NEC, PDA ,
	Jaundice, Hypothermia, Feeding difficulty, lung disease, and
	Retinopathy
	• Minimize RF
	Treat infections
Mx of future	 TV cervical length – if short do cerclage
	Pessary
	 Progesterone (NOT ESTROGEN!)

PROM

Hx • Gush of fluid (Timing, Amount, Color, Odor) • SFH • Presentation PE • Tenderness (chorioamnionitis) • Speculum (Definitive Dx): pool of fluid, cough sign • Cord: prolapsed of not • NO DIGITAL EXAMINATION • Seminal fluid collection: sexual intercourse • Amniotic fluid: PROM DDx • Infections: UTI, Vaginal • Urinary Incontinence (color: yellow) • Leukorrhea: cervical glands ↑ active, ↑ discharge • Abruption of placenta (color: red) • Nitrazine test • Ferning Pattern • Genital tract swabs: HVS to rule out GBS infection • Maternal and Fetal wellbeing • US • AmniSure ROM test: 99% sensitive, 100% specific detects PAMG-1 protein marker • Based on GA • Admission (risk of labor, cord prolapse) • Medications: Mx	Definition	ROM prior labor, either at term or preterm			
 SFH Presentation PE Tenderness (chorioamnionitis) Speculum (Definitive Dx): pool of fluid, cough sign Cord: prolapsed of not NO DIGITAL EXAMINATION Seminal fluid collection: sexual intercourse Amniotic fluid: PROM Infections: UTI, Vaginal Urinary Incontinence (color: yellow) Leukorrhea: cervical glands ↑ active, ↑ discharge Abruption of placenta (color: red) Nitrazine test Ferning Pattern Genital tract swabs: HVS to rule out GBS infection Maternal and Fetal wellbeing US AmmiSure ROM test: 99% sensitive, 100% specific detects PAMG-1 protein marker Based on GA Admission (risk of labor, cord prolapse) Medications: Mx 	Нх	Gush of fluid (Timing, Amount, Color, Odor)			
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 Cord: prolapsed of not NO DIGITAL EXAMINATION Seminal fluid collection: sexual intercourse Amniotic fluid: PROM Infections: UTI, Vaginal Urinary Incontinence (color: yellow) Leukorrhea: cervical glands ↑ active, ↑ discharge Abruption of placenta (color: red) Nitrazine test Ferning Pattern Genital tract swabs: HVS to rule out GBS infection Maternal and Fetal wellbeing US AmniSure ROM test: 99% sensitive, 100% specific detects PAMG-1 protein marker Based on GA Admission (risk of labor, cord prolapse) Medications: Hydration 		 Speculum (Definitive Dx): pool of fluid, cough sign 			
• NO DIGITAL EXAMINATION • Seminal fluid collection: sexual intercourse • Amniotic fluid: PROM • Infections: UTI, Vaginal • Urinary Incontinence (color: yellow) • Leukorrhea: cervical glands ↑ active, ↑ discharge • Abruption of placenta (color: red) • Nitrazine test • Ferning Pattern • Genital tract swabs: HVS to rule out GBS infection • Maternal and Fetal wellbeing • US • AmniSure ROM test: 99% sensitive, 100% specific detects PAMG-1 protein marker • Based on GA • Admission (risk of labor, cord prolapse) • Medications: • Hydration		 Cord: prolapsed of not 			
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 Admission (risk of labor, cord prolapse) Medications: Mx - Hydration 		Based on GA			
Medications: Mx - Hydration		Admission (risk of labor, cord prolapse)			
IVIX - Hydration		Medications:			
	IVIX	- Hydration			
- IV Anti-biotics		- IV Anti-biotics			
- Tocolytics (Q)		- locolytics (Q)			
- Dexamethasone					
Chorinam - infection of the amniotic fluid & membranes, we deal with it by	Chorioam-	- infection of the amniotic fluid & membranes, we deal with it by			
nionitis Abx and immediate delivery	nionitis Abx and immediate delivery				

- Dx:
- Maternal pyrexia (>38) & at least 2:
- maternal PR (>100bpm), Fetal
- tachycardia (>160bpm),
- Uterine tenderness,
- Offensive vaginal discharge,
- Raised CRP

o Pre-viable ROM <24 w:

- by definition it is a miscarriage and some pts. Are given the option to terminate the pregnancy, before 2 years, a 20 weeks pregnant lady with history of infertility and miscarriages had ROM, even though the Dr. advised her to end the pregnancy she was determined to continue so they continued with Abx and follow up, at 24 & 28 weeks she was given steroids and gave birth at the 34 week as an emergency C/S for abruption and she delivered normal male baby, she was very lucky for this to happen.

- <u>Major risks</u>:

- chronic pulmonary morbidity, fetal limb contractions,

extremely preterm with co-existent morbidity/mortality

Examples of some Cases

- <u>Treatment</u>:

- Individualized Mx: after full & frank discussion with parents

o 24-34 weeks:

- confirmation of dx and presentation
- baseline FBC, CRP, swabs, and MSU (mid-stream urine)
- US for fetal wellbeing
- Steroids, oral erythromycin for 10 days
- MOD (either we wait for 37 weeks or we deliver at 34 weeks, the Dr. prefers the latter)

o **34-37 weeks**:

- controversial, immediate IOL (less hospitalization, perinatal infection & NN morbidity)

Antepartum Hemorrhage (APH)

" APH Weakens, PPH Kills "

Definition	bleeding from after 24 w and before delivery		
	• Abruption Placenta (mc – 30%)		
	 Placenta praevia (20%) 		
Causes	Uterine rupture		
	• Vasa previa		
	 Other: local causes, GU tumor, unknown 		
	 Rapid assessment of both mother & fetus 		
	 Quick history initially then detailed 		
	o Maternal assessment:		
	• Vital signs.		
	• Abd:		
	- SFH (Symphysial fundal height),		
Mx despite	- tenderness		
the cause	- lie & presentation, in placenta previa we said whatever the lie and		
	presentation, you deliver by a cs, while in placental abruption our target		
	is to deliver vaginally, but if the baby is transverse, you do CS		
	 NO Vaginal exam until a placenta previa has been excluded 		
	The 1 st investigation to do is U/S, to localize the placenta		
	• Assess bleeding		
	o Fetal Assessment:		
	• fetal heart		
	• If gestational age is >26 weeks: CTG not before 26 weeks, it is difficult		
	because there is fetal heart tachycardia		
	Placental Abruption		
	Premature separation of a normally sited placenta		
Definition	** the decidua (line of separation between placenta & uterus) is		
	detached from uterus (decidua = endometrium)		
	A. Concealed , patient complain of abdominal pain but she doesn't have		
Can be	any Accurint vaginal bleeding		
	B. Apparent, Accurint vaginal bleeding.		
	** Uterine pain is local while labor pain is generalized		

	 Vaginal bleeding: mostly dark 				
	 Abdominal pain: mostly constant 				
SSx	 Uterine tenderness 				
	 Uterine contractions (35% - indu 	ice labor PTL 25%)			
	 Fetal distress/fetal death (15%) 				
	 Disseminated IV coagulopathy: r 	non-clotting bleeding			
	Class	Presentation			
	0 • Asymptomatic				
	 Diagnosis is made retro 	spectively (finding)			
	 Mild/No vaginal bleedir 	ng			
	No maternal/fetal com	promise			
Classific-	 Moderate/No vaginal b 	leeding			
ation	2 • Possible maternal blood	d loss			
	(tachycardia/mild hypote	nsion/no coagulopathy)			
	Fetal distress				
	 Severe/No vaginal blee 	ding			
	3 • Maternal compromise	(tachy/hypo/coagulopa)			
	 Tense, tender uterus, 'woody hard' 				
	Intrauterine fetal death				
	• Clinical				
	• US:				
	- not used to Dx but to know when	 not used to Dx but to know where is the placenta, to exclude other 			
	causes, assess the fetal viability, c	onfirm GA			
Dx	- Retroplacental haematoma	- Retroplacental haematoma			
	• Lab tests:				
	- FBC, Blood group and crossmatch				
	- Urea, creatinine and electrolytes				
	- LFT				
	- Coagulation screen				
	- Kleihauer-Betke test: to quantify the feto-maternal hemorrhage or				
	blood transfusion between them				
	 Previous (MI! 10x!, 10%,25%) 	 Chorioamnionitis 			
	• FHx	• HTN			
	 Underlying thrombophilia's 	 Abnormal placentation 			
RF	• Trauma	• 个 parity			
	 Rapid uterine decompression 	 Smoking 			
	•				
	• 个 maternal age (>35 & <20)	 Drug misuse 			

	 Depend on: blood loss amount, mother/fetus status, GA 			
	• Resuscitate as indicated:			
	- Conservative: consider sterc	oids		
	- Delivery			
	• Case-Related:			
	- Maternal/Fetal Jeopardy: EF	C/S		
	- Term/stable/in labour: Vagi	nal delivery (main	target)	
Mx	- Pre-term/stable: Conservati	ve in hospital/ster	roids	
	• Notes:			
	We manage these patients as	In-patient, never	send the patient home	
	even if she is stable , if preter	m we give steroid	ls to enhance lung	
	maturity , <u>and Anti-D if neede</u>	<u>d</u> .		
	We wait 48-72 hours, if the pa	atient is stable an	d want to continue	
	pregnancy, send her home. if	mother is - & her	r husband is + we give ant	
	d , in abruption we need \uparrow d	ose		
	Maternal		Fetal	
	 Hypovolemic shock 	• IU	FD	
	• DIC •	PPH • Hy	/poxia	
Complica-	 ARF (mostly reversible) 	• Ar	nemia	
tions	 Couvelaire uterus (bruised, 	can cause • IU	GR	
	Atony of the uterus)	• Pr	eterm birth	
	 Ischemic necrosis of distal organs 			
	Feto-maternal hemorrhage			
	Placent	al Previa		
	Insertion of the placenta, partially or fully in the lower uterine segment			
Definition	(lower segment usually is formed after 28 w, b4 that the uterus is 1			
	segment), the placenta normally in the fundus			
	Minor I in lower segment but does not reach os			
Grading	II Reaches os but does not cover it			
	Major III Cover part of the os			
	IV Completely cove	ers the os		
	 Hx of PP with a previous 	 Abnormally sh 	naped uterus	
RF	pregnancy	• Age >35 (9x! >	>40 y)	
	 Uterine scars 	 Asian 		

	 Painless bleeding (main Sx – unlike abruption)
	• Bright red
Symptom	 Variable amount (from 'spotting to torrential/life threat)
	May be recurrent
	 Provoked by sexual intercourse or labor onset
	 The fetus is usually well and in good condition
	Shock (due to blood loss)
	• Presenting part is usually high/non-cephalic (or think of an abnormality
	that is blocking the lower part of uterus)
	Soft and non-tender uterus
Signs	 Digital vaginal examination is CI
-	Speculum exam: to exclude local causes
	• Note: Multipara mothers engagement is late, it's even could be at
	labor compared to primary, if not engaged at 38 week think of an
	abnormality like pp .
	• US: to know where is the placenta
	- Safe, more accurate
Dx	- we start by TA then TV
	• MRI:
	- Expensive/not superior
	 considered if suspected accreta when US is inconclusive
	Case-Related:
	- Maternal/Fetal Jeopardy: ER C/S
	- Term/stable/in labour: Vaginal delivery (main target)
	- Pre-term/stable: Conservative in hospital/steroids
	>> Timing of delivery:
	 The ultimate plan is to deliver CS (always) at 37 w
	• indications before 37w:
	- onset of labor
Mx	- fetal distress
	- severe growth restriction
	- intrauterine death
	- severe bleed
	 suspected placenta accrete (advisable before 36-37 w)
	• Anti-D as always

	• Notes:					
	- we deliver by cs (always) , grade of cs (is it elective or emergency) ?					
	emergency is grade 1 and we do it if there is a distress whatever the gestational age. Stable conservative and deliver as plan (elective) if					
	gestational age, Stable conservative and deliver as plan (elective) if					
	preterm and stable					
	- Timing – minor previa we deliver at 37 weeks					
	- Major at 36 - 35 w bcuz we worry about sudden bleeding					
	Morbidly Adherent Placenta					
- Placenta is	usually at the level of residua					
- Anterior pla	acenta previa more worrying than posterior previa					
- if no bleedi	ng think of adherent placenta					
• Placenta A	ccreta: 80% Villi penetrate through the <u>decidua</u> :					
- antenatal d	x using color/power Doppler US MRI					
- repeated C	/S with an anterior placenta previa					
- Dx in 3 rd sta	ige					
• Placenta Ir	i <mark>creta</mark> : villi penetrate into <u>myometrium</u> .					
• Placenta P	ercreta: through the myometrium to <u>serosa/bladder</u> .					
	Vasa Previa					
Definition	Fetal vessels traverse the membranes over the internal os					
	Classic Triad:					
	- Membrane rupture					
	- <i>Membrane rupture</i> - <i>Painless</i> vaginal bleeding					
	 Membrane rupture Painless vaginal bleeding Fetal bradycardia: this is fetal blood loss (not maternal) 					
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Dx RF Mx Complic-	 Membrane rupture Painless vaginal bleeding Fetal bradycardia: this is fetal blood loss (not maternal) Dx is usually at the time of ROM unless we did Doppler antenatally and saw the vessels over the internal os, and this is when we don't allow the vaginal delivery, we do a cs Colour-flow Doppler: crossing vessel Dx is usually confirmed after delivery on examining placenta and membranes Velamentous cord Accessory placental lobe Multiple pregnancy Emergency CS (crash: emergency section grade one) Fetal death from hypovolemia 					
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	Ruptured Uterus
Definition	Full thickness loss of integrity of the wall & visceral peritoneum
	 Painful bleeding (tearing): even + epidural (pain not relieved)
	• Lost FHR
Diagnostic	 Loss of station (fetal head): It is supposed to be head in a previous cs
Criteria	because you wouldn't allow a breech in a previous cs
	 Inability to identify uterine contractions: due to integrity loss
	 Unscarred uterus: no previous D/C, myomectomy or cs
Notes	 Any lady who had myomectomy should be asked if the doctor told her
	she can have a vaginal delivery, if she can't, that means the uterine
	cavity was opened, & it is better if she has a report.
	 VBAC: vaginal birth after CS, higher risk of rupture uterus
	 If the pt is in labor & there is a catheter, you will see hematuria
	 Have a low threshold for rupture uterus in a lady with previous cs
	 In labor you have to think and act, if the fetal heart rate is in variable
	deceleration, think if it's a rupture uterus, do an ER CS.
	 Classical C/S (opening in the upper uterine segment)
	Previous myomectomy
	Excessive oxytocin
	** The common indications for a classical CS (5%) include:
	- preterm
RF	 breech in a woman with an undeveloped lower uterine segment,
	transverse back - down fetal position,
	- poor access to the lower segment because of myomas or adhesions, or
	a planned cesarean hysterectomy.
	- The presence of cervical cancer is a rare indication.
	LUSCS: 0.5% lower uterine segment cs, the common procedure
	nowadays, but it cannot be done before 20 w of pregnancy, as
	this is the GA at which the lower uterine segment is formed
	 Uterine Repair (Hysterectomy)
	• Notes:
Мх	- Laparotomy, do a cs
	- Uterine Repair in ladies with low fertility
	- Ideal management: (hysterectomy), this decision is not easy but does
	not have to be delayed, due to risk of death for both
Complic-	 Maternal and fetal death

Post-Partum Hemorrhage (PPH)

	• Excessive	bleeding	g that make	s patient symptomatic: light-headedness,	
	vertigo, syncope, also results in signs of hypovolemia (hypotension,				
tachycardia, oliguria)					
Definition	Definition • Blood loss >500 ml after vaginal delivery or 1000 after CS				
	 HCT drop 	of 10% c	or need for l	blood transfusion	
	** ladies tolerate blood loss well, until there is significant blood loss. 1				
	L are lost b	efore hav	/ing tachyca	ardia, don't wait, act!	
	Class	loss	ss Lost% Physiologic response		
Classific-	1 1	000 cc	15	Dizziness, palpitations	
ation	2 1	500 cc	20-25	Tachycardia, tachypnea, sweat	
	3 2	000 cc	30-35	Significant tachycardia, hypotension	
	4 >	2500 сс	40	Shock, oliguria, anuria, air hunger	
Diagnosis	 Estimatio 	n of bloo	d loss: 1ml	of blood = 1 gram	
	- Visual und	derestima	ate by 33-50	0%	
	• Primary:	• Primary: PPH within 24 hr			
Primary	Secondar	y : more t	than 24 hr –	- 6 weeks (RCOG: up to 12 w):	
VS	- Mainly ca	used by i	ntra-uterine	e infection with pyrexia due to retained	
Secondary	tissues (end	tissues (endometritis) - (retained tissue by US!)			
	- Choriocar	cinoma:	noma: rare. If a patient comes 6 w after delivery		
	complainin	complaining of abnormal bleeding you will do b-HCG for her.			
		• <u>Ante</u>	partum:	• Intrapartum:	
Causes		- previ	ous PPH	 Prolonged labor >12 hr 	
	Tone	- place	nta previa	 Prolonged 3rd labor stage 	
	70-90%!	- mate	rnal obesity	 - Sepsis 	
PPH is not a		- baby	- baby >4kg		
Dx		- multi	- multiple pregnancy		
		- IOL			
		• <u>Ante</u>	partum:	• Intrapartum:	
		- PET		- Placental abruption	
Main	T hrombin	- Sepsi	S	- Sepsis	
Causes		- Antic	oagulants		
(4 T's)		- Inher	ited bleedir	ng	
	Trauma	- Uteri	ne/cervical/	/vaginal injury (instrumental, CS)	
	Tissue	- retair	ned product	s (placenta, membranes)	

• Multidisciplinary effort

Call for HELP, Check ABCs, vital signs, insertion of 2 large bore IV lines for IV fluids, send CBC, KFT ,clotting profile, prepare cross match 4-6 units of blood, insert a catheter, then check the uterus, is it atonic, if the placenta is out, check if it is complete, check for vaginal or cervical tears *How to check for coagulation before lab results are back*? Check the blood on the floor, there should be clotting immediately, if there is no clotting, think of coagulation defects

- Medications:
- o Uterotonics, stimulate the contraction of uterus
- Syntocinon (oxytocin), used commonly
- Methergine (Methylergometrine)
- Syntometrin (Oxytocin/ergometrine)
- **Haemabate** contains PG F2 α , given IM or intra-myometrial, it is not licensed to be given intra-myometrial though
- Misoprostol

Мx

- Examine the uterus to R/O atony
- Examine vagina, cervix to R/O lacerations (& repair if found)
- Explore the uterus to R/O retained placenta
- Manual Uterine Massage
- Removal of retained placental tissue: can occur in vaginal, CS
- Packing the uterus (to compress bleeding areas):

- **Ballooning**: inflating a balloon inside the uterus to cause compression, and filling it with 500 ml saline, using oxytocin to keep the uterus well contracting. After 24-36 hour, the balloon is deflated gradually until removed completely

• Trying off bleeding vessels

• **B-lynch suture:** Applied if we do a laparotomy, done to compress the uterus

- Internal iliac artery ligation
- Hysterectomy: it is a Definitive Mx (don't delay)
- Consideration the use of tranexamic acid: it is an antifibrinolytic, used in cs or PPH as injections to \downarrow blood loss
- Arterial Embolization: Only done in <u>stable</u> patients. call the *interventional radiologist*.

	Recombinant Activated Factor VIIa (Novoseven):
	- Enhances platelet aggregation
	- Promotes clotting by extrinsic pathway (binds to tissue factor)
	Complexes with TF activates Factor IX,X & generates thrombin
	- Controls bleeding rapidly –10 minutes!
	- Adverse effects < 1%
	- Short ½ life (2 hours)
	- High cost
	• Sheehan's syndrome : Pituitary ischemic injury (necrosis of the
	anterior lobe of the pituitary gland): clinically presents as difficult
	lactation or agalactorrhea
Complica-	Postpartum infection
tions	• DIC
	• Anemia
	• Transfusion hepatitis
	Asherman's syndrome
Document	- Documentation of PPH delivery is essential (medicolegal issue)
	Debriefing is at later time. Early the patient is only told that we are
Debriefing	dealing with a bleeding, and later debrief the patient, her family and the
	team themselves, bcz this is traumatic event
	Classic thinking:
	 Resuscitation using crystalloid and PRBCs
	 FFP, cryo, and plts only if hematologic parameters are abn (plts<50K;
	FBG<100K; PT/aPTT<1.5XNL)
Blood	
Transfer	FAILED TO PREVENT COAGULOPATHY IN MASSIVE HEMORRHAGE –
	<u>DILUTIONAL COAGULOPATHY</u>
	New Concept:
	- Limit early aggressive crystalloid use
	- Early admin. of FFP and PLTs (with PRBCs) ratio 1:1:1
	- Early use of fFVIIa

Stages	Pathophysio	Clinical	Lab tests
1: Hypercoagulable	Activation of clotting	Hyper-	- \downarrow clotting,
	factors & development of microthrombi	coagulation	- \uparrow platelet adherence
2:	Λ consumption of	Bleeding	- 个 clotting,
Consumptive	platelets & clotting factors		- \downarrow platelets,
Hypercoagulable			- greatly ↓ fibrinogen
3:	Substantial formation of	Marked	- 个 thrombin time
Secondary Fibrinolytic	fibrin degradation products and plasmin	bleeding	- ↓ clot lysis time
			- \uparrow fibrin degradation
			products

Туре	Contents	Indication	Volume	Shelf life	Effect
PRBCs	RBC's, WBC's,	Anemia	300	42 d	个 Hb 1g
	Plasma				
Platelets	Platelets, plasma	Bleeding due to low	50	5 d	个 Plt count
		plt			7500/unit
FFP	FBG, plasma,	DIC, coagulation	250	12 m frozen	个 FBG 10-15
	clotting factors	disorder, reverse		2 hr thawed	
		warfarin			
Cryoppt	FBG, factor VIII,	DIC, von Willebrands,	40	4-6 h thawed	个 FBG 10-15
	vWf, XIII	Hemophilia A			



Uterine Rupture

Definition	Full thickness loss of the uterine musculature through all of its layers		
	Previous uterine scars (dehiscence of a CS scar is the mcc)		
	- Classical C/S has x20 risk than LUS C/S		
	Trauma (External/Obstetric)		
RF	• Excessive use of oxytocin		
	 Uterus Distension (Polyhydramnios, Multiple Gestations) 		
	Placenta Percreta		
	Diagnostic Criteria:		
- Painful bleeding (tearing – not relived by analgesia)			
	- Loss of FHR		
	 Loss of station (fetal head) 		
C/P	- Inability to identify uterine contrac	ctions	
	• Other S/Sx:		
	 Maternal/Fetal distress 		
	- LUS is stretched and painful to tou	ch	
	- Prolonged fetal bradycardia is the	1 st sign mostly	
	 Bandl's ring: abnormal junction be 	etween the two segments	
	 Call for help, resuscitation (blood transfusion) 		
	 Good control: use of oxytocin, sto 	p bleeding (ligation)	
Mx	 Immediate laparotomy and delive 	ry of the fetus	
	 Broad spectrum antibiotics Hysterectomy v.s uterine repair 		
	• if the rupture is local, clean edges	not edematous	
	• no infection, good general condition	on	
When to repair	 desire for future childbearing 		
((low transverse		
the extent of the	No extension to surrounding area		
iniury"	No evidence of coagulation consequences		
	 if the patient undergoes a repair of the uterus, all subsequent pregnancies will be delivered by C/S birth at 36 week 		
<u> </u>			
Complications	Maternal	Fetal	
	Hemorrhage, Shock, DIC, Death	Hypoxia , Anoxia, Asphyxia	
	Amniotic fluid embolism (AFE)	Neurological sequalae, Death	
	Bladder laceration, Hysterectomy		

Retained Placenta

Definition	Lack of placental expulsion within 30 min of delivery of an infant, this period can extend to 90-120 min for births in 2 nd /3 rd stages if labor managed without oxytocin			
Types	 A. Trapped or incarcerated placenta (separated/detached but not delivered) B. Placenta Adherens: the placenta is adherent but easily separated C. Placenta Accreta Spectrum: pathological invading of myometrium - Accreta should be diagnosed antenatally 			
Phases in the	 Latent > Contraction > Detachment > Expulsion 			
3 rd stage				
RF	 Previous Hx Preterm gestational age (strongest factor) Use of ergotamine (Trapped placenta) Uterine Abnormalities PET, stillbirth, small for gestational age (defective implantation) Velamentous cord insertion (risk for manual removal) Maternal age > 30 year Delivery in teaching hospital 			
С/Р	 Placental separation signs present: Lengthening of the umbilical cord Gush of blood from the vagina Change in the shape of the uterine fundus from discoid to globular Elevation of palpable through a small but patent cervical os 			
Dx	 US Dx of placenta adherens or accreta: made by the absence of the S/Sx of the placental separation, also after separation. Adeherens leaves clean separation signs, in the Accreta no clean signs (due to invasion) 			
Complications	 PPH, Postpartum endometritis Uterine inversion Death (very rare) 			
Mx	 Retained placenta should be manually removed ASAP, globan uterine contractions (reduction of bleeding) In the absence of heavy bleeding, we suggest intervention when the third-trimester placenta has been retained for 30 to 60 minutes rather than expectant management or earlier intervention 			

	 Gentle cord traction is the initial maneuver. If unsuccessful and the lower uterus/cervix is constricted, we administer <u>nitroglycerin</u> to release the constriction. If the uterus is atonic, we administer an <u>oxytocin</u> infusion to promote uterine contraction. If these measures fail to result in placental expulsion, we suggest manual rather than instrumental extraction of the placenta administering a single dose of a broad spectrum prophylactic antibiotic before manual extraction of the placenta For women with a second-trimester birth and no significant bleeding, the time period before manual extraction can be extended as the frequency of retained placenta is higher and the risk of hemorrhage is lower. We suggest not waiting > 2 hour due to the risk of infection For women with a small area of placenta accreta, we slowly create a plane of separation at the maternal-placental interface using finger dissection. Curettage is a second-line option if finger dissection is unsuccessful
Complicated	Instrument extraction
Cases	Incomplete extraction
	Unexpected placenta accrete spectrum
Recurrence	• 6-12% recurrence risk
	• 1/% absolute risk of recurrent manual removal of the placenta

Uterine Inversion

Definition	Descent of uterine fundus into the cavity, through the cervix or vulva
	 almost all cases are in the 3rd stage (puerperal uterine inversion
Info	 life-threatening (due to shock & hemorrhage)
	• Frequently followed by endometritis
	Traction may elicit vasovagal response
Types	Complete: Incomplete, complete (through the cervix)
	• 1 st : the uterus is partially turned out
Degrees	• 2 nd : fundus has passed through the cervix but not outside the vagina
J. J	• 3 rd : the fundus prolapsed outside the vagina
	• 4 th : the uterus, cervix and vagina are out and visible
	• Acute: 1 st 24 hr before the cervix constricts
Onset	 Subacute: within 4 weeks of delivery
	• Chronic: after 4 weeks of delivery
	Oxytocin upon delivery of the anterior shoulder
Active Mx of	 Controlled cord traction
the 3 rd stage	** never pull on the cord to deliver the placenta. Gentle traction will be
	sufficient in a normally implanted placenta
	 Brandt-Andrews maneuver
	• Previous uterine inversion (the most common risk factor!)
	 Maternal: Uterine anomaly, connective tissue disease (Marfan's)
	 Placental: Fundal placenta, abnormal adherence, placenta previa
RF	 Short umbilical cord
	Macrosomia
	Uterine Atony
	 latrogenic: Antepartum tocolysis (MgSO4) mis-Mx in 3rd stage
	Vaginal bleeding
	 Lower abdominal pain
	 Sensation of vaginal fullness with a desire to bear down after delivery
C/P	of placenta
	 S/Sx of hypovolemic shock
	 Abdominal Exam: Cupping of the fundus, absence of the uterus
	 Vaginal exam: soft purple (dark bluish red mass in the vagina/vulva)
	• PPH, based on the degree of blood loss the C/P is more prominent
Dx	• C/P (bimanual exam)
	• US to confirm

	• Death		
	• Anemia (mc!)		
	 Hypotension and Hypovolemic shock 		
Complications	- the most important complication is shock wither hemorrhagic or not		
	Renal failure		
	 Sheehan's syndrome 		
	 Risk of blood transfusion 		
	 Surgery complications and Sepsis 		
	1) Call for help (resuscitate, evaluate, identify cause, surgery if needed		
	2) Repositioning:		
	a) Tocolytics to relax the uterus (nitroglycerin, terbutaline or MgSO4)		
	b) Try manually to return it (Johnson method)		
	c) if failed: try hydrostatic correction		
	- complications: infection, failure or saline embolus		
	d) if failed: laparotomy: pull the fundus up and that's it you don't need		
Mx	to fix it in place and after you put it in consider oxytocin agent to		
contract the uterus well and avoid PPH			
	• Post procedure care:		
	- Oxytocin infusion, if still failed to contract give ergometrine or		
	prostaglandins		
	- Give prophylactic antibiotic: ampicillin/cefazolin + metronidazole. if		
	there is signs of infection add gentamicin + analgesics		
	• consider uterine inversion in both vaginal and C/S delivery:		
	- why C/S: once the baby is out you can go and pull the placenta		
	immediately (that's wrong)		
	Prompt recognition of uterine inversion		
	(consider accreta)		
	-Initiate uterotonics		
	-Attempt placental removal -Consider activating transfusion protocol depending on estimated -Consider activating transfusion -Leave placenta in place if not already removed -Immediate attempt at manual repositioning (Johnson maneuver)		



Breech Presentation

Info	 most common type of malpresentations is breech 				
	• 25% of breech fetuses becomes vertex at a later time of gestation				
	Prematurity (most important, most common)				
	 Uterine anomalies (bicornuate) 				
	Multiple Gestation				
RF	 Extended legs preventing spontaneous version 				
	Placenta previa				
	Hydramnios				
	 Contracted maternal pelvis 				
	 Pelvic tumors that obstruct the birth canal 				
Types of breech					
	Complete Breech Footling (Incomplete) Frank Breech				
	Complete Breech Footling (Incomplete) Frank Breech Breech				
	Complete Breech Footling (Incomplete) Frank Breech Breech ** Footling is more common in multiparous due to laxity of abdomen				
Dx	Complete Breech Footling (Incomplete) Frank Breech ** Footling is more common in multiparous due to laxity of abdomen • By Leopold's maneuvers and vaginal exam				
Dx	Complete Breech Footling (Incomplete) Frank Breech ** Footling is more common in multiparous due to laxity of abdomen • By Leopold's maneuvers and vaginal exam • External Cephalic Version (ECV): immediate FCV 25, 75% success rate				
Dx	Complete Breech Footling (Incomplete) Frank Breech ** Footling is more common in multiparous due to laxity of abdomen • By Leopold's maneuvers and vaginal exam • External Cephalic Version (ECV): - immediate ECV 35-75% success rate				
Dx	Complete Breech Footling (Incomplete) Frank Breech ** Footling is more common in multiparous due to laxity of abdomen • By Leopold's maneuvers and vaginal exam • External Cephalic Version (ECV): - immediate ECV 35-75% success rate - It reduces the CS rate				
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	 Vaginal Delivery: <u>Criteria for VD in breech</u>:
	1. Frank or complete breech
	2. Term baby (>37w)
	3. Weight 2.5 – 3.8 K
	4. Fetal head must be flexed
	5. Normal maternal pelvis dimension
	6. No other C/S indication present
	 Assisted breech delivery (piper forceps – fully dilated)
	• Cesarean Delivery:
	 Nearly all breeches are delivered by C/S now to avoid complications
	 Less perinatal mortality and morbidity than vaginal delivery
	- C/S Indications:
	1. Hyperextension of the head
	2. Abnormal pelvic bone
	3. Failure of 1 st /2 nd stage of labor
	4. Previous C/S
	5. Previous difficult labor
	Complications: IUGR, HTN, DM, Hypoxia, Asphyxia
	7. Premature baby (Risk of head entrapment)
	 Breech & Transverse lie if there is ROM, your main concern must be
	cord prolapse
	• Twin delivery:
Notes	- both breech: perform C/S
	- 1 st vertex, 2 nd breech: the 1 st is delivered vaginally then the 2 nd might
	turn, if not use breech extractor (grabbing the 2 nd baby feet and pulling
	him into the birth canal to help deliver him vaginally if failed then C/S)
	- 1 st breech, 2 nd vertex: locked twins – C/S immediately

Shoulder Dystocia



Definition	• Arrest of normal labor after delivery of the head by impaction of the				
	anterior shoulder against the symphysis pubis				
Info	Obs emergency, unpre-	edictable, unpreventable	2		
	• Very rare (0.6 - 0.7%)				
RF	Maternal Fetal Intrapartum				
" DOPER "	 Abnormal pelvic 	Male Gender	 Prolonged active 1st 		
	anatomy		stage of labor		
DM	 Short stature 	 Macrosomia: RF: 	 Prolonged 2nd stage 		
Obesity	 Multiparity 	- Gestational DM	(labor dystocia)		
Duclose and successory (2ry arrest of cervical 	- Maternal obesity	 Instrumental 		
Prolonged 2 nd stage of	dilation	- Hx of macrosomia	delivery		
labor	 Excessive pregnancy 	- Post-date gestations	• IOL		
Expected big baby	weight gain	- Advanced maternal	 Use of oxytocin 		
	 Vaginal delivery 	age			
Previous shoulder	after longer gestation				
uystocia	(40-41 weeks)				
Characteristics	Additional maneuvers required to deliver the fetus				
	 Head-to-body delivery time > 60 seconds 				
When to expect	• Failure of rotation • Difficulty of delivery of face and chin				
the dystocia	 Turtle sign Failure of delivery of the anterior shoulder after traction 				
	• Brachial plexus damage	ge (Erb's palsy – only 15%	6 are permanent)		
	Umbilical cord entrap	ment			
Complications	 Inability of child's chest to expand properly 				
	• Severe brain damage or death due to hypoxia or acidosis (Asphyxia)				
	• Pneumothorax				
	• Birth trauma				

Call for help

 70% will deliver by Mcrobert (Fixation & abduction of the maternal leg) + Suprapubic pressure

• Patient head should be down

- Why we do the previous 2 points?

- a) increase anterio-posterior diameter
- b) Flattening of the sacrum
- both a/b give more space

the mother is told not to push
 excessive neck rotation, excessive
 downward traction on head an fundal
 pressure should be avoided
 distended bladder, should be drained

Episiotomy:

- We do it to increase space, not to

Mx deliver because he is stuck behind the bone, it might cause trauma

 McRoberts Maneuver: (1 in the pic)

 it needs 2 assistants grasping the maternal legs flexing the thighs against the abdomen; resulting in a cephalad rotation of the symphysis pubis and a flattening of the sacral promontory. It Can relieve 40-50% of shoulder dystocia (especially when combined with suprapubic pressure)

Suprapubic pressure (Rubin I – 2nd pic):
 hand should be placed on top of the mother's abdomen over the fetal anterior shoulder, so that the shoulder will adduct and pass under the symphysis (less space between the shoulders), the pressure is not continuous just like the CPR





•	Enter ((Internal	Rotation –	Rubin II):
---	---------	-----------	------------	------------

- the anterior shoulder is impacted against symphysis pubic
- go to the posterior aspect of the anterior shoulder and try to push it out
- if failed then go to the anterior aspect
- if failed go for the posterior shoulder
- posterior arm try to pull it out
- if still failed: do roll on all four/clavicle fracture/symphysiotomy

• Other Methods:

- Reverse wood's screw
- Reverse wood's screw maneuver
- Jacquemier's maneuver (Barnum's maneuver removal of the posterior arm)
- Rolling the patient (All-fours or gaskin maneuver)
- Zavanelli's maneuver
- Intentional fetal clavicular fracture
- Maternal symphysiotomy

Case: a lady just had instrumental delivery with forceps, the head is coming out with difficulty, but the delivery is completed: **what you will worry about?**

Maternal	Fetal	
 PPH: due to atony, trauma, prolonged 2nd 	 Hypoxia/Asphyxia 	
stage of labor	 Brachial plexus injury (Erb's palsy) 	
Uterine rupture	• Death	
 Vaginal/Cervical tear 		

Cord Prolapse

	Descent of the umbilical cord into the LUS where it lie adjacent to the		
Definition	presenting part or below it with <i>rupture fetal membranes</i> , if the		
	membranes are intact it is cord presentation (here do elective C/S)		
	• rare, most commonly associated in transverse lie (20%), footling		
Info	(15%) <i>,</i> compl	ete breech (5%)	
	ncidence because it can be detected only by fetal HR		
changes characteristic of cord compression			
	• Occult (hidd	den) cord prolapse:	
 cord adjacent to presenting part 			
	- not felt on e	exam, might lead to unexplained distress	
Types	• Funic (cord)	prolapse:	
	- cord below presenting part, with no rupture of fetal membranes		
	- palpated		
	 Overt cord 	prolapse:	
	- below + rup	ture of membranes and displacement through the vagina	
	Fetal	Congenital/fetal anomaly, prematurity/IUGR, multiple	
		pregnancy, abnormal lies, malpresentation (footling)	
RF	Maternal	ROM (Spontaneous/ARM), Pelvic tumors (fibroids), Pelvic	
		contraction, PTL	
	Placental	Polyhydramnios, Minor degree of placenta previa	
	Procedure	ARM, ECV, IOL, Applying fetal scalp electrode, amnion	
		infusion, placement of cervical ripening balloon catheter	
	 Umbilical ar 	rtery vasospasm	
Risks	 Birth Asphy 	vxia	
	 Hypoxic iscl 	hemic encephalopathy	
	 Perinatal de 	eath	
	 Mostly clini 	cal	
	 CTG: abnormal FHR, marked carriable decelerations 		
	• Vaginal exam:		
Dx	 sudden appearance of a loop of umbilical cord after rupture 		
	- you can palpate it in absence of membranes		
	- you can pal	bate it in absence of membranes	
	 you can palp or cord pres 	sentation if membranes are intact	

• CORD:			
Consider cord prolapse			
Organize for help			
Release pressure by 4 maneuvers			
Decrease manipulation			
 Immediate vaginal exam to R/O cord prolapse 			
 if cord presentation: 			
- Term: C/S before membrane rupture			
- Pre-term: no consensus on Mx (monitoring), left lateral position			
 Mx is based on the type: for occult we monitor, for overt we inte 	rfere		
• Prevent/relieve cord compression and vasospasm by:			
- Manual placement (elevation)			
- Bladder filling	-		
- Adjust maternal position:	IN		
a) knee chest position (Genuperctoral)			
b) Sim's lateral position,			
c) Trendelenburg position	APP-		
Fetal Assessment Viable or not:	-		
- Category 1 (cord prolapse + abnormal HR)	_		
- Category 2 (cord prolapse + normal HR)			
Prompt delivery:			
- Cervix fully dilated, tend to do vaginal birth	REST4MED		
within 20 min of Dx (instruments might be used)			
- if not dilated, do a C/S within 30 min of Dx			
- Don't forget to drain the bladder, check for HR, call a neonatologi	st		
due to risk of needing resuscitation (based on APGAR score)			
Admission: if abnormal lie/non-cephalic or PROM/PTL at 37 weel	<		
• Labor/Ruptured membranes of an abnormal lie are indications for	• Labor/Ruptured membranes of an abnormal lie are indications for CS		
Prevention • Fetal distress is associated with cord prolapse so do a vaginal exa	Fetal distress is associated with cord prolanse so do a vaginal exam		
• Artificial runture of membranes should be avoided (if it's a must	Artificial rupture of membranes should be avoided (if it's a must then		
to it in the OT if you needed to do immediate C/S)	inen		
Oblique lie transverse lie unstable lie: we admit them at 37 wee	k		
Notes and we monitor them			
PROM & non-cenhalic: we manage them as an innatient			
Case O: Patient came to hospital with cord prolanse 1 st thing to do is?			
- Check for EHR if ILIED then no need for emergent C/S			
- Check for EHR if ILIED then no need for emergent C/S			

Amniotic Fluid Embolism (AFE)

Definition	 Sudden cardiorespiratory collapse and DIC
	 Similar DDx to shock
Info	 Quickly R/O hemorrhage
	 Incidence: 2-6 : 100,000 deliveries
	• Multiparity (Gravida >=5)
	 Advanced maternal age
	Male fetus
	• Trauma
RF	 CS, Operative vaginal delivery, D/C, IOL, Recent amniocentesis
	Placental problems: Placental Abruptio, Placenta Previa
	Polyhydramnios
	• PET
	• Short labor
	Uterine rupture
	Cervical laceration
	• Triad: Hypotension, Hypoxia, Coagulopathy
	- Sudden cardiorespiratory arrest or both hypotension
	- Respiratory compromise (dyspnea, O2 sat <90%)
	- Documentation of over DIC (Coagulopathy)
	• Dyspnea, Agitation
C/P	• Onset: during labor or within 30 min of the delivery of the placenta
	• Absence fever (>38.0 C) during labor
	• Physical signs:
	- Hypotension dyspnea seizure cough cyanosis fetal bradycardia
	nulmonary edema cardiac arrest uterine atony coagulonathy (severe
	hemorrhage) altered mental status
	• Pulmonary edema
Complications	• Left heart failure
	• DIC. Shock
	Cogguiation (DIC): Elevated D dimer low fibringen
	thrombocytopenia
Investigations	• CBC
investigations	ABC: hypoxemia metabolic acidosis rarely hypercappia
	• CXR: nulmonary edema, effusion or enlarged heart
	- CAN. pullional y euclia, enusion or eniargeu fiedri

	• ECG: tachycardia or arrythmias
	 Echo: rise in pulmonary pressure or left ventricular failure
	 FHR if before delivery: absent baseline FHR variability, late
	decelerations or terminal bradycardia
	• The principal objectives of treatment for amniotic fluid embolism are
	to support the respiratory system, correct the shock, and replace the
	coagulation factors.
	• <u>Unstable patients</u> :
Мх	- Call for HELP
	 Manual displacement of the uterus to the left ABCs
	- Respiratory/Hemodynamic support: Intubate, O2, IV line + fluids, if
	still hypotensive consider vasopressor therapy (norepinephrine)
	- Control hemorrhage and coagulopathy: rapid transfusion required
	- R/O other causes and deliver the fetus if still alive
	Urgent delivery includes
	1) Category 3 EHR tracing (preterminal) in a fetus at or above the limit
Delivery of the	of viability
Fetus	2) Rapid and progressive deterioration of the mother's condition
	 Perimortem C/S (Resuscitative hysterotomy):
	- Done at 4 min and complete delivery by 5 min following cardiac arrest
	• 10% of al maternal death (mortality rate in AFE 20-80%
	• Hypoxemia causes 50% of deaths in 1 st hour
Prognosis	• Those who survive have a poor outcome with 85% suffering
U	neurological injury due to cerebral hypoxia
	• if AFE occurs prior to delivery: neonatal outcome is poor (mortality
	rate 20-60% & only up to 50% of the survivors are neurologically intact)
	• Eclampsia
	Anaphylactic Shock
	Septic Shock
AFE DDx	Pulmonary Embolism
	Drug toxicity
	• APH, PPH
	Aortic Dissection

Maternal Collapse

Defined	as an acute event involving the cardiorespiratory systems and/or brain resulting in a reduced or absent conscious level (even death), at any stage in pregnancy & up to six weeks after delivery 1) Vasovagal attack (1 of the mc)		
	2) Post-ictal state foll	owing an epileptic seizure (1 of the mc)	
	3) Hemorrhage/Hypo	volemia (APH, PPH, ICH)	
	4) Eclampsia and PET	(Eclampsia seizure is self-resolving in minutes)	
	5) Sensis	(8	
	6) Cardiac causos (ao	rtic discostion cordiamy anothy MI)	
	b) Carulac Causes (ab	ruc dissection, cardiomyopathy, ivity	
	7) The 4 T's and 4H's		
	Reversible cause	Cause in pregnancy	
	4H's Hypovolaemia	Bleeding (obstetric/other; may be concealed) or relative hypovolaemia of dense spinal block, septic or neurogenic block	
	Hypoxia	Pregnant women can become hypoxic more quickly.	
Causes		Cardiac events – peripartum cardiomyopathy, myocardial infarction, aortic dissection, large vessel aneurysms	
	Hypo/hyperkalaemia and	Hypo and hyperkalaemia are no more likely. Hyponatraemia may be cause	
	i i porti per taracina ana		
	Hyponatraemia	by oxytocin use	
	Hyponatraemia Hypothermia	by oxytocin use No more likely	
	Hyponatraemia Hypothermia 4T's Thromboembolism	by oxytocin use No more likely Amniotic fluid embolus, pulmonary embolus, air embolus, myocardial infarction	
	Hyponatraemia Hypothermia 4T's Thromboembolism Toxicity	by oxytocin use No more likely Amniotic fluid embolus, pulmonary embolus, air embolus, myocardial infarction Local anaesthetic, magnesium, other	
	Hyponatraemia Hypothermia 4T's Thromboembolism Toxicity Tension pneumothorax	by oxytocin use No more likely Amniotic fluid embolus, pulmonary embolus, air embolus, myocardial infarction Local anaesthetic, magnesium, other Following trauma/suicide attempts	
	4T's Thromboembolism Toxicity Tension pneumothorax Tamponade	by oxytocin use No more likely Amniotic fluid embolus, pulmonary embolus, air embolus, myocardial infarction Local anaesthetic, magnesium, other Following trauma/suicide attempts Following trauma/suicide attempts	

threatening airway &/or breathing or circulation problems

- skin or mucosal changes

- we are facing more cardiac cases due to increased age so there is IHD pregnant lady & correction of congenital cardiac diseases. Those patients need certain care during pregnancy

- IV lidocaine or regional anesthesia on high level or large doses regionally given and increase systemic absorption is one of the causes of heart failure

	 mcc of direct maternal death 	
Thromboembol	 use thromboprophylaxis 	
ism	 Amniotic fluid C/P: Acute hypotension, respiratory distress and ac hypoxia 	
	hypoxia	
Sepsis	• Bacteremia	
	 mc organisms: GAS, GBS, GDS, Pneumococcus, E. coli 	
Drug toxicity	 Magnesium sulphate in the presence of renal impairment 	
	Local anesthetic agents	
	 ABCDE approach: Check for breathing, pulse, start CPR immediately 	
	 Relieving aorto-caval compression: 	
	 done in women above 20 week GA (uterus above umbilicus) 	
	 At what GA it become a challenge? 20 weeks (significant cut off) 	
	 Placing it left lateral 30 degree positioning 	
	 Placing Oxygen 	
	• Two wide-bore cannula	
	 Fluids (volume replacement) 	
	• US	
	 If all that didn't work go for perimortem C/S: 	
	- Perimortem C/S (Resuscitative Hysterotomy) Definition: is	
	a hysterotomy performed to resuscitate a woman in middle to	
	late pregnancy who has entered cardiac arrest. Combined with	
Mx	a laparotomy, the procedure results in a Caesarean section that	
	removes the fetus, thereby abolishing the aortocaval	
	compression caused by the pregnant uterus. This improves the	
	mother's chances of return of spontaneous circulation, and may	
	potentially also deliver a viable neonate	
	 we use midline/classical incision because it gives more rapid access 	
	 4 minute rule (resuscitate for 4 minutes if still failed do C/S) 	
	 perimortem C/S is mainly to save the mother than the fetus, the 	
	gravid uterus impairs venous return and thus reduces cardiac output by	
	approximately 60% secondary to aortocaval compression !	
	- <u>Delivery of the fetus and placenta (Patient response after)</u> :	
	a) reduces oxygen consumption	
	b) improve VR & CO	
	c) Facilitates chest compressions	
	d) make ventilation easier	
	e) allows for internal chest compressions	

 <u>PMCS</u>: you only need a scalpel + 2 clamps for the umbilical cord (if only scalpel is found then cut the umbilical cord and manually compress until a clamp is found) in the OR, no circulation, minimal blood loss and no anesthesia required

System	Changes in pregnancy	Impact on resuscitation
Cardiovascular sy	/stem	
Plasma Volume Increased by up to 50% Diludonal anaemia		Dilutional anaemia
		Reduced oxygen carrying capacity
Heart rate	Increased by 15-20 bpm	Increased CPR circulation demands
Cardiac output	Increased by 40%	Increased CPR circulation demands
-	Significantly reduced by pressure	
	of gravid uterus on IVC	
Uterine blood	10% of cardiac output at term	Potential for rapid massive haemorrhage
flow		
Systemic	Decreased	Sequesters blood during CPR
vascular		
resistance		
Arterial blood	Decreased by 10-15 mmHg	Decreased reserve
pressure		
Venous return	Decreased by pressure of gravid	Increased CPR circulation demands
	uterus on IVC	Decreased reserve
Respiratory syste	m	
Respiratory rate	Increased	Decreased buffering capacity, acidosis more likely
Oxygen	Increased by 20%	Hypoxia develops more quickly
consumption		
Residual	Decreased by 25%	Hypoxia develops more quickly when apnoeic
capacity		
Arterial pCO2	Decreased	Decreased buffering capacity, acidosis more likely
Laryngeal	Increased	Difficult intubation
oedema		
Other changes		
Gastric motility	Decreased	Increased risk of aspiration
Lower	Relaxed	Increased risk of aspiration
oesophageal		
sphincter		
Uterus	Enlarged	Diaphragmatic splinting reduces residual capacity and makes ventilation more difficult
		Aortocaval compression causes supine hypotension,
		reduces venous return and significantly impairs CPR
Weight	Increases	Large breasts may interfere with intubation, makes
		ventilation more difficult

Physiological changes affecting resuscitation

• Difficult to intubate, why?

- 1) Laryngeal edema,
- 2) Weight gain,
- 3) Increase breast size

• Tolerance to hypoxia in pregnancy is reduces so she will collapse at the same O2 concentration

• *Causes of aspiration*: delay gastric emptying, relax sphincter

• \uparrow Uric acid indicate worse fetal and maternal outcome it will elevate before other markers so it will give you an alarm that this patient with chronic HTN will have severe PET

Gestational Trophoblastic Disease (GTD – Moral Pregnancy)

Types	 o Pre-malignant: Hydatidiform mole (complete/partial) o GTN – malignant GTD: persistent/invasive GTN Choriocarcinoma Placental site trophoblastic tumors (rare) 			
Hydatiform mole				
Definition	 abnormal pregnancy by varying degrees of trophoblastic proliferation and vesicular swelling of villi + absent or an abnormal fetus/embryo 			
RF	 Asian Age extremes Previous molar COCPs use in ↑ β-HCG: ↑ malignant transformation Familial/sporadic clusters of CHM (AR): chromosomes 			
Clinical S/Sx	 Vaginal bleeding in early pregnancy (common) excessive uterine size (50%) Theca lutein cysts (50%) Hyperemesis gravidum (25%) early PET (25%) Hyperthyroidism (<10%) Pulmonary emboli (respiratory distress - <2%) Vaginal hydropic vesicles passage 			
Postpartum non-molar	 GTN can occur after non-molar pregnancies AUB after non-molar: do a pregnancy test to R/O GTN GTN considered in women having acute respiratory, abdominal, neurological Sx after any pregnancy 			

DE	 enlarged uterus 		
PE	• bilateral ovarian cysts		
	vaginal METS 30% (vascularized & infection prone)		
	 β-HCG (>100K in CHM): mostly associated with: 		
	 Ovarian enlargement due to theca lutein cysts 		
	- Hyperemesis gravidarum		
	- Early PET (<20w)		
	- Hyperthyrodism		
Investigate	 CBC, LFT, KFT, Thyroid FT 		
	 Blood type, antibody screening, confirm Rh(D) state 		
	• CXR		
	• Pelvic US		
	 Histopathology analysis 		
	• Registration of confirm molar (for β -HCG surveillance)		
	By histological examination, enhanced by flow		
Dx	cytometry to determine karyotype		
	• all POC after miscarriages be sent for analysis to R/O		
	Surgical evacuation: suction curettage		
	 Medical termination (we try to avoid it: RISKY) 		
	 Anti-D: at time of surgical evacuation 		
Treatment	 Hysterectomy: rarely (elective/ER: life-threatening) 		
	 2nd Uterine Evacuation: rarely, in: 		
	- plateauing or raising β-HCG (1500)		
	 abnormal intra-uterine tissue on US 		
	 heavy vaginal bleeding 		
	 monitor weekly β-HCG until 3 normal values in row 		
	• 50% reach normal β -HCG 6-14 w after evacuation		
	 rise/plateau of β-HCG indicate chemo treatment 		
Follow up	 Register with specialist center to check β-HCG 		
	• 2 weekly serum & urine samples until β-HCG is normal		
	 Avoid pregnancy (Avoid for 6 month) 		
	• use non-hormonal barrier contraceptives / No COCPs		
	 check for B-HCG after every future pregnancy delivery 		

	Complete (CHM)	Partial (PHM)		
	Monospermic or dispermic	Dispermic fertilization of		
Origin	fertilization of empty ovum	ovum, often misdiagnosed		
U	. ,	as incomplete or missed		
S/Sx	More frequent	Less frequent		
Karyotype	Diploid	Triploid 90%:		
	(paternal: 46 XX, 46 XY)	69 XXY, 69 XYY, 69 XXX		
Prevalence	1/1000	3/1000		
Fetal tissue	Absent	+ (abnormal – high IUFD)		
Histopath-	Diffused, the cystic villi	Focal		
ology	show (cluster of grapes)			
P57	not expressed	expressed		
FISH	Diploid	Triploid		
GTN Risk	15%	0.5%		
	 Embryo absence 	 Fetus + (maybe viable) 		
	 No amniotic fluid 	 Amniotic fluid + 		
US	 Central heterogeneous 	 Focal anechoic spaces 		
features	mass with anechoic spaces	(Swiss cheese pattern)		
	(diffused: snowstorm)	• \uparrow transverse diameter of		
	 Theca lutein cysts 	gestational sac		
		 Absent theca lutein cysts 		
Persistent	tent 15-20% 3-5%			
GTD risk	GTD risk			
	GTN / Persistent	GTD		
	 90% of GTN cases are personal 	sistent		
Persistent	 MC Sx is vaginal bleeding 			
	 Uterine rupture is rare 			
	 15% localized (invasive GTN) & 4% METS (chorio-Ca) 			
	 enlarged irregular uterus 			
Suggestive	 bilateral ovarian enlargem 	ient		
S/Sx	• 个 β-HCG			
	• S/Sx of METS			
	• pre-evacuation β -HCG > 1	00К		
---	--	---		
	• pre-evacuation: uterine gr	rowth & theca lutein cysts		
	• Age > 40			
RF	 Recurrent molar 			
	 Aneuploidy mole 			
	Molar medical complication	ons (PET, hyperthyroidism)		
	Evidence of distant tropho	oblastic embolization		
	• plateau in β-HCG for atlea	ast 4 values over 3 weeks		
Dx of	• β-HCG rises (by 10% for 3	values over 2 weeks)		
persistent	 Detectable β-HCG for > 6r 	nonths post-evacuation		
	Histology of Choriocarcine	oma		
	 Plateau or rising β-HCG 			
Chemo	 Heavy vaginal bleeding or 	GI/intraperitoneal hemorr.		
indications	 Choriocarcinoma Histolog 	У		
for GTD	 METS: brain, liver, GI, lung 	5		
	• β-HCG of 20K, 4 weeks po	st-evacuation		
	 β-HCG raised for 6 m post 	-evacuation (even if \downarrow)		
FIGO	I GTN confined to uterus			
anatomical	II GTN outside uterus but	limited to genital structures		
GTN	III GTN extends to lungs			
staging	IV GTN extends to all site (METS everywhere)			
	• 0-6 score: low risk of mon	o-chemotherapy resistance		
	 >7: high risk of mono-chei 	motherapy resistance		
	1 1 1			
FIGO	mono-chemo with metho	trexate (or dactinomycin)		
FIGO chemo	 mono-chemo with methor Low risk (95%) 	trexate (or dactinomycin) High risk		
FIGO chemo system	 mono-chemo with method Low risk (95%) stage I, maybe II-III 	trexate (or dactinomycin) High risk • Etoposide, methotrexate,		
FIGO chemo system	 mono-chemo with method Low risk (95%) stage I, maybe II-III Methotrexate + Calcium 	trexate (or dactinomycin) High risk • Etoposide, methotrexate, dactinomycin (EMA),		
FIGO chemo system	 mono-chemo with method Low risk (95%) stage I, maybe II-III Methotrexate + Calcium folinate (folic acid) 	trexate (or dactinomycin) High risk • Etoposide, methotrexate, dactinomycin (EMA), cyclophosphamide,		
FIGO chemo system	 mono-chemo with method Low risk (95%) stage I, maybe II-III Methotrexate + Calcium folinate (folic acid) Survival rate 100%! 	trexate (or dactinomycin) High risk • Etoposide, methotrexate, dactinomycin (EMA), cyclophosphamide, vincristine (CO)		
FIGO chemo system Response	 mono-chemo with method Low risk (95%) stage I, maybe II-III Methotrexate + Calcium folinate (folic acid) Survival rate 100%! monitored weekly 	trexate (or dactinomycin) High risk • Etoposide, methotrexate, dactinomycin (EMA), cyclophosphamide, vincristine (CO)		
FIGO chemo system Response Assessment	 mono-chemo with method Low risk (95%) stage I, maybe II-III Methotrexate + Calcium folinate (folic acid) Survival rate 100%! monitored weekly Remission: 3 consecutive reference in 2 1100 in 1000 in 1	trexate (or dactinomycin) High risk • Etoposide, methotrexate, dactinomycin (EMA), cyclophosphamide, vincristine (CO) normal β-HCG over 14-21 d		

	Inpatient		
	• measure β-HCG		
Principles	 Adequate response: 50% reduction in weekly β-HCG 		
of Chemo-	• Chemo is remained till β-HCG is normal and for further		
therapy	6 weeks to eliminate any residual cells		
	• low-risk GTN presents soon after CHM/PHM Dx, while		
	the high-risk GTN presents months or years		
	 S/Sx depend on site of METS 		
In Chemo	• High risk: ↑ 2 nd tumors, fastens menopause by 3 yrs		
long-term	• Low risk: no 2 nd tumors, fastens menopause by 1 year		
	 neither high/low affects fertility or congenital abnor. 		
Future Con-	• GTN requiring chemo should not conceive for 12 m		
traception	 GTD not requiring chemo (6 m after β-HCG is normal) 		
	Choriocarcinoma		
	 Highly malignant, β-HCG secreting 		
	 most aggressive GTN 		
	Macroscopically:		
Info	 soft, purple, large, hemorrhagic mass 		
	Microscopically:		
	 abnormal trophoblastic hyperplasia & anaplasia with 		
	absence of chorionic villi, hemorrhage and necrosis with		
	direct invasion into myometrium and venous sinuses		
	 METS occur by vascular spread 		
	 Similar to HM: vaginal bleeding, abdominal pain, 		
	pelvic mass, high β-HCG Sx		
S/Sx	 1/3 no gyne features, only METS features 		
	• PPH		
	 AUB a year or more after an antecedent pregnancy 		
	 severe hemorrhage if tumor erodes 		
	 Excision biopsy (for Dx & Genetic analysis) 		
Mx	 Biopsy (often impossible – so clinical Dx mainly) 		
	 Classified in FIGO and treated as GTN 		

Ρ	lacental Site Trophoblastic Disease (PSTT)
Info	 uncommon consists predominantly of mononuclear intermediate trophoblast & syncytial elements, without chorionic villi, Infiltrating in sheets between myometrial fibres. Most PSTTs arise following non-molar pregnancies Generally present months to years after a gestation Produce few hCG & human placental lactogen (hPL) confined to the uterus mostly, and late metastasis
PSTT Vs ChorioCa	 Compared to choriocarcinoma, PSTT is associated with less vascular invasion and necrosis and greater tendency for lymphatic spread. PSTT are relatively insensitive to chemo & hysterectomy and pelvic lymphadenectomy remains the mainstay of ttt if there is residual disease confined to the uterus. Long-term (>5 y) clinical follow-up is recommended (hCG is not a reliable)
PSTT,	 mass enlarging the uterus (heterogeneous
Chorio US	appearance: necrosis & hemorrhage)
Features	 Hyper-vascularity on color Doppler
	 Tumor extends into parametrium
	Invasive Mole
Info	 Arises from myometrial invasion of CHM or PHM via direct extension through tissue or venous channels 10% of HM clinical: 15% show METS to lung/vagina diagnosed clinically responds to chemotherapy

Summary

- GTDs are curable with the preservation of fertility
- HM usually present with vaginal bleeding in pregnancy
- Suction by an experienced gynaecologist; oxytocics try to avoid it
- Registration with a specialised centre is mandatory
- hCG is an excellent marker for monitoring / the rare PSTT
- CHM or PHM not requiring chemo, hCG should be monitored for at least 6/12 following uterine evacuation & pregnancy is avoided
- Persisting GTD or GTN requiring CT, hCG should be monitored for at least 5 years to lifetime & pregnancy avoided for 12 months after completion of chemotherapy
- In women with a history of molar pregnancy, serum or urine hCG should be checked 6 w and 10 w after every future pregnancy
- COCPs should not be used until hCG become normal (undetectable)
- 15% of CHM and 0.5% of PHM will ultimately require CT for persistent GTD/GTN
- CT regimen is based on a prognostic scoring system
- The majority of women will conceive again following GTD/GTN with no increased rate of congenital fetal malformations.

Screening for Fetal Anomalies

Maternal US & Serum Mother Screening

- 1) Maternal age: alone has 30% detection rate
- 2) Fetal nuchal translucency (NT) thickness: 1 risk
- done between 10-14 weeks GA



1st trimester screening

	1 D 0.14cm
	3) <i>Maternal serum 8HCG</i> : 个 risk or down
	4) PPAP-A : pregnancy associated plasma protein A $(\downarrow) - \uparrow$ risk for Down syndrome
	- if abnormal between 10-14 weeks then it's associated with chromosomal abnormalities
	• If we use all 4 together: the detection rate for down is 79% with positive screening rate of 5%
	 US: visualization of nasal bone: ↓ risk of down and vise-versa if we use <u>all 4 + nasal bone</u> assessment the detection rate ↑ to 93% with +ve screening rate of 5%
2 nd trimester	 <u>Serum triple screening test</u>: αFP, βHCG, Unconjugated estriol (UE3) at <u>16-20 weeks</u>
screening	• αFP (maternal serum – MSAFP): 个 in:
-	a) it detect 80-85% of all open ' <u>neural tube defects'</u>
	b) <u>ventral wall defect</u> : gastroschisis + omphalocele



	 due to the false rate: a +ve NIPT must be confirmed by a diagnostic test as: a) Amniocentesis or CVS b) Provide full karyotyping analysis note: there is risk of pregnancy loss NIPT is offered to woman at risk for fetal aneuploidy, after pretest counseling 	
	 Methods: US, Amniocentesis, CVS, Cardiocentesis, Percutaneous umbilical blood sampling (PUBS) If high risk by screening do diagnostic test 	
	 Soft tissue marker: Fetal anomaly scan: Calcification on heart Short femur length Echogenic fetal bowel 	
Diagnostic Procedures	 Fetal anomaly scan 18-22 weeks: 1) Anencephaly 2) Ventriculomegaly 3) VSD, ASD, TOF, TGA 4) Gastroschisis, Omphalocele 5) Cleft lip 6) Absent limbs 	
	- if you discover a defect <u>incompatible of life</u> (anencephaly, dextrocardia): <u>terminate the pregnancy</u>	
	 - if you discover an <u>isolated defect</u> look for other "<u>detailed scan</u>" if normal offer amniocentesis, CVS because it might be associated with <u>chromosomal abnormality</u>, also do <u>TORCH</u> screening, if all (-ve), refer her for <u>genetic counseling</u> 	
Important	NIPT: done at 10 weeks Nuchal Translucency: done at 11 – 13 weeks + 6 days BPP: done at 16-19 week Fetal anomaly US scan: done at 18-22 week	

Rh alloimmunization (Rh-ai)

immunologic (Rh antibody mediated) disorder that occurs in a pregnant, <u>Rh (-)</u> <u>woman who is carrying an Rh (+) fetus</u>

General Information

• the majority of Rh-ai are due to <u>antibodies to D antigen 90%</u> (also it is the only preventable type) (other types: C, E, c, d, e, partial D antigens)

• A person who lacks the D antigen on the surface of RBCs is "*RhD-negative*," and an individual with the D antigen is "*RhD-positive*."

• in general, *two exposures* to the RhD antigen are required to produce any *significant sensitization* (unless the 1st exposure is massive) so with the 2nd pregnancy if the fetus (+) the antibodies attack the Rh antigens of the baby causing hemolysis > anemia > heart failure > baby death.

• The initial response to exposure to the RhD antigen is the production of (IgM -which cannot cross the placenta) for a short period of time, followed by the production of IgG (that can cross). If the fetus has the RhD antigen, these antibodies will coat the fetal RBC.

- Mild, moderate, severe (usually the same or more severe in next pregnancies)
- If a woman has a hx of fetal hydrops the risk of hydrops is 90% with a

subsequent pregnancy (usually at the same time as the previous one or earlier)

Identifying high risk woman

• Blood sample is taken in the 1st prenatal visit: check blood group, RhD type & antibody screening

- if (-) RhD with (+) antibody titer (RhD sensitized), check father RhD

- if father is RhD (-), then the baby is (-) also and hemolysis won't occur

- if father is RhD (+), check Rh genotype by PCR, if <u>homozygous</u> for D antigen, the baby will be RhD (+) and monitoring is required, if the father is <u>heterozygous</u>, the baby has a 50% of being RhD (+) so we need to check the fetal RhD genotyping, which is done by:

a) <u>Testing cell free fetal DNA</u>: from the maternal plasma (as early as the end of 1st trimester)

b) <u>Amniocentesis</u>: at 2nd trimester, RhD done using amniocytes, but risk is fetomaternal hemorrhage and worsening of hemolysis

c) <u>Chrorionic villus sampling</u>: greater risk of hemolysis if baby RhD (+)

Hydrops	 form of in utero heart failure
Fetalis	 characterized by: fetal ascites, pericardial effusion, pleural
	effusion, subcutaneous edema, polyhydramnios

	• <u>Ethnicity</u> :	
	- 15% White Americans are RhD (-), 8% African Americans, 1-2%	
RF	Native Americans, 1-2% Asians	
	• <u>Advanced GA</u>	
	<u>Previous history</u>	
	• Fetomaternal hemorrhage is very common, yet incidence of	
	RhD immunization within 6 months of the delivery of the 1 st	
Incidence	RhD (+), ABO-compatible infant is 8%. In addition, the incidence	
	of sensitization with the development of a 2ry immune	
	response before the next RhD (+) pregnancy is 8%.	
	 Severe extramedullary hematopoiesis 	
	Portal HTN	
	Hypoalbuminemia	
	 Hyperbilirubinemia: damage the CNS leading to <u>neonatal</u> 	
Complications	encephalopathy and kernicterus (cerebral palsy, sensorineural	
	<u>deafness)</u> , Rh₀(D) immune globulin (RhIG – Anti-D) prevent this	
	• Heart failure	
	Hydrops fetalis	
	• IUFD	
	** Note: if hemolysis is mild the fetus might compensate	
Hydrops	form of in utero heart failure	
Fetalis	 characterized by: fetal ascites, pericardial effusion, pleural 	
	effusion, subcutaneous edema, polyhydramnios	
Kleihauer-	 used to detect fetomaternal hemorrhage 	
Betke test		
	How to Diagnose / Screen	

Maternal Anti-D Antibody Titer

- Used as a screening tool to estimate the severity of hemolysis
- For women with a previous history, follow them up despite the titer
- Fetus in the first immunized pregnancy is not in serious jeopardy;
- 1) when the titer remains **below 1:16**. >> repeat titers every 2-4 weeks.
- 2) If the titer rises to 1:16 or greater:
- a) Detailed US to detect hydrops
- b) Doppler studies of the MCA are indicated

US Detection of Fetal Hemolytic Disease

1) Serial Doppler assessments of peak systolic velocity in the fetal MCA:

- it have proven to be the most valuable tools for detecting fetal anemia.

- One of the earliest signs of *fetal anemia* is an elevated doppler peak velocity.

- In at-risk pregnancies, test is performed every 1-2 weeks from 18-35 GA.

A fetal MCA peak systolic velocity value <u>above 1.5 multiples of the median</u> for GA is predictive of moderate to severe fetal anemia and is an indication for percutaneous umbilical blood sampling (PUBS) for determination of fetal hemoglobin concentration. Followed by Intrauterine fetal transfusion if needed.
 After 35 weeks' gestation, this test may produce a higher false-positive rate.

2) Detailed fetal assessment + placental size and thickness and hepatic size.

• Fetal hydrops is easily diagnosed on US (everything is enlarged, fluids)

Percutaneous Umbilical Blood Sampling (PUBS)

- most accurate method for the Dx of hemolysis.

- it measures fetal hemoglobin, hematocrit, blood gases, pH, and bilirubin levels.

- If the <u>fetal hematocrit is < 30</u>, or <u>more than two standard deviations (SD) below</u> <u>the mean</u> for GA, intrauterine transfusion is indicated.

- there is higher risk of fetomaternal hemorrhage so it should not be a first-line.

- PUBS indications:

- 1- US evidence of fetal hydrops.
- 2- MCA peak systolic velocity is greater than 1.5 multiples of the median for GA.
- 3- fetus is at <35 weeks GA.
- 4- moderate to severe fetal anemia

Mx of the at risk pregnancy

A) Intrauterine Transfusion

- The goal is to transfuse fresh group O, Rh-negative PRBCs.

- Cannot be done until 18-20 weeks', because fetal size limits vascular access.

- Repeat transfusions are generally scheduled at 1 to 3-week intervals, and the final transfusion performed at 32-35 weeks' gestation, then delivery when it reaches 37 weeks, or if antepartum testing indicates severe fetal compromise.

 overall survival rate following intrauterine transfusion is about 90%, unless hydrops occurred before transfusion then it's lower

- 90% of survivors are reported to have normal neurologic outcomes

- this technique is done under US guidance

- In nonhydropic fetuses, the blood should be absorbed within 7 to 9 days.

- IV transfusion is the method of choice for correcting fetal anemia, and if we can't do it (e.g. GA <20 weeks) then we do intraperitoneal transfusion.

- IV Transfusion: preferred method, why?

1- Fetal survival is better especially if there is no evidence of hydrops.

2- transfusion into the peritoneal cavity can result in fetal bradycardia or a pseudo sinusoidal fetal heart rate pattern following the procedure due to compression at the site of insertion of the umbilical cord.

- The volume of transfused blood is based on the estimated fetal body weight, as determined by, the initial fetal hematocrit, the target fetal hematocrit, and the hematocrit of the packed red cells to be transfused.

B) Maternal plasmapheresis

combined with IVIG may be helpful in cases of severe erythroblastosis when intrauterine transfusions have not been successful

C) Phenobarbital

used to induce fetal hepatic enzyme maturation, thereby increasing uptake and excretion of bilirubin by the liver. should be initiated 1 week before delivery

	rubin by the liver. should be initiated I week before delivery
Time of Delivery	 <i>evaluation twice weekly from at least 32 weeks until delivery</i> for fetal well-being (NST, BPP) & every 3 weeks for fetal growth. <i>Goal is term</i> delivery unless there are complications There is no absolute GA cutoff for transfusions, but after 35 weeks the risk of an intrauterine loss greater than neonatal death risk. It may be prudent in this setting to deliver the fetus
	 if delivery is expected to occur < 34 w (or if amniocentesis
	suggests an immature lung), <i>betamethasone</i> is given at least 48
	hr before delivery to <i>enhance lung maturation</i>
	 at 28 weeks (3rd Trimester) – the routine prophylaxis
	• During labor or immediately postpartum (time of the greatest
When to give	risk for fetomaternal hemorrhage – greatest blood loss)
Anti-D (Rh	 Within 72 hours of delivery of a RhD + baby
immune	 if any complication due to fetomaternal hemorrhage is
globulin)	recognized (at any antepartum event such as amniocentesis
	that may increase the risk of trans-placental hemorrhage)
"Prevention"	
	** 300 micrograms (or 1 U) of Anti-D can neutralize 30 mL of
	fetal RhD (+) blood in the maternal circulation.
	** Anti-D this will decrease the problem by 91%

BOX 15-1

INDICATIONS AND DOSING FOR RH IMMUNE GLOBULIN

- Blood type and antibody screen are performed for all pregnant women at their first prenatal visit.
- Women who are RhD-negative with a negative initial screen should have a repeat screen at 28 weeks.
- Those women with a negative screen at 28 weeks should receive 300 µg of Rh immune globulin (prophylactically).
- Those women with a positive screen should have their antibodies identified. If RhD-negative, they should also receive 300 µg of Rh immune globulin.
- All pregnant women who are RhD-negative and who are not sensitized (anti-D-negative) and who experience (1) spontaneous or induced abortion, (2) ectopic pregnancy, (3) significant vaginal bleeding, (4) amniocentesis, (5) abdominal trauma, or (6) cephalic version should receive 50-100 µg of Rh immune globulin before 12 weeks' gestation and be administered 300 µg if later than 12 weeks.
- Rh immune globulin is not necessary for complete molar pregnancies, but it is necessary for partial molar pregnancies, where fetal tissue may be present. Because this is not always clear at the time of evacuation, 300 µg of the immune globulin should be given.
- The greatest risk of fetomaternal hemorrhage is at the time of delivery. Rh immune globulin (300 μg) should be given routinely within 72 hours of delivery to all Rh-negative, anti-D-negative women who deliver an Rh-positive child.
- Additional Rh immune globulin is indicated if the delivery is complicated by excessive hemorrhage (>30 mL of fetal blood suspected or documented by Kleihauer-Betke testing).



The interaction occur in the 3rd trimester (28 weeks) and after delivery (because the largest blood loss in pregnancy is at time of delivery)



Instrumental Delivery " Assisted Vaginal Delivery "

Tools	Obstetric Fore	eps • Vacuum Extractor	
Pre- requisite for it	 Know presenting part Know Position No Cephalopelvic Disproportion (No true obstruction) Expert Obstetrician Fully dilated cervix Enlarged head Adequate analgesia Presence of uterine contractions Empty bladder Presence of an indication Ruptured membranes 		
	Indic	ations for Instrumental Delivery	
	Fetal	Mother	
 Fetal dis After co during a b Premate 	stress ming head preech delivery ure baby	 Failure of progress in 2nd stage of in nulliparous it is defined as lack of confor 2 hr without regional anesthesia (RA) in multiparous it is defined as lack of confor 1 hr without RA or 2 hr with RA 	labor atinuing progress or 3 hr with RA ntinuing progress
• Face mento anterior		• To Shorten the 2 nd stage of labor benefit (Exhausted mother): HTN, Ca pulmonary disease, Duchenne dystrophy these conditions there is an indication fo strenuous pushing	for maternal ardiac disorder, , neurological, & in r RA to lower the

	Risks of Instrume	ntal Delivery
	Fetal	Mother
 Scalp injury (cephalohematoma, large caput, subglueal hemorrhage) Erb's palsy, more NICU's Skull fracture, ICH Facial palsy (foreceps) Neonatal jaundice (hemolysis) 		 Maternal laceration of the genital tract (from vaginal tear to cervical to even uterine rupture) Uterine Atony (PPH) Infection (Endometritis) needs Abx! Pain, Psychological, urine retention
Mechani sm of Foreceps	 Blade of the forceps: Cephalic curve Pelvic curve Shrank Lock Handle Technique: insert the forceps left to the patient then right to the patient then it should lock easily (no forceful application – easily applied and easily locked), then do traction downward and upward according to the anatomy of the - it is allowed for 2 traction. if the patient is curve is a structure of the patient is curve of the patient is curve of the patient the patient of the patient is curve of the patient is curve of the patient is curve of the patient of the patient of the patient is curve of the patient is curve of the patient of the pat	Pelvic Pelvic Cephalic curve Blades Blades Shanks Handle
Mechani sm of	 Technique: put the vacuum on the vertex anterior fontanelle (median, pa (that's why you have to know the second seco	between the posterior and the ramedian) to enhance the flexion he position of the fetus, and if you

Vacuum	can't determine the position go for C/S)
Extractio	 with occipito-posterior: the traction should be upward, you can
n	also use it with occipito-transverse
	- it is allowed for 2 "pop-offs" if the progress down the birth canal it
	is not obtained with appropriate traction go for C/S
Failure	 Issue with the position (malposition)
of the	• CPD
instrume	 True Obs (Caput, Moulding)
nt	
	 Vacuum: Rigid (metallic), elastic (Kiwi)
Types of	• Forceps:
the	a. High (Kielland): it has no pelvic curve to allow rotation and it is not
instrume	use anymore
nt	b. Mid-cavity
	c. Outlet (Wrigley;s forceps): most commonly used
	d. Piper foreceps: used with after coming head during breech
Episioto	 used to provide more space for manipulation
my	 in vacuum delivery it is not an absolute contraindication
	 usually we do episiotomy at first to avoid more injury
	 The usage of the 2 instruments (sequential instrumentation) is not
	allowed!
	 Vacuum extractor cannot be used if premature <34 w !
	 Can we use foreceps in C/S? Yes!, when the head is high, and not
General	well applied to cervix
Question	• Vacuum delivery should be controlled you have to apply well
3	support on the perineum to avoid rapid detachment of flexion
	 Usually the injury occur with extension of the head coming out, however it was a flexion state

• You can apply vacuum on dilatation of 8-9 cm(nearly dilated cervix) when there is a fetal distress (cord prolapse)

• When you apply the cup of vacuum, you have to make sure that there is no tissue (maternal) in between the cup and the head if there was it will lead to severe laceration!

• the application of traction weather with forceps of vacuum, it have to be with uterine contractions and stop when the contraction stops

• if you are unhappy with the ongoing process go for C/S

Flash-cards for these topics:

https://quizlet.com/480026976/dr-firas-gyne-flashcards/?x=1jqU&i=2fgxb8&fbclid=IwAR1HbPECNRDN0oNeST_4Vh6BticY MK78zGhBB5fFppjVFoXPbrtG9tilRp0

Best Wishes

DONE BY: YAZAN ALAWNEH