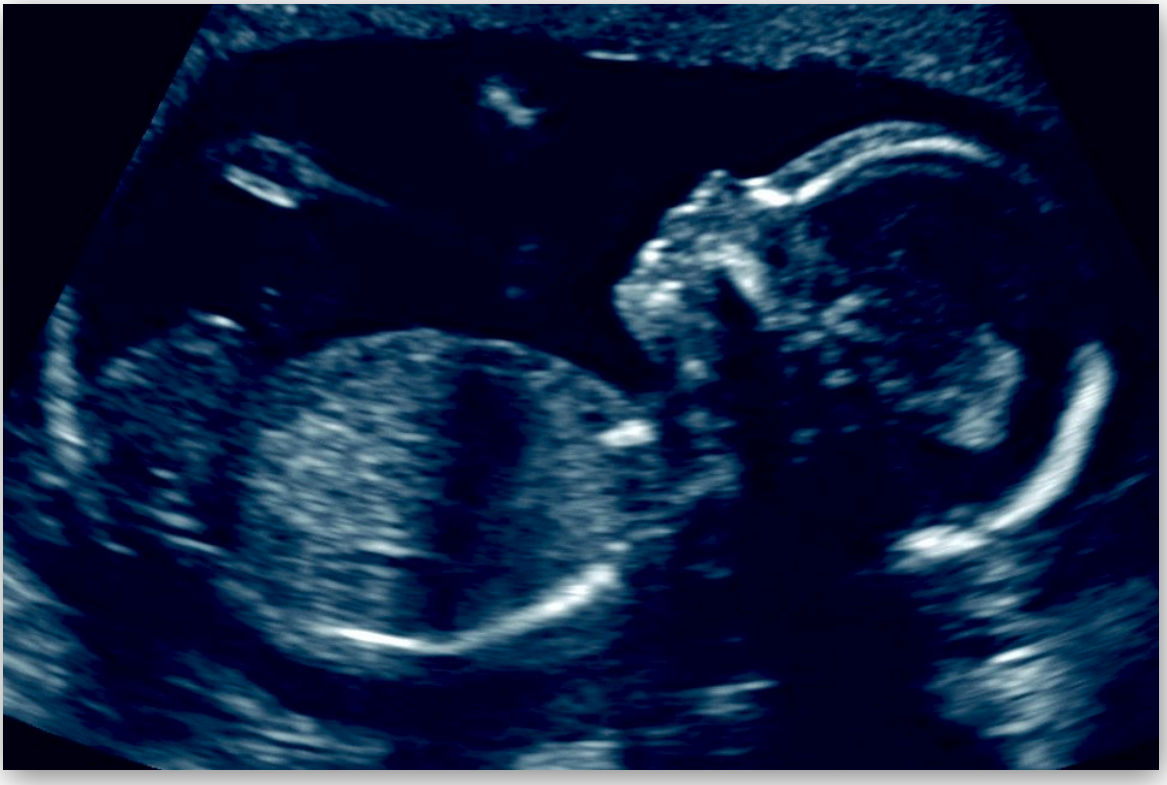
Study guide to

Ob/Gyn Registry Review



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2021 edition

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Gynecology

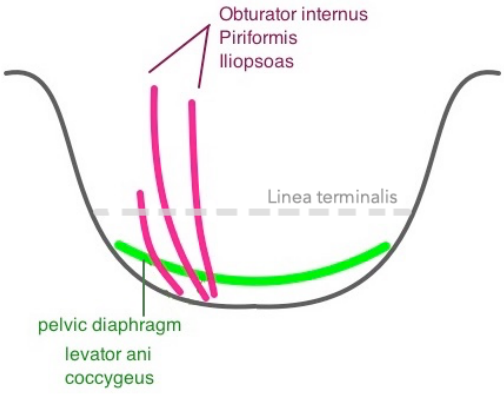
Normal Anatomy of the Pelvis

Bony boundaries - sacrum, coccyx, innominate bones (ilium, ischium, pubic symphysis)

- Linea terminalis - imaginary line from pubic symphysis to sacral prominence (top of sacrum)

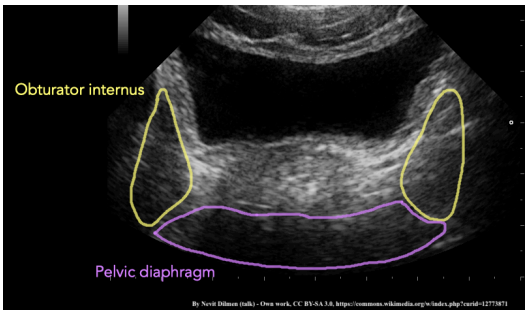
TRUE PELVIS is deep and below the linea terminalis (at the bottom/inferior). Imagine all the structures that can be seen or is in range of transvaginal imaging

- Bladder, small bowel, ascending and descending colon, rectum, uterus, ovaries, fallopian tubes, internal iliacs, and 5 muscles



Musculature: 5 true pelvic muscles

Pelvic diaphragm: *levator ani* and *coccygeus*.
Hammock shaped muscles that give support to pelvic organs. (Weakening of these muscles leads to Ut prolapse)
Location: Identified scanning transverse inferiorly at level of vagina. Posterior to bladder, vagina, and rectum



Adnexa: "O.P.I" muscles
obturator internus (lateral to bladder), *piriformis* (posterolateral), *iliopsoas* (anterolateral)
Location: when scanning adnexa in transverse, appear as ovoid hypoechoic structures that elongate in sagittal. Adjacent or lateral to bladder/ovaries/uterus

Any other muscle…psoas major, rectus abdominus, oblique, etc… is NOT in true pelvis.
And will not be adjacent to any true pelvic organ

Uterine Ligaments

- *Broad ligaments*: double fold of peritoneum. From lateral sides of uterus to walls of pelvis supporting pelvic organs. Only ligament ever visualized on sono, only when there is pelvic ascites
- *Round ligaments*: between folds of broad ligament. Supports fundus of uterus (superiorly)
- Cardinal ligaments: contains vasculature of uterus

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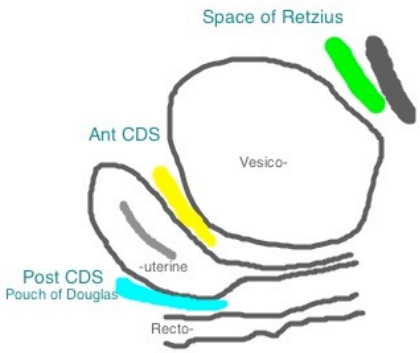
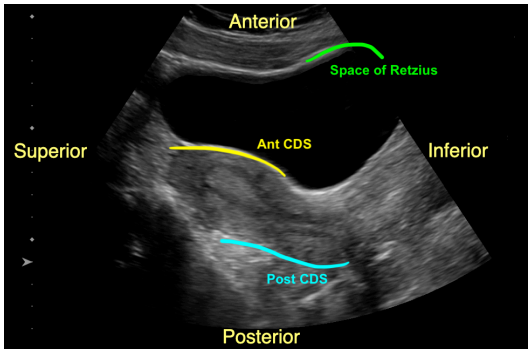
Spaces

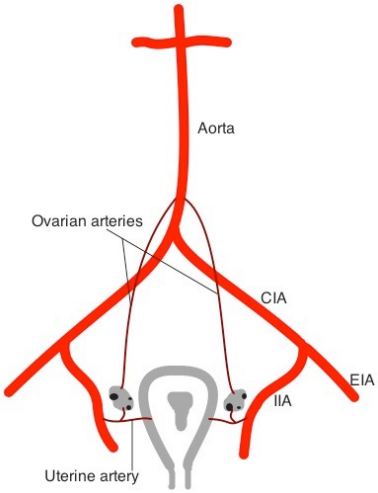
Intraperitoneal cavities where fluid can collect

- Retropubic space: AKA space of Retzius anterior to bladder
- Adnexa: lower quadrants of abdomen and lateral spaces to uterus. Ovaries are within the adnexa
- Anterior CDS : AKA vesicouterine pouch. Between bladder and uterus. (Ant to uterus)
- Posterior CDS : AKA rectouterine pouch / pouch of Douglas. Between uterus and rectum (Post to uterus)





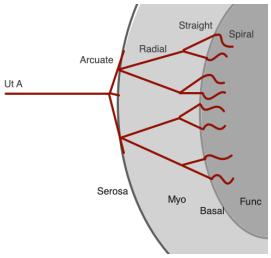
Vasculature



Arterial supply

Uterine arteries: branches of internal iliac artery (AKA hypogastric a.)

- Arcuate arteries: periphery of myometrium
- Radial arteries: deeper into myometrium
- Straight and Spiral arteries: layers of endo
  (Straight feeds basal layer and spiral to functional)



Ovarian arteries aka Gonadal arteries - originate from aorta

Note: Ovaries received a dual blood supply (ovarian a. and uterine a.)

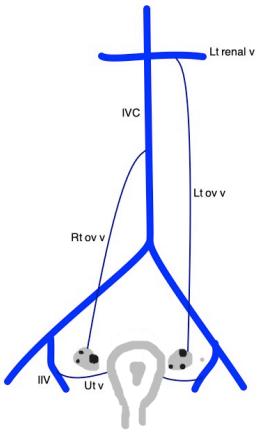
Venous drainage venous vessels mirror arterial counterparts.

Uterine veins return or drain into internal iliac veins.

Right ovarian vein drains into IVC

Left ovarian vein drains into left renal vein

(It ovarian vein is the longest pelvic vessel)



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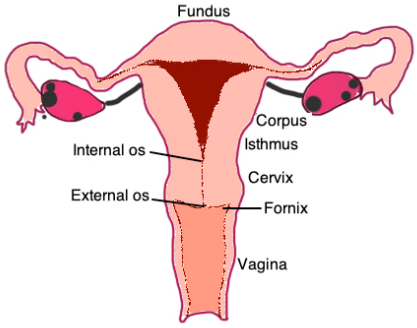
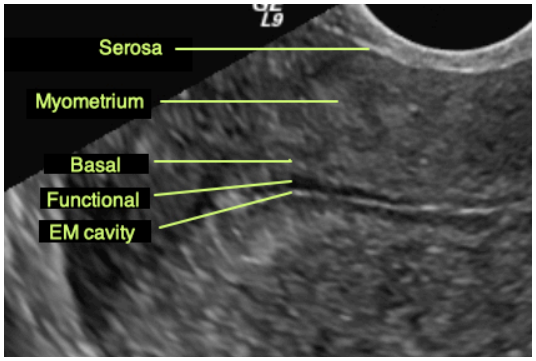
Uterus

Pear shaped, retroperitoneal organ located anterior to rectum, posterior to bladder, bound laterally by broad ligament.
Embryology: developed from fusion of paired Müllerian ducts.



FOUR DIVISIONS: fundus, corpus, isthmus, cervix

Fundus: most superior and widest. Tubes attach to uterine cornu
Corpus: body, largest area
Isthmus: AKA lower uterine segment in pregnancy
Cervix: internal and external os. External os opens into vaginal canal (most inferior part) and surrounded by vaginal fornix (ring like pouch or recesses)



THREE LAYERS: serosa, myometrium, endometrium

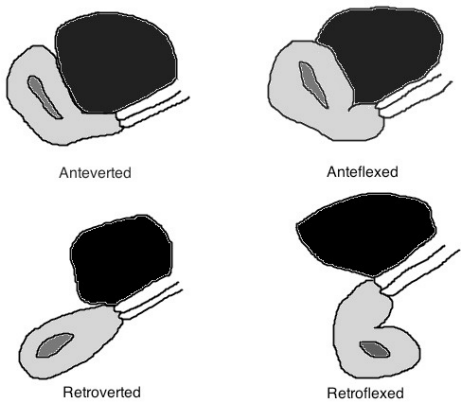
- Serosa: AKA perimetrium. Outermost/organ fascia.
- Myometrium: muscular layer
- Endometrium: mucosal layer consisting of 2 layers
  Basal layer (deep)
  Functional layer (superficial)This layer is shed during menses

SIZE AND SHAPE depends on age, parity, and presence of pathology

Neonatal uterus: Prominent uterus due to maternal hormone stimulation. Cervix is enlarged with approximate 2:1 ratio (double the size) to body
Prepubertal: Tubular in shape. Body = cervix
Puberty: Increase in fundal diameter = pear shaped. Reproductive years (6-8cm)
Menopause: Decreased uterine size (4-6cm)

POSITIONS/ORIENTATION:

Anteversion - body tilts forward, 90 degree angle with cx
Anteflexion - body folds forward, comes in contact with the cx
Retroflexion - body tilts back and comes in contact with back of cx
Retroversion - body tilts back without a bend.



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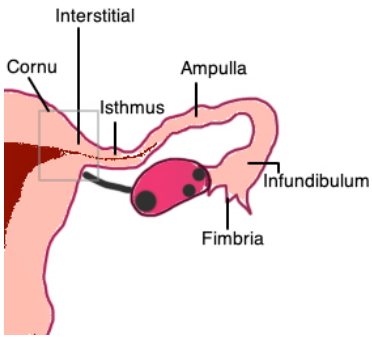
Fallopian Tubes

AKA oviducts, uterine tubes, salpinges. 7-12cm tubes extending from cornu within the broad ligaments to the adnexa. Means of fertilization and transportation to uterus. Tiny, hairlike structures, *cilia*, inside the tubes move back and forth to aid the movement of fertilized ovum.
Not typically seen on US. Only when pathology or distended by fluid can be visualized.

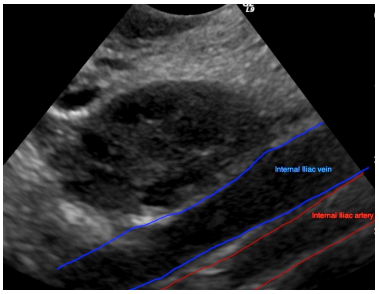


5 SEGMENTS:

- *Interstitial*- most proximal. Where tube attaches to uterus (at cornu)
- *Isthmus*- "bridge" connects interstitial to ampulla
- *Ampulla*- longest and most tortuous.

  Most common location of fertilization and ectopics
- *Infundibulum*- distal and widest portion. At ends fimbria: fingerlike extensions of infundibulum that draw unfertilized egg into tube

Ovaries



Paired, oval-shaped intraperitoneal endocrine organs surrounded by OPI muscles and internal iliac vessels

- Ovarian ligament supports ovary from lateral side of uterus to ovary.
- Suspensory ligament supports ovaries from lateral pelvic side walls

Produce estrogen and progesterone. *Stimulated by* FSH and LH.
Outer cortex: site of oogenesis/follicles. Medulla: vasculature and lymphatics
Ovarian Volume = L x W x H x 0.523

Basic physiology of ovaries:

Responsive to FSH and LH *Produce estrogen and progesterone*

In response to FSH = Follicles develop, Graafian (dominant) follicle matures. Thecal internal cells of follicles produce estrogen. Ovum is inside the *cumulus oophorus* of dominant follicle (seen as a daughter cyst within dominant follicle). *At this point, ovulation will occur within 36 hours.*
LH = Ruptures graafian and then is replaced by *corpus luteum*. Corpus luteum releases progesterone. When the corpus luteum regresses, *corpus albicans* takes its place.

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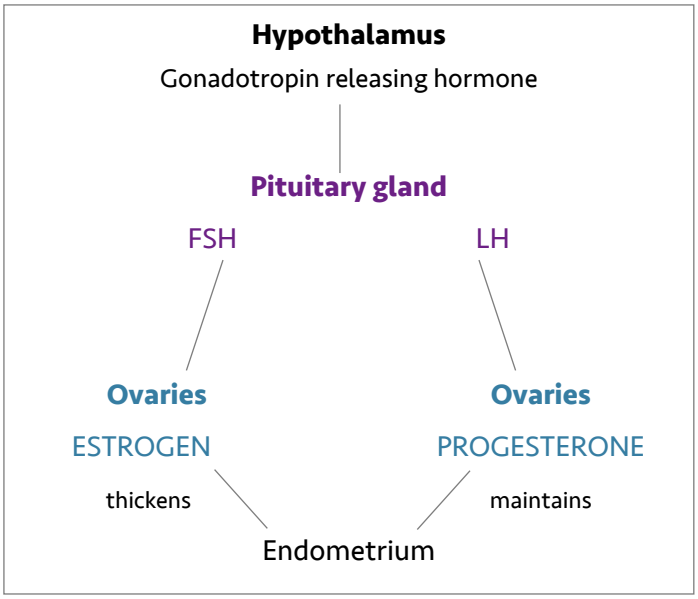
Normal Menstrual Cycle

Think about the whole purpose of the cycle. To get pregnant. So that means 2 main important things needs to happen: there must be an egg released and an endometrium prepared. All steps in the cycle are a means to that end.

Hypothalamus releases gonadotropin-releasing hormone to regulate release of hormones by the anterior pituitary gland. The pituitary gland releases FSH. FSH stimulates ovaries to develop follicles and maturation of the dominant follicle. Follicles in turn produce estrogen. As the dominant follicle reaches maturity, there is a peak in estrogen levels. This signals the pituitary to release the LH surge. This stimulates the rupture of the dominant follicle = ovulation. The ruptured dominant follicle is now termed the corpus luteum which secretes progesterone and small amounts of estrogen.

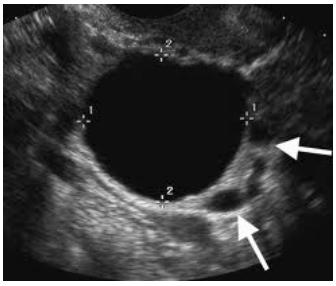


Effects on the endometrium

The endometrium is directly affected by estrogen and progesterone. Estrogen thickens the endo and progesterone maintains it and prepares for implantation.

If no pregnancy occurs, the corpus luteum regresses and progesterone levels drop. When the progesterone decreases, the endometrium begins to slough off and menses begins

Timing, Phases, Appearance



Days 1- 14 Ovary

Follicular phase of ovary FSH stimulates follicle development and dominant follicle matures increasing to about 2.5 - 2.7cm until ovulation around day 14. Follicles release estrogen

Days 1-14 Endometrium

Menstrual phase Days 1-5 approx are menses and shedding of endo (no specific appearance)

Proliferative phase Days 6-14 Endo changes so much during this phase, so we must use terms early or late depending on where we are in this phase

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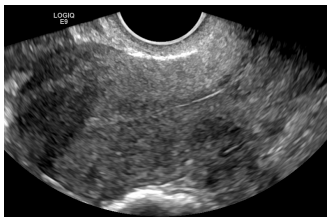
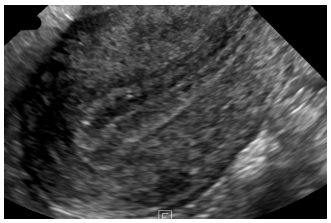


Early proliferative Immediately following menses, endo is thin, echogenic and measures no more than 4mm



Late proliferative (periovulatory) will reach 6-10mm and appears as *"three line sign" (echogenic rim is basal layer surrounding hypoechoic functional).*

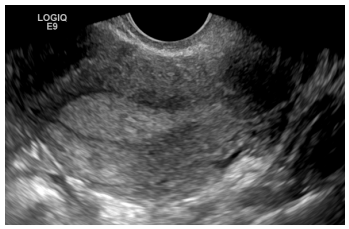
DAY 14 *Ovulation*. LH surges causes rupture of dominant follicle, releasing ovum. Free fluid may be settle in post CDS. Ovulation occurs 14 days prior to the start of the next menstrual cycle.
When calculating ovulation day = subtract 14 from # of total cycle days

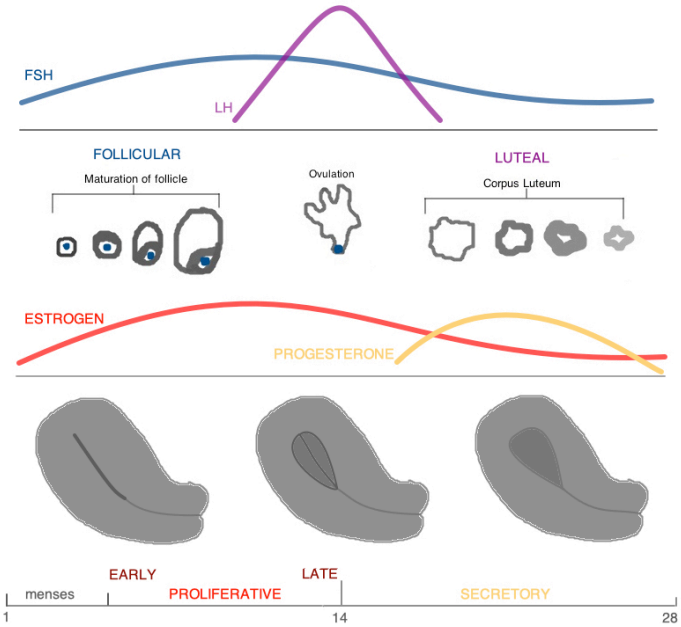
Example: In a 36 day cycle, ovulation would occur on day 22 (36 -14=22)

Days 15-28

Luteal phase of ovary Graafian follicle becomes corpus luteum which produces progesterone to maintain endo thickness. Towards end of phase corpus luteum regresses if no fertilization

Secretory phase of endo Progesterone maintains thickness to prepare for implantation. Endo appears thick and echogenic 7-16mm. Menses normally begins day 28 due to progesterone drop





Ovarian and Endometrial Relationship
Since the ovaries produce the hormones that stimulate the endo, they will "match"
Pay attention to the question, as it may show you an endometrium but ask about ovarian events.

- Follicular goes with Proliferative
- Ovulation with Late Proliferative
- Luteal with Secretory

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Clinical History

Anything you can know before you scan the patient. Including prior exams (including ultrasound, MRI, CT, Nuc Med, etc), and diagnosis relating to pelvis, patient history and complaints, age, parity, menstrual history including LMP (first day of last period), medicinal (oral contraceptives, Hormone Replacement Therapy HRT, tamoxifen), surgical, and family histories (Hx of CA)

- Gravida = # of pregnancies Para = # of pregnancies carried to term

Based on patient presentation and indication for exam, we can "build" a picture of what pathologies may present. This is will also determine which is exam is best indicated.

Indications	Definition/examples
Acute Pain	Sudden onset pain
Chronic pain/pressure	Long term symptoms
Abdominal distension	Pelvic fullness or enlargement
Abnormal ut bleeding	Any cause of bleeding inc lesions
Dysfunctional ut bleeding	Caused by endocrine abnormalities
Infertility	Unsuccessful conception after 1year
IUD	Intrauterine device
Increased cancer risk	Ex- family hx, tumor markers
Abnormal pelvic exam/labs	WBC, hematocrit, CA-125,etc
Delayed menses	Not reached menarche by age 15
Precocious puberty	Puberty changes by the age of 8
Congenital anomalies	Bicornuate, etc
Physical changes	Ex- hirsutism, obesity, inc girth
Follow up to abnormal study	Adnexal mass, endo hyperplasia

Transabdominal 2-6 MHz curvilinear: Prep: to drink 32 oz of water and not void, until bladder extends over uterine fundus. If patient is unable to drink, bladder may be filled retrofilled via Foley catheter. Global view of entire pelvis or when large pathology is present. Lacks detail due to distance from organs and lower frequency. Limited when pt is obese or retro uterus

Transvaginal 6-9 MHz endocavity: Prep: empty bladder. Improved resolution of uterus, endometrium, and ovaries due to superficial imaging and increased frequency. Limited due to shallower FOV and scanning depth. Contraindicated in pediatrics or pt's that cannot tolerate

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Terminology

Additional helpful terms

Learn your prefixes and suffixes!
You may not know the word or the pathology right away. But if you know how to break the word down, you can figure it out!

Prefixes		*Suffix*	
a-	without/none	-menorrhea	menses
dys-	abnormal/painful	-rrhagia	bleeding
hyper-	increased	-uria	urination
hypo-	decreased	-pareunia	intercourse
oligo-	less	-plasia	growth
poly-	many	-genesis	formation
sub-	under	-oma	mass
intra-	inside	-itis	infection
inter-	between	-colpos	vagina
hydro-	fluid	-metra	uterus
hemato-	blood	-salpinx	fallopian tubes
meno-	heavy		
metro-	irregular		

Mittelschmertz: "middle pain" pain in middle of cycle, near ovulation.
Primary amenorrhea: Failure to have menses by the age of 16. Never reached menarche
Secondary amenorrhea: Menses stopped

Protocol

- Uterus to be eval for size, shape, and orientation. Measured length and AP in sagittal fundus to external os of cervix (length), width of body in transverse.
- Endometrium measured ML SAG at greatest AP dimension excluding fluid, if present. If there is fluid, measure both layers of endo and add them together. Fluid is not included
- Adnexa regions for ovaries and tubes. Ovaries measured LxWxH and sweep through to adnexa to eval for pathology. Ovarian Volume = L x W x H x 0.523
- All masses or abnormal structures to be documented in 2 planes and measured noting location in relation to organs or landmarks.
  Most important when documenting pathology is documentation in 2 planes in B-mode

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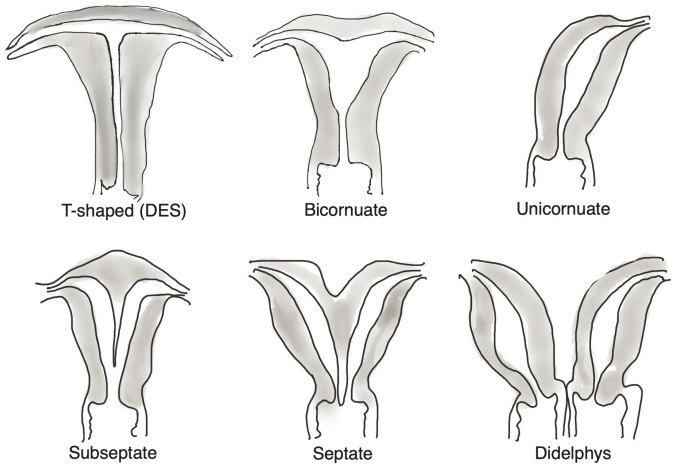
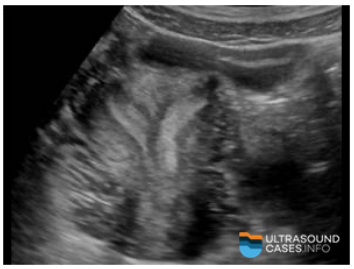
Congenital Malformations

Incomplete, abnormal fusion or lack of formation of paired Müllerian ducts. May be associated with menstrual disorders, infertility, and OB complications. *** Check KIDNEYS for anomalies

- Inc risk with fetal exposure to diethylstilbestrol **DES** (drug given to treat threatened miscarriages in the 70's) **Most commonly associated with DES is the T shaped uterus**



- Arcuate: Mildest of all. *Normal contour*, slight indentation of fundal endo
- Bicornuate: AKA Bicornis Unicollis 1 endo cavity that divides into 2 at fundus. "Y" shaped. Uterine fundus has concave contour. Uterine width NL
- Subseptate: *Normal uterine contour* with 2 separate endo cavities
- Septate: **Most common congenital uterine anomaly** 2 completely separate endo cavities, Uterine contour is concave at fundus. (Image right)
- Didelphys: Complete lack of fusion. 2 vaginas, cervices, and uteri
- Unicornuate: Lack of formation of one duct. Single horn



<u>Congenital malformations of vagina</u>

Vaginal atresia and imperforate hymen. Both can lead to accumulation of fluid due to obstruction. Both have same clinical presentation: pain and primary amenorrhea in a adolescent girl

Imperforate hymen is most common cause

Sonographic findings: depending of level of obstruction… distention of uterus, vagina, or both with anechoic or complex fluid. Fluid accumulated proximal to level of blockage

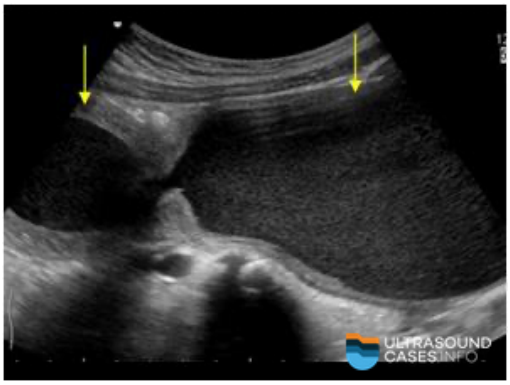
- Vaginal Atresia : absent or closed vagina, so it cannot be distended. Only uterus and cervix will be distended with fluid or blood = hydrometra or hematometra
- Imperforate hymen : closed hymen. Everything above it can be distended = hydrocolpos, hydrometracolpos, hematometracolpos

Case Study

This image demonstrates fluid with low level echoes distending the vaginal canal and uterus

>> hematometracolpos

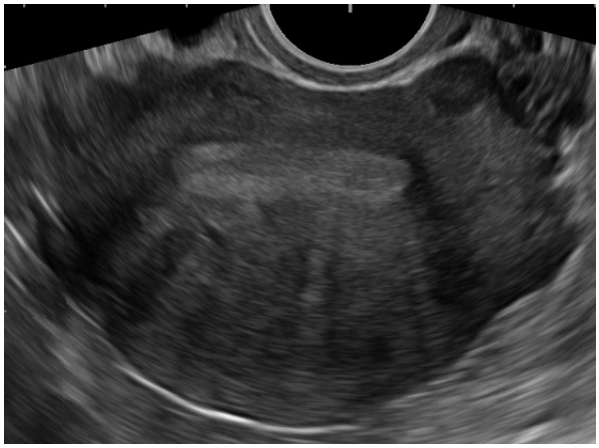
Indicates imperforate hymen



Uterine Pathology

Adenomyosis

Invasion of endometrial tissue into myometrium (inside uterus). Can be either focal or diffuse usually noted by thickening of the posterior uterus. Focal mass-like adenomyosis is called adenomyoma. MRI important for diagnosis. Increased risk in patients with fibroids.



Clinical

Dysmenorrhea, menometrorrhagia, pelvic pain, and dyspareunia, multiparous

Sonographic

Enlarged uterus with diffusely heterogeneous myometrium

Thickening of posterior uterus

Small cystic spaces scattered throughout myo

"Linear striations"

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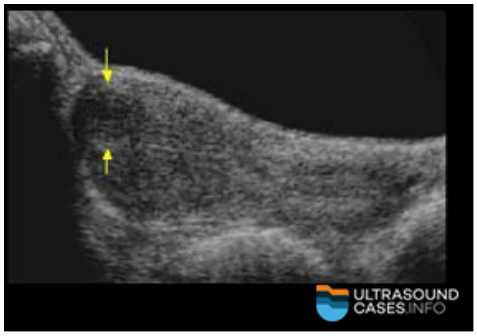
Leiomyoma (Fibroid/myoma)

Benign, smooth muscle tumor. **Most common benign gyn tumor** and leading cause of hysterectomy and gyn surgery

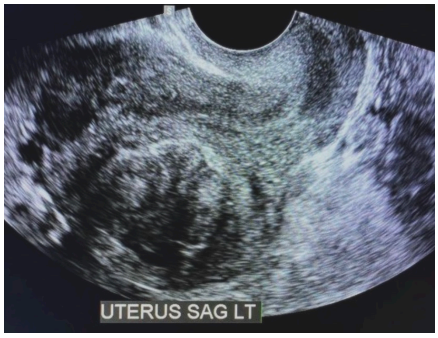
Stimulated by estrogen ∴ increase during pregnancy and decrease after menopause. May impact fertility depending on location, size, and number.

Clinical	Sonographic
Depends on location and size Abnormal bleeding, pelvic distension, pressure, infertility, urinary frequency	Hypoechoic mass with poor thru transmission Multiple myomas appear as diffusely heterogenous, bulky, enlarged UT

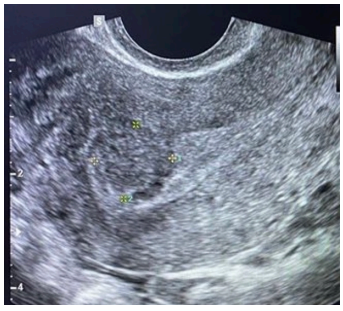
- Intramural- Most common. Within the muscle wall of the uterus. May make the whole uterus bulky but will not directly change the contour



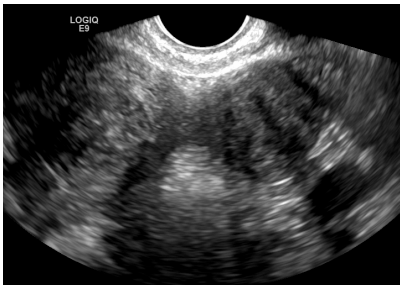
- Subserosal- grows under the serosal layer. Distorts the outer contour



- Submucosal- adjacent to endo and distorts contour of endo. Most likely to cause bleeding issues. Intracavitary are pedunculated, submucosal fibroids that project into endo



- Pedunculated- a type of subserosal myoma that grows out and attaches by a stalk. May resemble adnexal masses. Most likely cause abdominal distension or pelvic pressure.



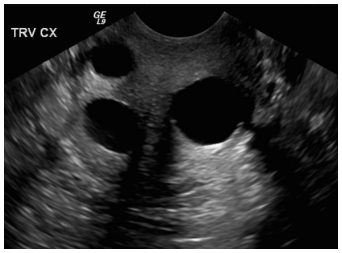
Leiomyosarcoma

Malignant form of fibroids. Rapid increase in growth most commonly found in perimenopausel and postmenopausel women.

SAME clinical and sonographic findings as fibroid except it is a rapidly growing mass in the UT.

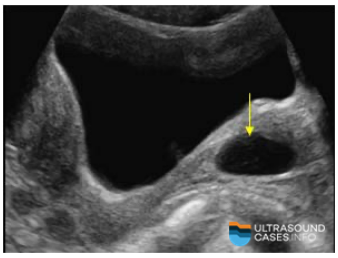
Cervical Carcinoma

Most common female malignancy under the age of 50. Not routinely Dx with ultrasound. May present as heterogeneous, enlarged CX or focal mass within the CX.



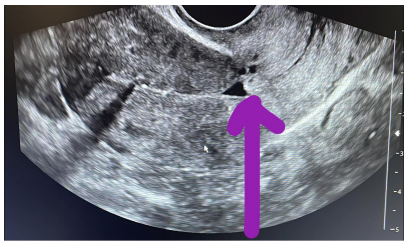
Nabothian cyst

Common and usually incidental finding. Benign retention cysts within the cervix. Usually simple but may contain debris or septations. Asymptomatic



Vaginal Gartner Duct cyst

Small cyst located along the vaginal wall. Can be imaged during TA. Asymptomatic



Cesarean section scar defect / dehiscence

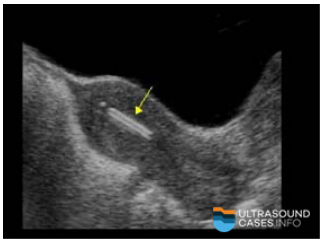
Normally the c-section scar would just show a slight indentation of the isthmus. When fluid or separation occurs, it's a defect. Dehiscence is when the myometrial walls are separating

Intrauterine Device (IUD)

Placed in uterine cavity to prevent implantation of fertilized ovum. Some IUD's release hormones to impede implantation. Usually referred to sono to confirm correct placement. Most would appear as echogenic linear echo with posterior shadowing or reverberation artifact noted centrally within endometrial cavity

Copper 7, Copper T, Mirena, Skyla, etc : all appear linear

Lippes loop : 5 equally spaced dots with posterior shadowing



Obstetrics/Gynecology Registry Review
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Endometrial Pathology

Endometrial Hyperplasia

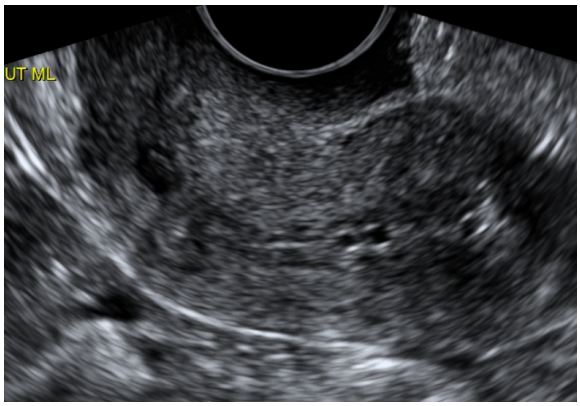
Hyper = increased -plasia = growth. Thickening of endo.

Results from *unopposed estrogen stimulation*. If estrogen thickens the endo and it's "unopposed" (not being stopped), then it will continue to thicken the endo abnormally.

May be secondary to PCOS (chronic anovulation), tamoxifen treatment, estrogen only HRT, or estrogen producing ovarian tumors.

Most likely dx in post-menopausal pt with thickened endo

Differential DX is always hyperplasia vs carcinoma (need bx to confirm)



Clinical

PMB, abnormal Ut bleeding, Hx of PCOS, HRT or Tamoxifen treatment

Sonographic

Abnormal thickening of endo (see chart)

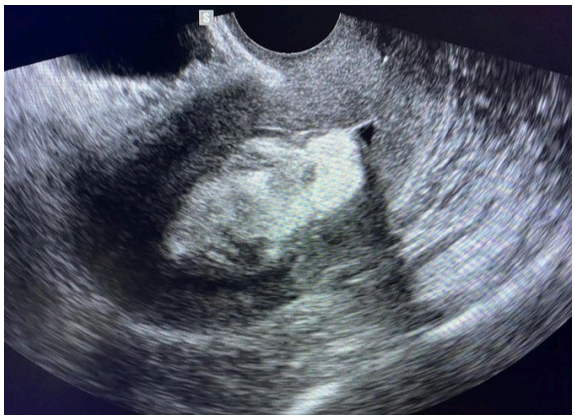
Heterogeneous with cystic changes (especially in patient with hx of tamoxifen)

Endometrial Appearances

Postmenopausel	Normal limit of endo depends on if symptomatic and Hx of HRT	
	No bleeding (asymptomatic)	≤8mm
	Bleeding (symptomatic)	≤5mm
	HRT	Variable/premenopausal appearance
Reproductive age	Normal limits depends on current menstrual phase	
	Early proliferative	4-6mm
	Late proliferative	6-10mm
	Secretory	≤16mm

Endometrial carcinoma

Most common GYN malignancy. Type of adenocarcinoma. Same associations as above. Linked with nulliparity, obesity, chronic anovulation, estrogen-producing ovarian tumors, tamoxifen, and unopposed estrogen therapy (HRT). HRT is given with progesterone to lower the risk of carcinoma.



Clinical

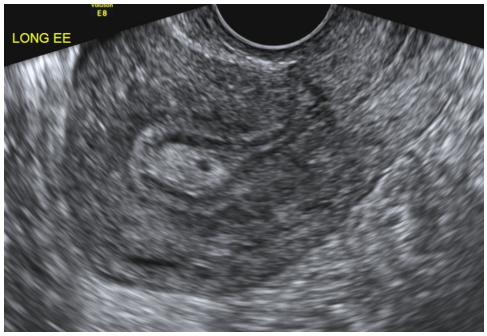
PMB, abnormal Ut bleeding, elevated CA-125

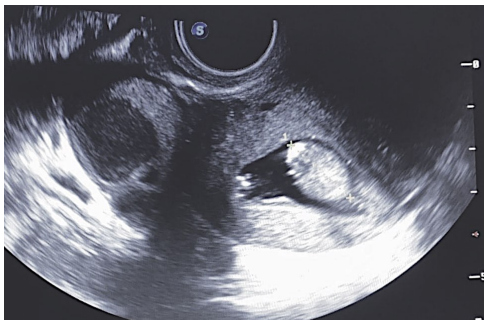
Sonographic

Abnormal thickening of endo/heterogeneous with cystic changes
Enlarged/hetero uterus
Polypoidal mass within endo with inc vascularity
Low resistance flow

Endometrial polyps

Small nodules of hyperplastic endometrial tissue. **Most likely reason for abnormal bleeding/ thick endo in reproductive age pt** (unless with DX PCOS). Saline Infused Sonohysterography is best to visualize. Most likely seen as focal or diffuse thickening of endometrium





Clinical

Intermenstrual bleeding, menometrorrhagia, infertility
May be asymptomatic

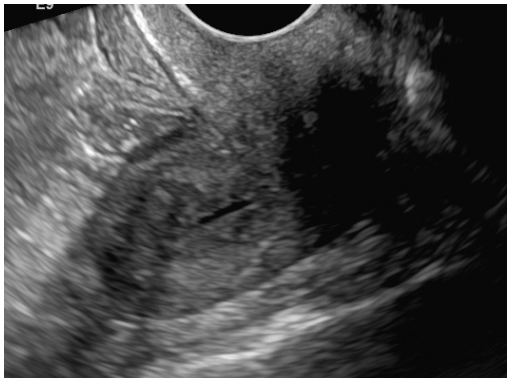
Sonographic

Single polyp: focal thickening of endo
Multiple polyps: diffuse thickening
Echogenic nodules with vascular stalk
SIS : nodules easily outlined by fluid

Endometrial atrophy

Thinning (atrophy) of endo in postmenopausel pts. **Most common cause of PMB**.

Most likely finding and diagnosis in a PMB pt with no other history



Clinical

PMB

Sonographic

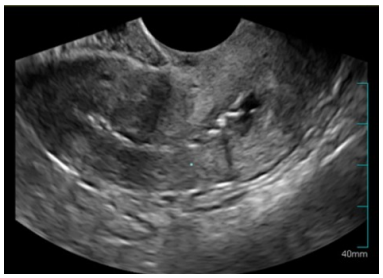
Thin endo ≤4mm

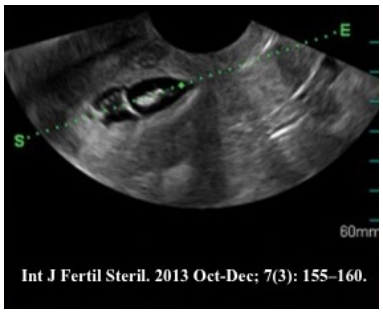
Possibly intracavitary fluid

Hyperplasia	Polyps	Atrophy
PMB / Tamoxifen / PCOS	Reproductive age AUB	PMB only
Thick endo	**Thick endo**	**Thin endo <4mm**

Asherman syndrome

Adhesions or synechiae within the uterine cavity as result of scar formation after surgery, D&C (dilatation and curettage)





Clinical

Amenorrhea or hypomenorrhea,

Hx of miscarriages or surgery

Sonographic

Thin endo with echogenic regions/scarring

On SIS - webb-like or stringy appearance and visualization of the synechiae

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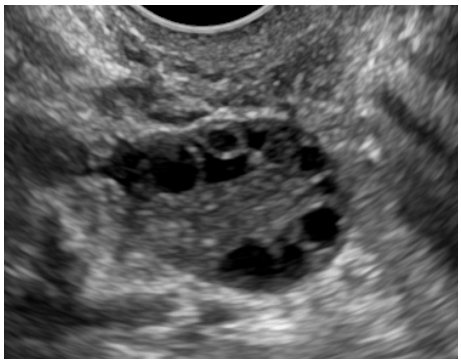
Ovarian and Adnexal Pathology

Polycystic Ovarian Disease (PCOD)

Endocrine disorder = hormonal imbalance and chronic anovulation. Menstrual cycle is unable to function normally, follicles do not mature leading to anovulation. Related clinically to Stein-Leventhal syndrome (obesity, hirsutism, amenorrhea) and therefore, infertility. **Most common cause of infertility**



Clinical

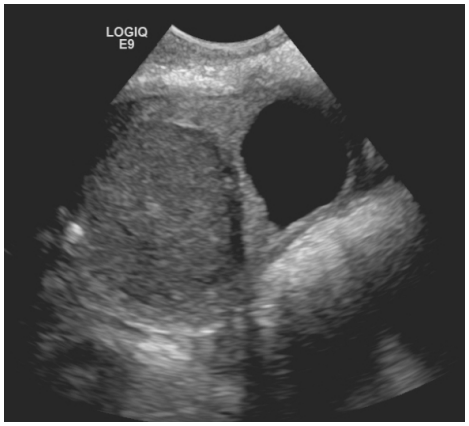
Stein-Leventhal (hirsutism, obesity, amenorrhea) or hypomenorrhea, infertility

Sonographic

Bilaterally enlarged ovaries with multiple small follicles along periphery "string of pearls"
Secondary endometrial hyperplasia

Endometriosis

Ectopic endometrial tissue outside the uterus into adnexa. Similar process as adenomyosis except OUTSIDE the uterus. The endometrial implants attach anywhere in pelvis/abdo and localize forming blood-filled cysts called endometriomas or chocolate cysts. **Most common location is the ovaries.** Infertility associated due to damage and scarring of tubes and ovaries.



Clinical

Dysmenorrhea, dyspareunia, chronic pelvic pain, painful bowel movements, infertility, nulliparity, reproductive age patients

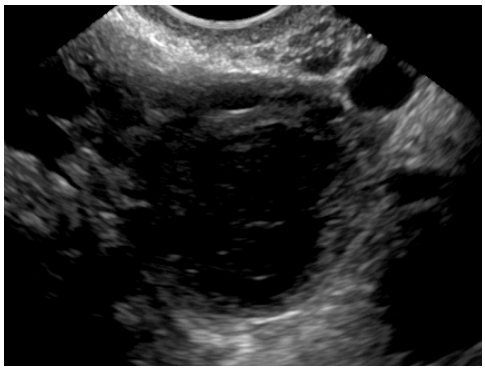
Sonographic

Cystic mass with low-level echoes, anechoic, or complex with posterior enhancement
May demonstrate fluid/fluid level

Infertility

Functional = hormone related …. PCOS
Physical = damage or blockages … endometriosis, polyps, chronic PID

Benign Focal



Clinical

May be asymptomatic, may cause cramping due to size or rupture

Sonographic

Simple - anechoic, thin, smooth walls with posterior enhancement

Hemorrhagic - complex or echogenic with post enhancement

- Follicular cysts

  Most common adnexal mass. Graafian follicle that fails to rupture and continues to enlarge >3cm. May also be due to hyperstimulation from infertility treatment such as clomid or perganol

- Corpus Luteal cyst

  Most common adnexal mass in pregnancy. Typically hemorrhagic "lacy" appearance

- Paraovarian cysts

  Located adjacent or next to ovary. Typically <2cm and asymptomatic. NOT physiologic

Theca Lutein cysts

Found only with elevated levels of hCG (>100,000) May coincides with gestational trophoblastic disease or associated with multiple gestations due to high hCG

OHSS = Ovarian Hyperstimulation Syndrome caused by hCG found in fertility treatments.

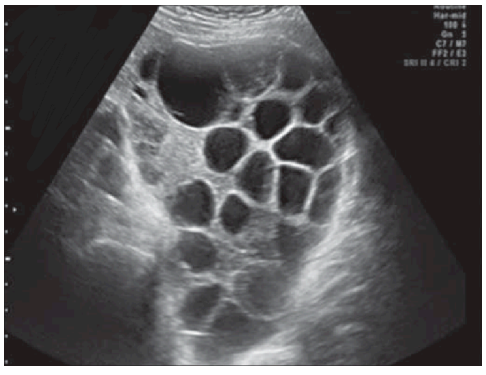
Clinical

Nausea and vomiting, high hCG, possible pain

Sonographic

Bilaterally enlarged multiloculated ovarian cysts "grape clusters"

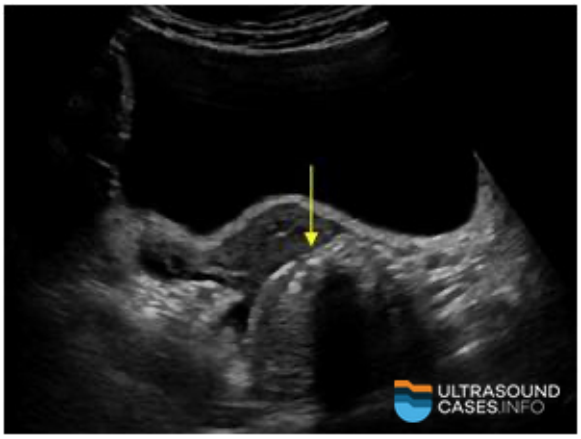
No normal ovarian parenchyma



Cystic Teratoma AKA Dermoid

Most common benign ovarian tumor. Germ cell tumor most often seen in reproductive age. Retained of unfertilized ovum that is composed of 3 layers (ectoderm, mesoderm, endoderm). May include tissues: bone, hair, fat, sebum, cartilage, teeth, etc.

Most common complication is ovarian torsion.



Clinical

Asymptomatic, palpable mass

Sonographic

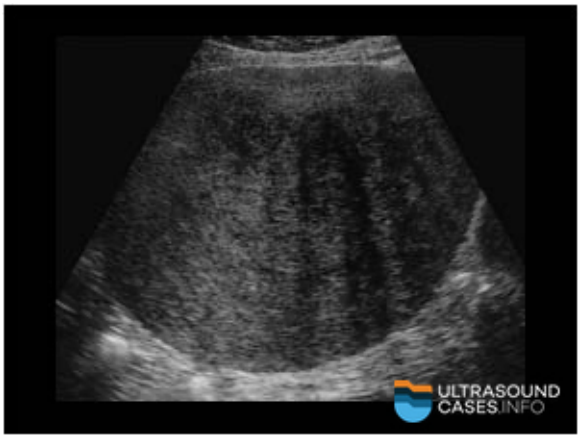
Complex, cystic, solid mass

"tip of the iceberg": post shadowing

Dermoid plug: poor thru transmission

Dermoid mesh: produced by hair, numerous linear echoes

The following solid, benign ovarian tumors most often seen in middle age and PM group. They all may present and look very similar



Clinical

Depends if estrogen producing or not

May be related to abn bleeding

Complications of Meigs

Sonographic

Most are similar: hypoechoic with poor thru transmission

Some are more complex or calc

Secondary endometrial hyperplasia (if estrogen producing)

Fibroma

Most common solid benign tumor. Most likely tumor to be associated with Meigs syndrome.
NOT associated with estrogen production. NOT related to thick endo or abnormal bleeding.
Sono: Solid, hypoechoic mass with poor through transmission.

Meigs syndrome
Ascites and **pleural effusion** in the presence of a **benign ovarian tumor**

Brenner tumor

Transitional cell tumor that are small, solid, and unilateral with calcifications.
Sono: similar to thecoma or fibroma.

Fibroma and Brenner tumor do NOT produce estrogen. NO estrogen = NO bleeding or endometrial thickening.

Estrogen producing Ovarian tumors : Thecoma and Granulosa Cell (*only these 2)*

Both are sex cord-stromal tumors, which are frequently related to hormone production. In these cases, estrogen. Estrogen thickens endo. When ovarian tumor produces estrogen = unopposed estrogen stimulation leading to endometrial hyperplasia and possibly carcinoma. Clinically associated with abnormal uterine bleeding.

Granulosa Cell tumor

Most common estrogenic tumor. Unilateral and typically postmenopausel. "Gran" = big. Grows larger and faster than thecoma. 10-15% chance of developing endometrial carcinoma due to consistent estrogen stimulation = bleeding.

- In pediatrics, cause pseudoprecocious puberty such as breast development. Malignant potential. May be solid, hypoechoic mass or complex mass. May be unpredictable in appearance

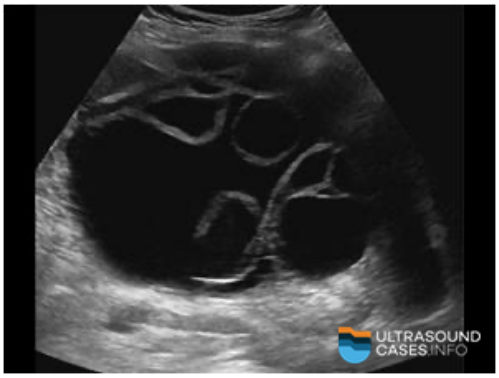
Thecoma

Most often seen in postmenopausel pt with PMB. Generally unilateral and hypoechoic.

Cystadenomas

2 types: Serous and Mucinous.

Large cystic masses with thin septations



Clinical

May be asymptomatic

Pelvic pressure and swelling due to large size

Serous

50-70% are benign

Large and typically bilateral cysts with septations.

Serous, think 'simple'= anechoic

Mucinous

Larger than serous and usually unilateral.

Septations and presence of internal debris helps to distinguish from serous.

Mucinous, think 'mucous' = debris filled

<u>Ovarian Malignancy</u>

Cystadenocarcinoma

Serous cystadenocarcinoma most common ovarian malignancy. Similar to benign forms but with more *prominent papillary projections (mural wall nodules/irregularities) and thicker septations*.

Mucinous cystadenocarcinoma will present with intraperitoneal extensions of mucin-secreting cells (like the junk inside is leaking out and forming extensions) that appear similar to complex ascites termed <u>pseudomyxoma peritonei</u>

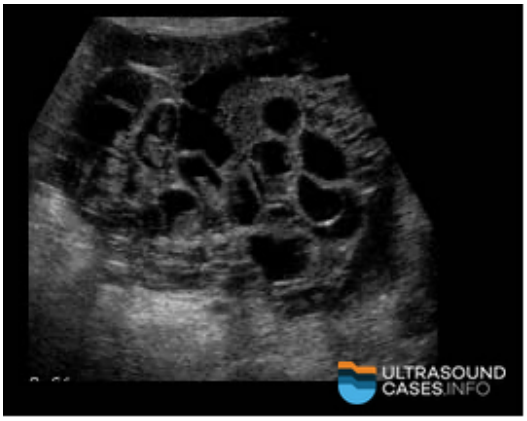
Clinical

Weight loss, pelvic pressure/swelling, abnormal bleeding, GI symptoms, elevated CA-125, acute abd pain associated with torsion or rupture

Sonographic

Cystic mass with thick septations and papillary projections with internal vascularity

Abnormal decreased resistance flow patterns



Krukenburg tumor

Metastasis from GI tract (usually stomach/gastric). Bilateral ovarian masses and ascites. Pt may be asymptomatic or may have weight loss and pelvic pain.

Sertoli-Leydig Cell tumor AKA Androblastoma

Sex-cord stromal tumor associated with virilization (development of masculine qualities and physical characteristics). Pt presents with abnormal menstruation and hirsutism. Most commonly found in women < 30 YO. May be benign or malignant.

Dysgerminoma

Most common malignant germ cell tumor. Most often seen in young pt's < 30YO and may be found in pregnancy. Children present with precocious puberty. Male version is called seminoma (testicular).
Labs: elevated **hCG** levels in non-gravid (not pregnant) female.
Tumor marker: serum lactate dehydrogenase

Yolk sac tumor AKA Endodermal Sinus Tumor

2nd most common malignant germ cell tumor. Characterized by rapid growth. Presents in females less that 20YO and elevated **AFP** in non-gravid female

<u>Ovarian Torsion</u>

Results from ovary twisting on its mesenteric connection and cutting off blood supply. Can be complete or partial since ovary has dual blood supply. **Most commonly on right side** and usually caused by ovarian mass or cyst.
***make sure doppler settings are high sensitivity = low scale/PRF and low wall filter

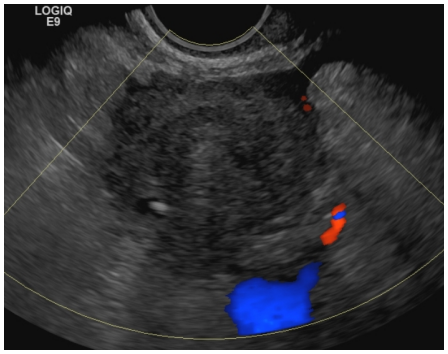
Clinical

Acute unilateral pain, nausea, vomiting

Sonographic

Enlarged heterogenous ovary with diminished or lack of blood flow

Hemorrhagic cyst or tumor may be present



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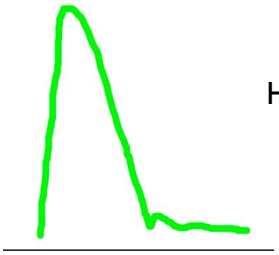
Doppler

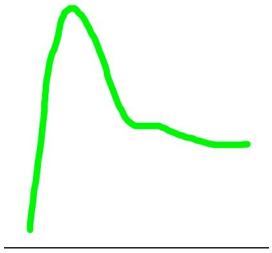
Resistance is determined by the demands of the organ. When an organ requires or wants more blood, the volume flow is increased by lowering the resistance. We can measure resistance by evaluating the change to the diastolic flow. Resistive index and pulsatility index are ways to measure resistance.



Higher Resistance

Less Diastole

Less volume flow



Lower Resistance

More Diastole

More volume flow

When to Doppler in GYN?

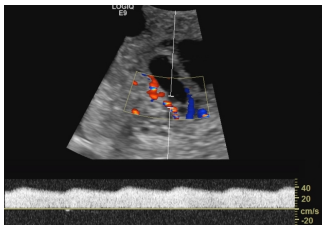
Primarily, when we suspect cancer or any abnormality that can alter the flow patterns. Malignant masses want to grow and invade; forming abnormal vascular networks = LOW resistance.

Abnormal patterns expected with Malignancy

Resistive Index (RI) <0.4

Elevated velocity >15cm/s

Absence of diastolic notch



Ovary

Normal ovarian flow is higher resistive (less diastole) during menstrual and early proliferative phases because the demands for blood are low. Resistance decreases (more diastole) at mid cycle and luteal. Most accurate time to doppler the ovary especially in the presence of a mass would be during menstrual and early proliferative phases since the ovaries would be expected to be higher resistance.

Uterus

Non-gravid uterus is normally high resistance as there is an overall low demand for blood supply. Decreased resistance waveforms can be found in cancer and in traumatic AVM.

Arteriovenous malformations

Abnormal connections between arterial and venous channels. More likely to be formed after pregnancy or miscarriage or D/C. Color Doppler will show increased "cluster" flow within myometrium, maybe into endo. On PW, the flow will be high velocity, turbulent and low resistance.

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Pelvic Inflammatory Disease (PID)

Infection of the upper genital tract. Typically starts from outside and extends internally.

Most common initial clinical presentation is vaginitis

- Risk factors: previous Hx of PID, IUD, post abortion/surgery, post childbirth, douching, multiple sexual partners, early sexual contact. **Most common cause is STD's** (chlamydia and gonorrhea).

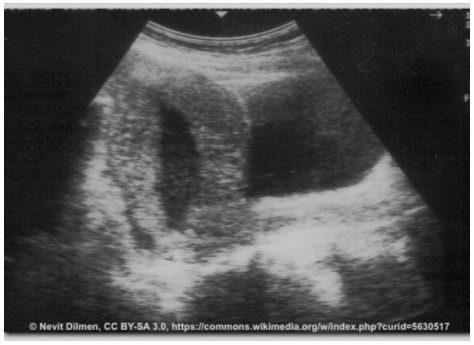
As PID progresses, may affect uterus and fallopian tubes and possibly into ovaries. Eventually causes damage and becomes chronic. All PID starts out acute and becomes chronic if organs are damaged

Difference between acute and chronic infections

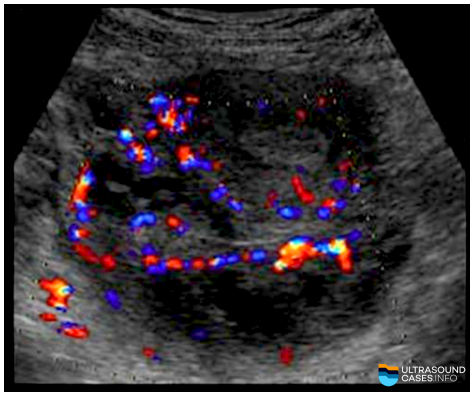
Acute *is the active infection/*inflammation. ***Chronic*** *is the damage that is done.*

Fever and leukocytosis is always related to active infection!

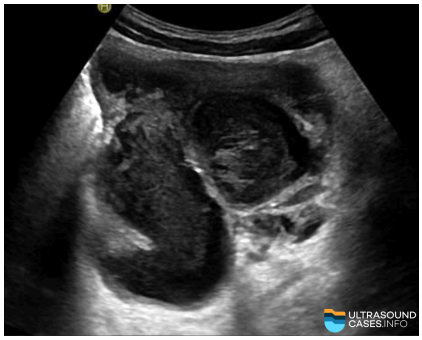
ACUTE	CHRONIC
Clinical Hx of STD, fever, chills, pelvic pain/tenderness, purulent vaginal discharge, vaginal bleeding, dyspareunia, leukocytosis	**Clinical** Chronic pelvic or abdominal pain, infertility (adhesions), palpable adnexal mass, irregular menses, vaginal discharge
Sonographic Endometritis* Pyosalpinx* or hydrosalpinx Free Fluid CDS Complex adnexal masses (TOA) * only acute related	**Sonographic** Hydrosalpinx Adhesions seen as echogenic bands within tube Complex adnexal masses (TOA)



Stage 1: early acute PID. Confined to uterus. Evidence of endometritis. Thickened endo, heterogenous, with maybe blood or pus. Comet tail/reverb artifacts classic with infection (caused by air bubbles)

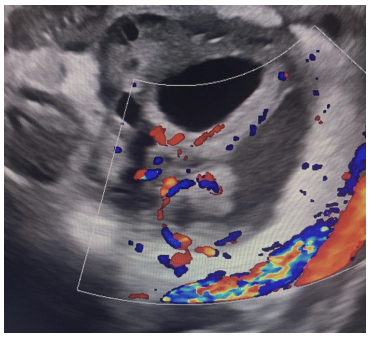


Stage 2: Spread into tubes and adnexa. Evidence of salpingitis, hydrosalpinx or pyosalpinx. Hyperemia (increased blood flow) of tube may also be documented



Stage 3: Severe progression of infection into adnexa. Bilateral complex adnexal masses known as TOA. Once it's reached this stage, it will always remain chronic

Tubo-Ovarian Complex/Abscess



PID progression into adnexa. Adhesions develop between tubes and ovaries leading to fusion (tubo-ovarian complex). Loss of discrete borders between the 2 on ultrasound making it difficult to distinguish pelvic structures. Unable to separate with TV probe pressure. Will appear as one complex adnexal mass on both sides.

Chronic findings: Hydrosalpinx and possibly bilateral TOA's depending on extent of PID

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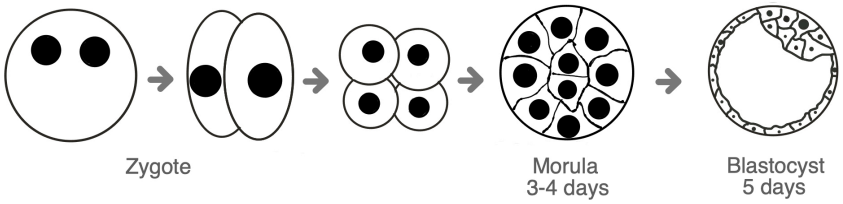
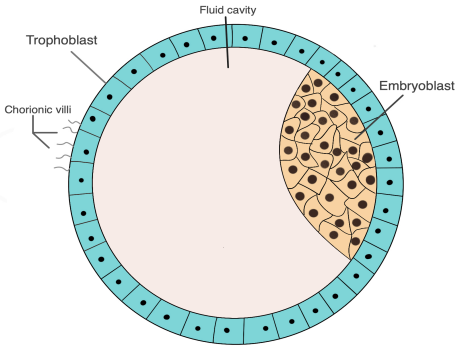
OB

Normal First Trimester Embryology and Anatomy (weeks 1-13)



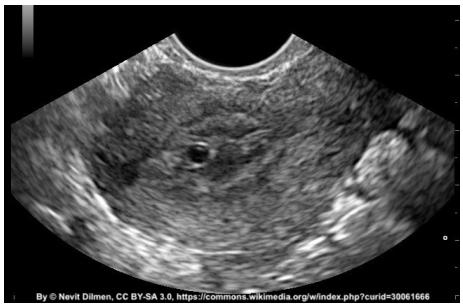
Fertilization occurs usually in ampulla, within 24 hours of ovulation (day 14 in a 28 day cycle). Once conception occurs, fertilized egg is termed zygote. The *zygote* will undergo rapid cell division in the next couple days. At 3-4 days after fertilization, m*orula* when it is a cluster of cells. By day 5, 1st time cell differentiation takes place and it is now a *blastocyst*.



BLASTOCYST:
Trophoblast: outer ring of trophoblastic cells begin to produce hCG. The hCG maintains the Corpus Luteum so that it will not regress and will keep sending progesterone to endo. Endo prepares for implantation and becomes decidualized (AKA decidual reaction). This outer layer will eventually become chorion and placenta.
Embryoblast: Inner cell mass that will develop into embryo, amnion, cord, and yolk sacs.

Implantation occurs 7-9 days after fertilization. Finger-like projections of the trophoblast called **chorionic villi** form links into decidualized endometrium. By day 28 from LMP, completed implantation. By this week, the primary yolk sac is already regressing and is replaced by secondary yolk sac. We never see the primary YS, only the secondary. **hCG levels continue to double every 48 hours until 9 weeks**

4 weeks: Before a gestational sac is seen, the endo will take on a multi-layered appearance due to the increase in progesterone. *Earliest visible sign of pregnancy is a decidualized endo, may also have tiny anechoic sac as well*



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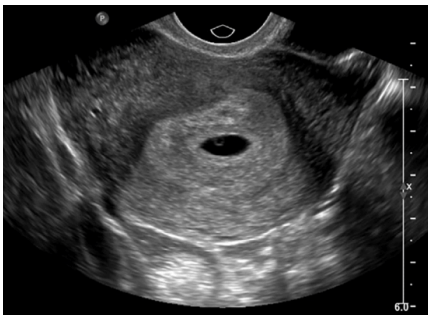
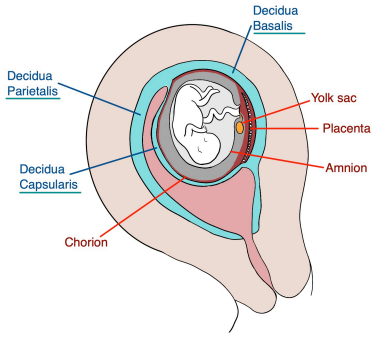


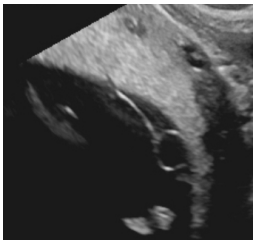


Gestational Sac

5 weeks: By **1000-2000 mIU/mL hCG**, GS should be seen TV. By 3500 mIU/mL for TA

Visualizing a double decidual sign confirms 2 layers of decidua (capsularis and parietalis) and helps to rule out pseudogestational sac

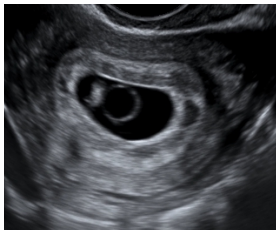
MSD (mean sac diameter): length + height + width and divided by 3. Grows 1mm per day



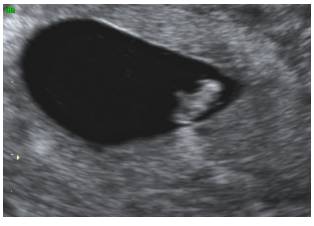
5.5 weeks: *The first definitive sonographic evidence of IUP is a gestational sac with yolk sac*. Should be vis by a MSD of 10mm. Located in the space of the chorionic cavity between amnion and chorion = **extraembryonic coelem**

Secondary yolk sac is responsible for producing AFP, angiogenesis, and hematopoiesis. Connects to embryo by the vitelline duct (1 artery and 1 vein).

- Normal appearance: round, anechoic with thin echogenic rim and measuring <6mm



Abnormal appearances indicate an abnormal pregnancy.
Enlarged yolk sac indicates impending demise as seen in image to left.
"double bleb" = large YS next to amnion (looks like 2 bubbles)



6 weeks: Fetal pole (embryo) is now seen within the amniotic cavity adjacent to YS. Must be seen by MSD of 25mm

Most reliable estimation of gestational age in 1st trimester is CRL (crown rump length). Starting at 6 weeks, FP grows 1mm per day.

- *To calculate GA from CRL : CRL (cm) + 6.5 = GA (weeks)*

Cardiac activity must be noted by 5mm CRL although it may be discernible earlier. Initially approx 100 BPM increasing to >140 later in 1st trimester.

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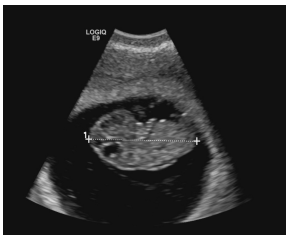
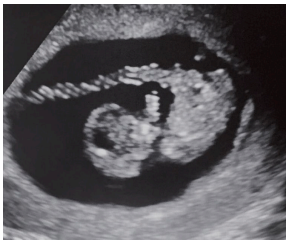
7weeks: Formation of limb buds and fetal head appears larger than body.



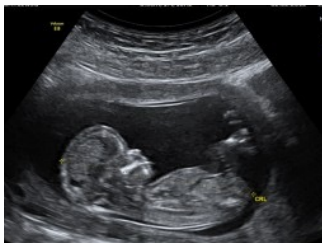
8 weeks: Cystic structure noted within head = rhombencephalon (eventually develops into 4th ventricle).

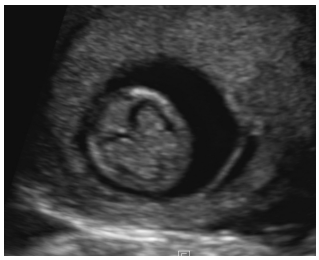
9-12 weeks: Physiologic bowel herniation is a normal migration of midgut/ bowel into the base of the umbilical cord. Should be completed by **12 weeks**.
10 weeks: Embryonic phase has ended and it now referred to as FETUS



As the fetus grows, basic anatomical structures may also be evaluated depending on age

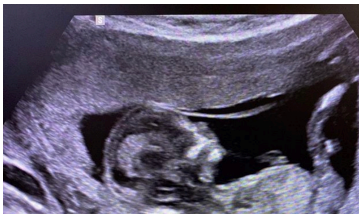
11 weeks onward: Fetal limbs and facial profile/cranium should be clearly evaluated and normalized. May also be documented: heart, stomach, cord insert, and possibly bladder.





Within the fetal head, lateral ventricles filled with echogenic choroid plexus (jelly beans) on either side of falx cerebri.

Placenta and umbilical cord: by the end of 1st trimester, seen as well-defined, crescent shaped and slightly echogenic mass of tissue. Formed by decidua basalis (maternal) and chorion frondosum (fetal).
Produces several hormones including pregnancy-associated plasma protein A (PAPP-A)



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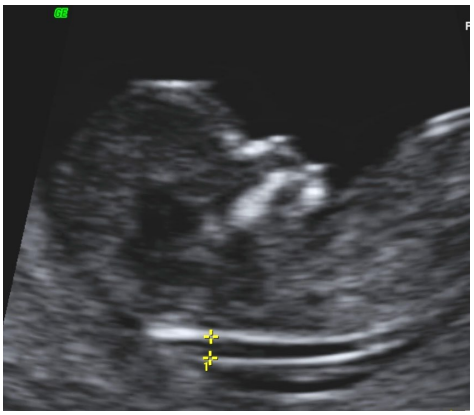
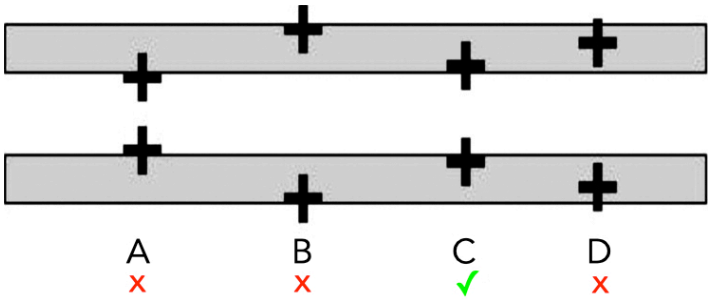
First Trimester Screening 11 - 13wks/6days

Screening tool (risk assessment) primarily for Trisomy 21 and Trisomy 18. Combines Nuchal Translucency measurement with maternal levels of hCG and PAPP-A (pregnancy-associated plasma protein A). NT is the fluid filled layer between the fetus and the skin layer and should never measure >3mm. NT may also be enlarged with Trisomy 13, Turners, and cardiac defects. Any abnormal measurement requires karyotyping



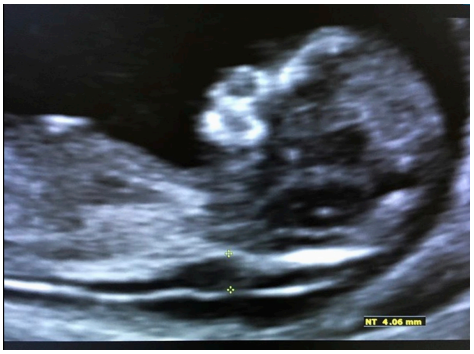
Guidelines:

- CRL between 45-84mm.
- Midsagittal view. Midline view landmarks include rectangle shaped palate and mandible, nasal bone, thalamus, and brain stem.
- Fetal head in neutral position
- Amnion noted separately from NT Area between amnion and chorion is the chorionic cavity. (Typically fuse during the first trimester, but total fusion by **16 weeks.**)
- Magnified image. Head occupies most of image
- Measured inner to inner at widest section in the back of neck when fetus is in neutral position.
- Calipers perpendicular to NT and placed along skin lines including entire fluid section but not skin.



Abnormal NT

In this image, pocket of fluid appears visibly enlarged in comparison to the above normal image



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Abnormal First Trimester

Clinical Findings	
Low hCG or S<D	**High hCG or S>D**
Incorrect dating	**Incorrect dating**
Ectopic	Multiple gestations
Non-progressive/failed preg	Gestational Trophoblastic Disease

Ectopic pregnancy

A pregnancy located anywhere other than the central uterine cavity. **Most common cause of pelvic pain with positive pregnancy test. Most common location within the ampulla of fallopian tube**.

Most dangerous: interstitial/corneal because of risk of rupture and hemorrhage.

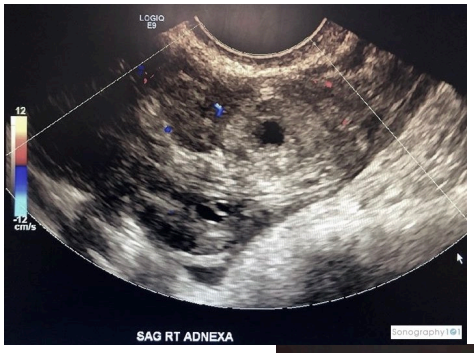
Heterotopic: Rare. IUP and coexisting ectopic, most likely associated with assisted reproduction

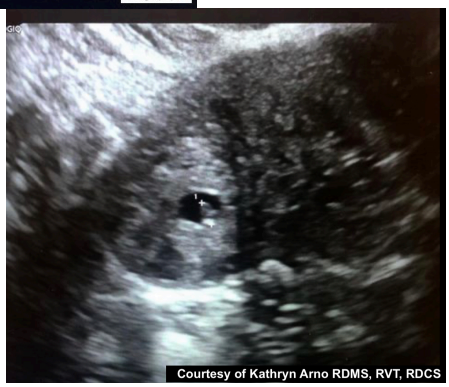
- Risk factors: assisted reproduction, Hx of PID, endometriosis, IUD, previous ectopic

hCG 1000-2000 mIU/mL on TV

Expecting to see IUP

No IUP + hCG above this level = suspect ectopic!!





Clinical

Classic triad: pain, bleeding, palpable mass

Lower than expected hCG, low hematocrit, shoulder pain

Sonographic

Extrauterine GS "live" ectopic

Complex adnexal mass or adnexal ring sign

Free or complex fluid in pelvis

Pseudogestational sac

Poor decidual reaction in endo

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Gestational Trophoblastic Disease

AKA molar pregnancy. Abnormal combination of male and female gametes resulting in rapid proliferation of trophoblastic cells (what will form the placenta). Trophoblasts produce hCG; generally there will be excessive levels of hCG or rapidly rising levels. Simply put the placenta grows out of control, takes over, and then undergoes degeneration becoming complex with cystic changes.

- Hydatidiform Mole

  Complete: **most common GTD**. Absence of fetus or gestational sac. Benign with malignant potential. Contained within myometrium, clear defined borders

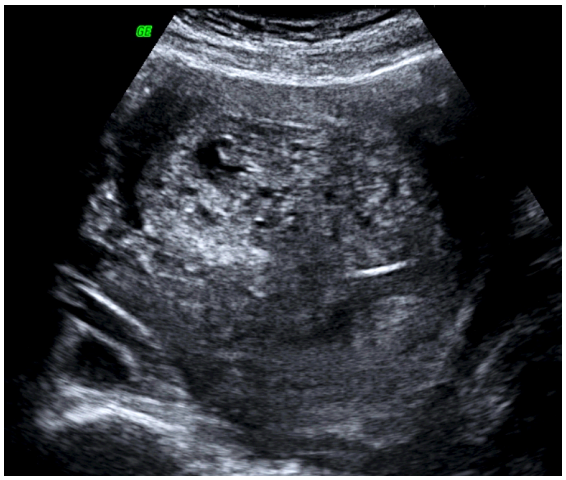
  Partial: Coexisting IUP/GS and possibly fetus. Minimal malignant potential
- Invasive molar (chorioadenoma destruens): Molar preg becomes malignant and invades into myometrium, thru UT wall and into peritoneum.
- Choriocarcinoma: Most malignant progressive form with possible mets to lung (most common), liver, and brain.

Clinical

Hyperemesis, markedly elevated hCG, bleeding, enlarged uterus, hypertension

Sonographic

Large complex mass with uterus "vesicular snowstorm" (increased blood flow)
Multiple cystic areas throughout "swiss cheese"
Loss of myometrium or borders if invasive
Bilateral theca lutein cysts



Non-progressive pregnancies / Miscarriages

When either the hCG or the sonographic appearances do not match to what is expected. LMP is not reliable, must base on hCG + sono or sono alone if no other history is known.

Sonographic Indications of Abnormal Development

- Gestational sac greater than 10 mm MSD without a visible yolk sac
- Gestational sac greater than 25 mm MSD without a fetal pole
- Enlarged YS ≥6mm
- Collapsed gestational sac/poor decidual reaction

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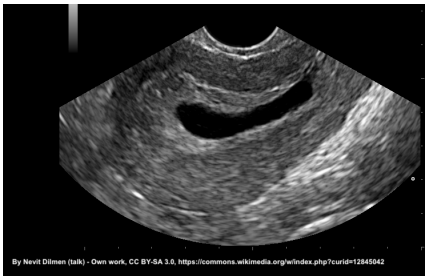


Blighted ovum or Anembryonic pregnancy

Large gestational sac without yolk sac or embryo based on sac size.

Usually shows poor decidual reaction

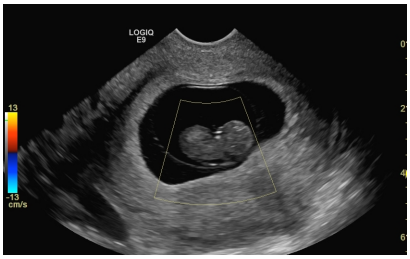
- GS >10mm + no YS
- GS >25mm + no FP

Clinical: vaginal bleeding, low b-hCG

Embryonic/fetal demise

Death of embryo or fetus. Demise is confirmed by a fetal pole ≥5mm with no cardiac activity. Irregular shaped GS, enlarged YS can indicate early demise or impending demise. Documented with lack of color in image

Clinical: bleeding, small for dates, low hCG



Miscarriages AKA Spontaneous Abortion

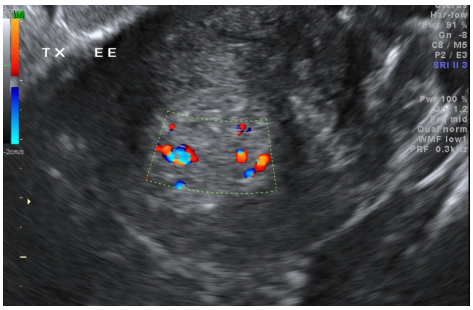
Abortion: Termination of pregnancy before viability whether elective or not. Spontaneous abortions refer to naturally occurring miscarriage. The clinical listed is the likely symptoms, but the sono findings are most important when determining what type of abortion is happening

Threatened	Missed	Incomplete*	Complete*	Inevitable
Spotting **Low FHR**	Spotting/ low hCG **Intact demise**	Heavy bleeding/+hCG **RPOC**	Bleeding/- hCG **Normal endo**	Cramping/spotting **Low lying GS**

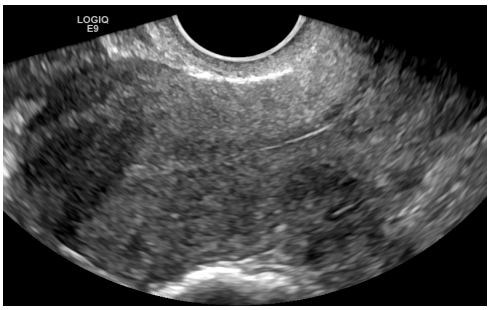
* Incomplete vs Complete

Incomplete implies the miscarriage is still in process and there are retained products of conception (RPOC) with internal flow within the cavity. Complete means it's done, cavity is empty and endo is thin, similar to early proliferative endo

Incomplete



Complete



Subchorionic hemorrhage

Bleed between endometrium and gestational sac. *Not poor prognosis*: often noted on routine sono's. Only if the hemorrhage is 50% or more the GS and close to the internal os would it be considered worrisome. Appearance depends on age of bleed, starting echogenic becoming more hypoechoic with age

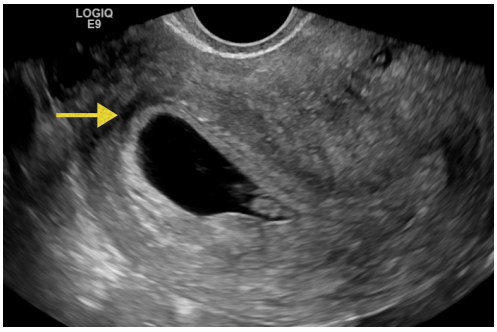
Clinical

Vaginal bleeding or spotting

Possible cramping

Sonographic

Crescent-shaped, hypoechoic or medium level echoes area adjacent to sac



Miscellaneous findings

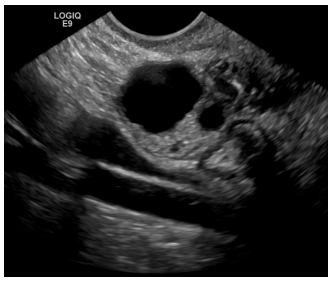
Myomas and pregnancy

Location and size are important findings to be documented as they may complicate delivery. Fibroids are stimulated by estrogen and therefore, will grow during pregnancy. Must be differentiated from uterine contraction. Contractions may appear as round masslike area within the myometrium but will disappear within 30 minutes.

Corpus Luteum of Pregnancy

Most common pelvic mass in 1st trimester. Physiologic, functional cyst that maintains endo by secreting progesterone. Corpus Luteum is maintained by hCG. Usually 2-3cm but may grow large up to 10cm. Clinical: asymptomatic or pain due to size or hemorrhage

Sono: simple or complex/hemorrhagic cyst.



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2nd Trimester (weeks 13-26)

Lab screening: 15-20 weeks. Maternal serum (blood sample from mother)

- Quadruple screen: hCG, AFP, estriol, inhibin-A
  hCG, estriol, and inhibin-A are produced by the placenta. AFP by the fetus
  "Open" or protruding fetal abnormalities will show elevated AFP.

Most common cause of abnormal serum screening is incorrect dating

Establishing Fetal Lie and Presentation

Longitudinal lie: Baby is parallel to mother

Cephalic AKA Vertex. Head presenting or closest to cervix

Breech Complete: Feet first
Frank: Buttocks closest to cervix
Footling: One leg closest to cervix

Transverse lie: fetus lie is perpendicular to mother. Head is maternal's right or left

Fetal Situs Solitus

Basically the abdominal organs and chest "match" the fetal lie

Identifying fetal lie is based mainly on the location of the head. But to confirm normal fetal situs, we need to know what the abdomen and chest should look like to compare it to the lie.

In an axial view of the fetal abdomen, the fetal lie is perpendicular to transducer position. In other words…. if we just see the abdomen like a circle, the lie is opposite the scan (transducer) plane

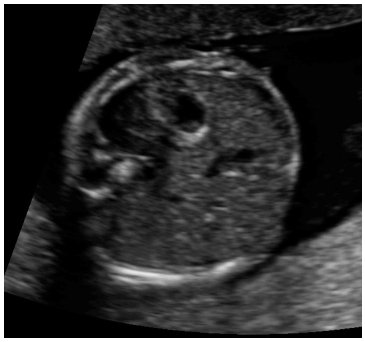
Example: If transducer is in transverse scan plane and abdomen is axial, then fetus must be longitudinal.

Follow these steps

1. Establish if fetus is transverse or longitudinal (based on scan plane)
2. Draw an arrow from the spine to the stomach
   - LONG baby and clockwise = Cephalic
   - LONG baby and counter = Breech
   - TRANS baby and clockwise = Head right
   - TRANS baby and counter = Head left

Cephalic OR Head maternal right

Breech OR Head maternal left



PRACTICE
Q - Based on this image taken from a transverse scan plane, what would be the fetal lie?

A - **Cephalic/Vertex**
Transverse scan plane means baby must be lying LONGITUDINAL
Spine to stomach is clockwise orientation = Cephalic or Vertex lie

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Fetal Heart and Circulation

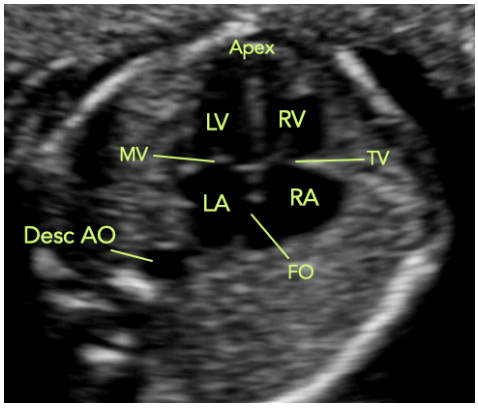
Embryology: initially 2 tubes that fuse and fold to form 4 chambers. Heart begins to contract at 36 days gestation (5 weeks) and detected on US by CRL of 5mm. NL rate 120-180 BPM
Fully formed by 10 weeks

Normal Anatomy and Imaging

Apex of heart angled to left of midline at 45° angle from spine. Occupies 1/3 of chest
Situs solitus is confirmed with imaging of fetal lie, stomach and apex pointing to left of fetus

- Levocardia: normal cardiac position
- Dextrocardia: abnormal cardiac situs, apex pointing to right chest

Confirm normal cardiac rhythm: atrial and ventricular beats need to be documented simultaneously. Either performed with M-mode through a ventricle and atrium or Doppler of inflow and outflow



4CH view

Chamber closest to spine (posterior) and desc aorta is left atrium. Chamber closest to sternum (anterior) is right ventricle. Left and right ventricles separated by interventricular septum. RV and LV should be similar in size. Lt and Rt atria are separated by atrial septum which opens at Foramen Ovale. Moderator band within RV. LA opens into LV through Mitral valve. RA opens into RV through Tricuspid valve.

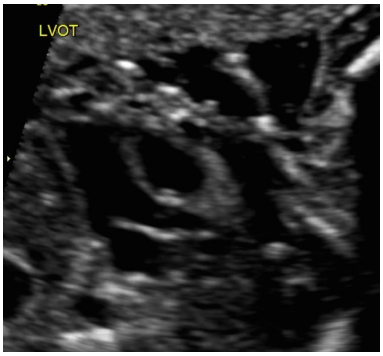
Valves are located alphabetically.

L/M = left/mitral R/T = right/tricuspid

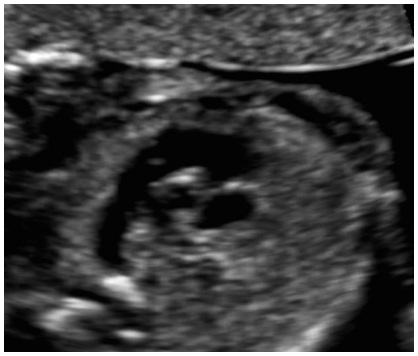
Outflow Tracts

Correct vessel placement, size, and orientation. Outflow tracts and vessels should be comparable in size. LVOT shows LV outflows only to Ao. MPA arises anterosuperior and crosses perpendicular to Ao (seen in RVOT). Superiorly at the 3VV, orientation and size can be documented

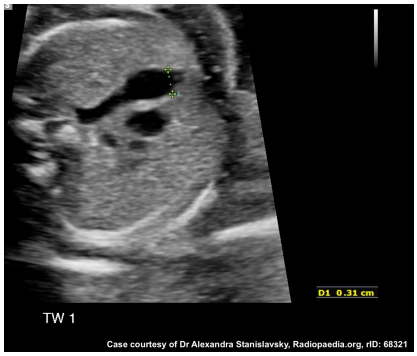
LVOT LV to Ao



RVOT RV to MPA



3VV (PA, Ao, SVC)



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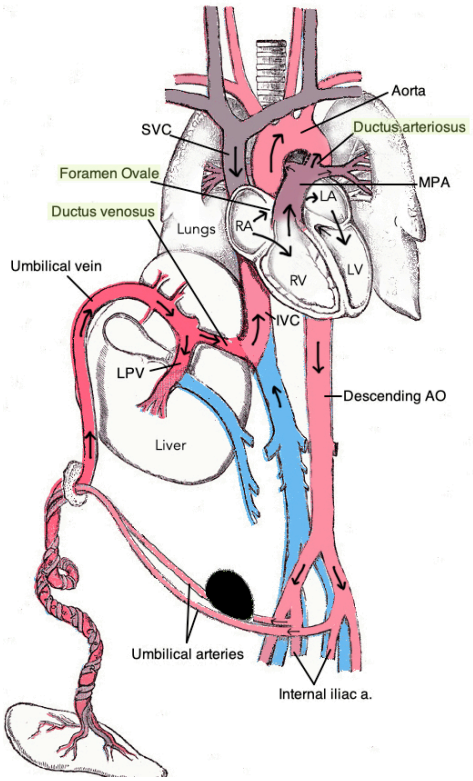
Fetal Circulation

Since oxygenated blood comes from the umbilical vein and not the lungs, it requires "bypasses" to make sure the oxygen rich blood is evenly distributed and not spent on only 1 or 2 organs. The left side of the heart and aorta supplies blood to most of the body. The goal of the shunts is to direct the flow to that side and to the aorta. 3 bypasses: *Ductus venosus, foramen ovale, and ductus arteriosus*

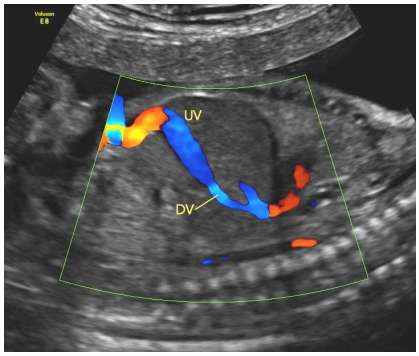
FLOW of blood: Umbilical vein carries oxygen-rich blood from placenta to fetus. Travels superiorly into liver and connects to left portal vein. Some of blood bypasses the liver via the **ductus venosus** and goes into IVC. IVC and SVC drains into Rt atrium. Some blood travels through the **foramen ovale** = into Lt atrium. The blood that continues from the right atrium through the tricuspid valve, into RV and out through MPA. From the MPA, some of the blood is shunted via the **ductus arteriosus** into the aorta to bypass the lungs. Blood returning to heart through pulmonary veins and drains into Lt atrium. Continues through mitral valve, into LV, and out through aorta. Aorta supplies the rest of body. Deoxy blood returns to placenta through umbilical arteries that branch from internal iliac arteries.

3 fetal shunts and connections

- **Ductus venosus : umbilical vein to IVC (bypasses liver)**
- **Foramen ovale: Rt atrium to Lt atrium**
- **Ductus arteriosus: MPA to aorta (bypasses lungs)**



DV



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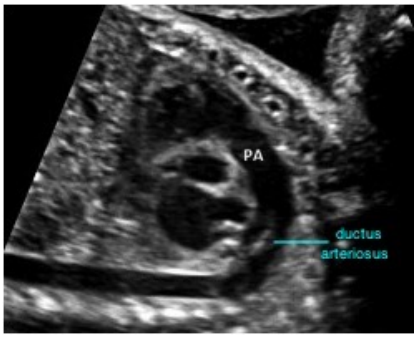

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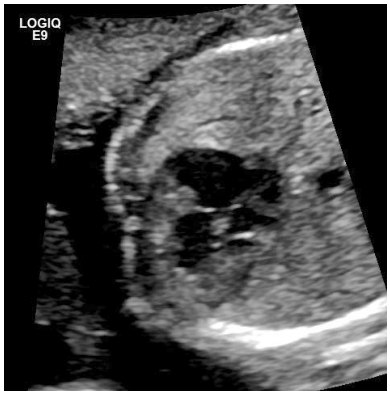
Heart Pathology

Cardiac defects can be difficult to recognize initially. Establish normal and identify anatomy by recognizing what you do know then you can identify abnormal anatomy. It should be easier to recognize what's wrong with the picture and detect pathology. Too small, missing/closed, misplaced

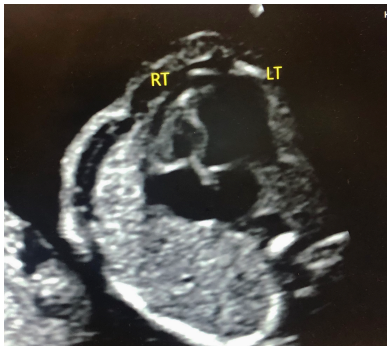
** next to name indicates previous exams have included these images*



Hypoplastic left heart *

Small or absent LV. Leading cause of cardiac death in neonate. Surgery must be performed within 1st month of life. Possible causes: aortic atresia, aortic stenosis, coarctation. Connection with T13

Cardiac view: 4CH - small/absent LV



Hypoplastic right heart *

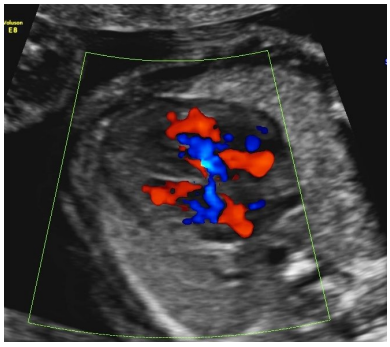
Small or absent RV. Caused from pulmonary stenosis or tricuspid atresia

Cardiac view: 4CH view - small/absent RV, enlarged LV

Add'l findings: fetal hydrops

Hydrops fetalis

Accumulation of fluid in 2 fetal body cavities. Can be caused by heart failure, tumors, syndromes, and others



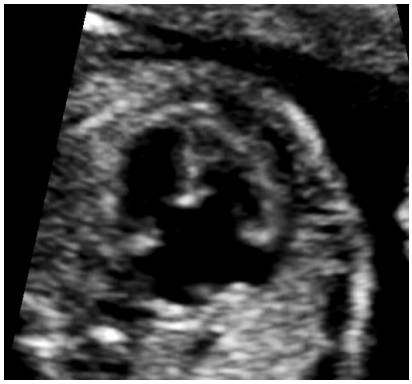
Ventricular Septal Defects (VSD) *

Opening or hole in ventricular septum. **Most common cardiac defect.** May be isolated or may be associated with other defects such as Tetrology of Fallot. Assoc with T21

Cardiac view: 4CH with beam perpendicular to IVS, showing "gap" with color flow crossing the septum

Atrial Septal Defects (ASD)

Absence of segment of atrial septum. Foramen ovale should normally be seen as a large opening in septum. But any other parts of the septum missing is ASD.



Atrioventricular Septal Defects (AVSD) *

AKA endocardial cushion defect or AV Canal defect

Lack of development of central part of heart. The endocardial cushion is where everything connects in the center: the IVS with IAS and the septal valve leaflets. Commonly associated with T21

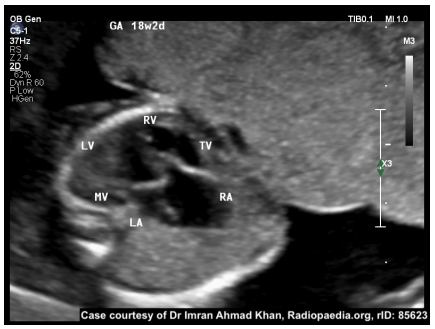
Cardiac view: 4CH- absence of atrial/ventricular septum. Lack of apical offset of MV and TV.

Ebstein Anomaly *

Tricuspid valve is incorrectly positioned apically within the RV. "atrialized" RV. Commonly associated with tricuspid regurgitation, ASDs, tetralogy of Fallot, transposition of great vessels, and coarctation. Poor prognosis

Cardiac view: 4CH- malpositioned TV, TR, enlarged RA

Associated findings: fetal hydrops (cardiac failure)



Tetralogy of Fallot

Combination of the following 4 findings. Abnormal cardiac views: 4CH and outflow tracts

- Overriding aorta
- VSD
- Pulmonary stenosis
- RV hypertrophy

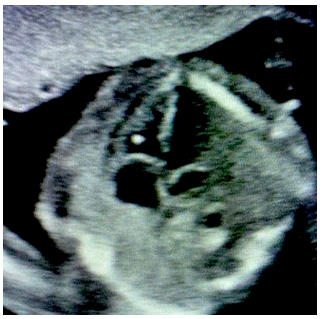
Rhabdomyoma

Most common fetal cardiac tumor. Echogenic tumors within myocardium. Associated with tuberous sclerosis (tumors on mult organs) , cardiac failure, fetal hydrops.

Echogenic Intracardiac focus (EIF) *

Calcification of papillary muscle or chordae tendonae usually seen in LV.

May be normal or **marker for T21**. Echogenicity must be comparable to bone



Syndrome Markers

"Soft markers" are findings that can be normal or may indicate a syndrome. Need to look for associated defects!

Ectopia Cordis

Heart located outside the chest through defect in sternum. Associated with Pentalogy of Cantrell (includes ectopia cordis and omphalocele). *Elevated AFP*

Transposition of great vessels

Great vessels (aorta and MPA) are switched. Aorta arises from RV and MPA from LV (outflow tract views). Would be seen running parallel to each other

Cardiac views: Outflow tracts. 4CH may be normal

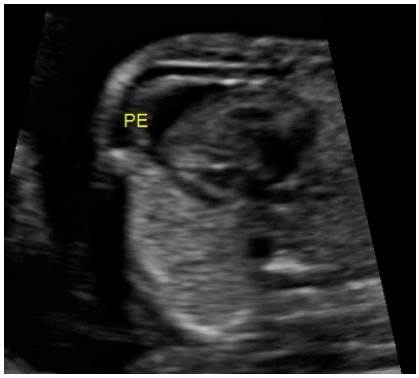
Coarctation of aorta

Narrowing of aortic arch. Commonly located between Lt subclavian artery and ductus arteriosus. Associated findings are RV and MPA enlargement.

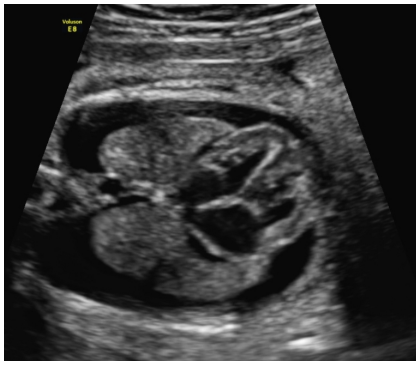
Cardiac views: Outflow tracts, sagittal aortic arch. 4CH may be normal

Pericardial effusion

Fluid located around the heart. Associated with hydrops



Chest/Lung Pathology



Pleural effusion*

Fluid surrounding lungs. Associated with hydrops or other chest pathology

"bat wing" sign = bilateral pleural effusion.

Pulmonary hypoplasia

Underdevelopment of lungs. This is not a specific sonographic finding as it can be caused by oligohydramnios, skeletal deformities, chest masses. In other words, result of other defects.

Most common cause of chest mass causing pulmonary hypoplasia is diaphragmatic hernia.

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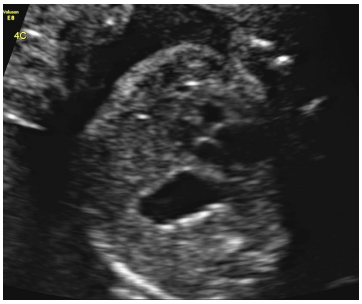
Diaphragmatic Hernia *

Most common chest mass / reason for cardiac malpositioning / chest mass causing pulmonary hypoplasia.



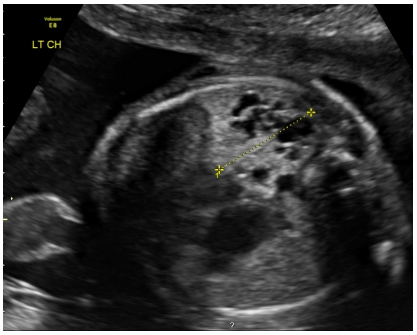
Most common on left side = Bochdalek hernia containing stomach, bowel, and left lobe of liver. No stomach will be seen in the abdomen (inferior to the diaphragm). Heart pushed to right. Stomach lateral to heart
On right = hernia through foramen of Morgagni may allow entire liver into chest. Heart pushed to left.
** On exam, may use the proper names of the hernia. Instead of left sided diaphragmatic hernia, it may say Bochdalek hernia.

Congenital Pulmonary Airway Malformation *

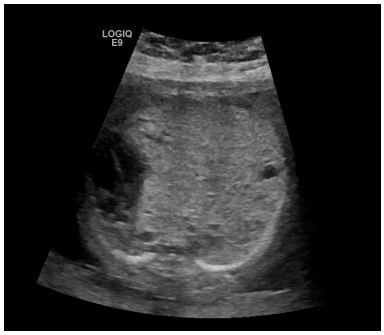
Aka Cystic Adenomatoid Malformation. Mass consisting of abnormal bronchial and lung tissue. Causes displacement of heart to contralateral side. Normally regress spontaneously or if large, can lead to hydrops or pulmonary hypoplasia
There are 3 types.

- Type 1 : Most common type. Macrocystic, large visualized cysts.
- Type 2: Mixed, cystic and solid appearing.
- Type 3: Microcystic. Cysts are too small to be vis, entire mass is echogenic. This type has similar appearance as pulmonary sequestration.

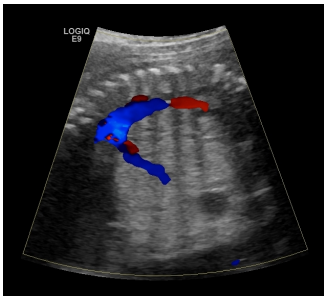
Type 1



Type 3



Pulmonary Sequestration *



Echogenic mass of nonfunctioning lung tissue with own blood supply.
Difference between CCAM and sequestration = *sequestration has its own blood supply or vascularity arise from thoracic ao*

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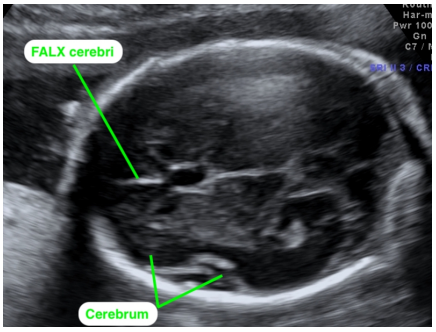
Fetal Head and Brain

Normal Anatomy and Imaging

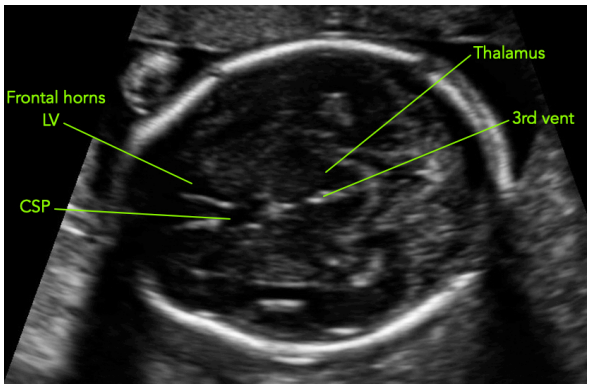
- Skull made up of 8 cranial bones connected by sutures. Foramen magnum is the opening in the base of cranium that allows passage of spinal canal.



- Cerebrum: Two hemispheres divided by interhemispheric fissure. Contains sulci and gyri. Covered by 3 layers of meninges: pia mater (inner), arachnoid (middle), dura mater (outer).

- The falx cerebri is a double fold of dura mater which separates the 2 hemispheres. Seen as an echogenic line perpendicular to beam in axial plane

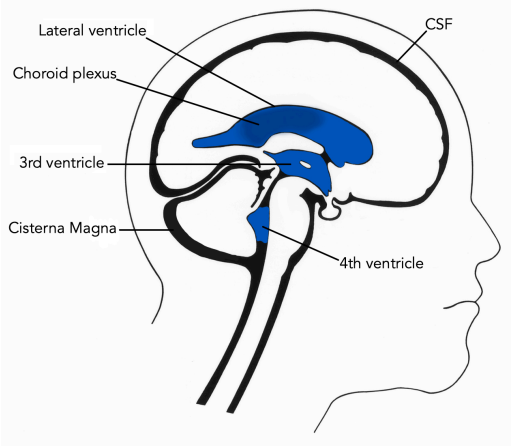
- Cavum Septum Pellucidum: Box-shaped, midline brain structure seen between frontal horns of lateral ventricles. Does not communicate with the ventricular system. Closes after 37 weeks.
  ** LANDMARK for Corpus Callosum
  Corpus Callosum: bridge between 2 hemispheres and provides communication between 2 sides



- Thalamus: 2 lobes of thalamus located on either side of falx. The massa intermedia connects the 2 lobes together and 3rd ventricle travels within. BPD and HC done at this level.

<u>Ventricular system</u>

Produces and transports CSF to cushion the brain. Lateral ventricles contain choroid plexus which produce CSF. CSF then flows to 3rd vent via foramen of Monro. *3rd vent connects to 4th vent through the* ***aqueduct of Sylvius*** (cerebral aqueduct). From the 4th vent, CSF flows through openings called foramina of Luschka and foramen of Magendie into cisterna magna and then subarachnoid space.



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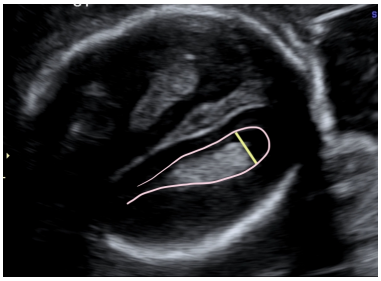


- Lateral Ventricles

  Imaged superior to level of thalamus. Evaluate for shape and size.

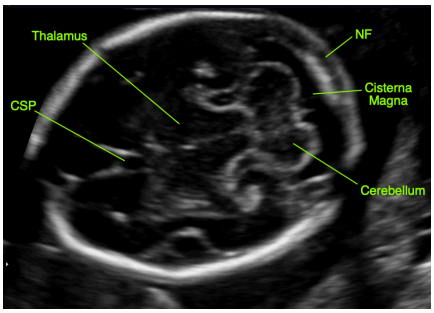
  Choroid plexus should fill most of LV.

  Measure at atrium (location of glomus of CP) Normal <10mm

Posterior fossa

Cerebellum length, cisterna magna, nuchal fold at same level

Imaging technique: From axial view of thalamus (BPD level), angle coronally or obliquely until posterior fossa comes into view. Orbits or mandible should NOT be seen in this view.



- Cerebellum: 2 lobes that are joined by cerebellar vermis forming dumbbell shape . 4th ventricle travels between (not seen normally). Normal transcerebellar size = weeks of GA. (Ie: 20 wks measures 20mm)
- Cisterna Magna: Posterior fossa of brain. Largest cistern in head. Normal 2-10mm
- Nuchal thickness/fold also measured at this level. Outer cranium to outer skin. Normal <6mm

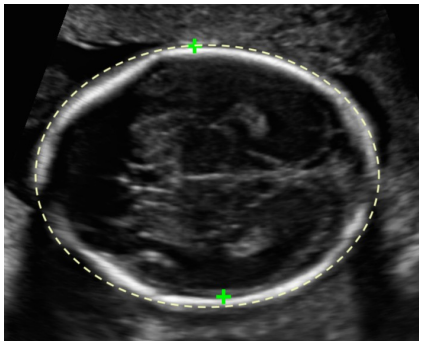
MIDLINE	NOT MIDLINE
All falxes/fissures	Lateral ventricles
Cerebellar vermis	Thalamus
Corpus callosum/ CSP	Cerebellum
3rd vent / 4th vent	Cerebrum

Head Biometry 14 weeks +

Level of thalamus. May be included: CSP, falx, 3rd vent, lat vents.

NOT including: cerebellum, cisterna magna (post fossa), orbits

HC is most accurate for gestational age in 2nd trimester



BPD Leading edge to leading edge / Outer to Inner /

Anterior margin (table) of anterior calvarial echo to anterior margin of posterior calvarial echo (top of top bone to top of bottom bone)

HC Outer circumference of cranium, no scalp included

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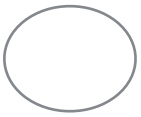
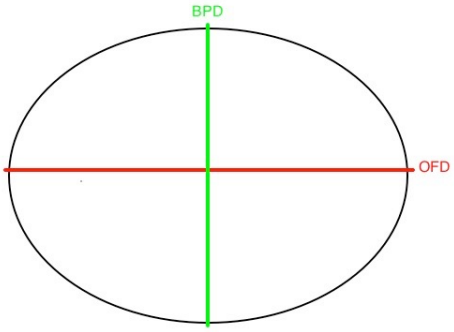
Cephalic Index

Indicates head shape. Ratio of BPD/OFD

< 0.75	Dolichocephaly Flattened	
0.75-0.85	Mesocephaly Normal	
> 0.85	Brachycephaly Circular	



Brain Pathology

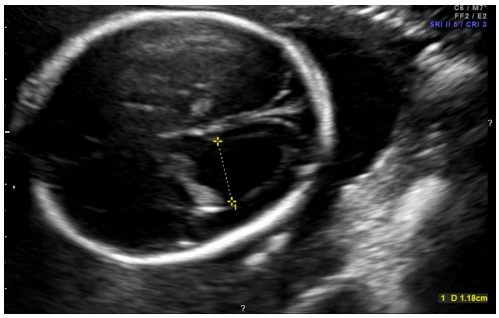
Ventriculomegaly/Hydrocephalus *

Enlargement of ventricles. **Most common brain abnormality.**

Ventriculomegaly due to obstruction is termed hydrocephaly.

Criteria: LV >10mm at atrium and "dangling choroid" sign

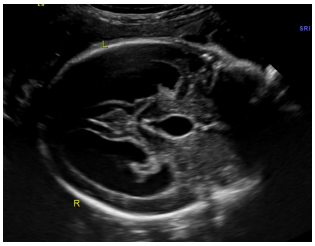
**In severe cases, look for rim of cerebral tissue



- Aqueductal stenosis *

  Most common cause of hydrocephalus. Narrowing of cerebral aqueduct (aqueduct of Sylvius) causing dilatation of 3rd and LV

  Criteria: Ventriculomegaly + dilated 3rd vent (circle in midline)

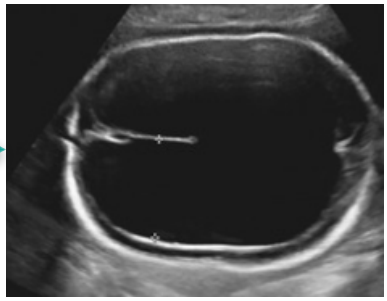


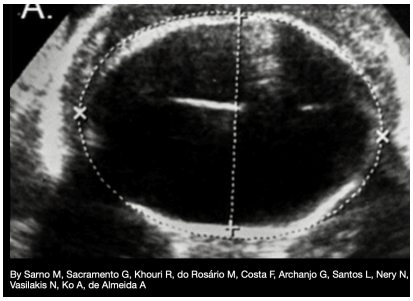
Hydranencephaly *

Hydr = fluid anencephaly = no brain… Cerebral tissue is replaced by fluid. No cerebral hemispheres, only brain stem/basal ganglia present. ** No rim of cerebral tissue seen within cranium

Hydrocephalus vs hydranencephalus??

Look for the rim of cerebral cortex!
Severe hydrocephalus will look like a fluid filled cranium but with a rim!





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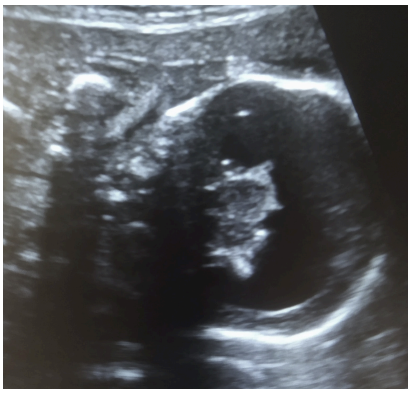
Holoprosencephaly *

Holo = whole/complete Pro = Forebrain. Varying degrees of absence of midline and fusion of non-midline brain structures starting anteriorly. Associated with midline facial defects.

Strong association with **T13 (Patau syndrome)**

3 degrees of severity: lobar, semilobar, and alobar



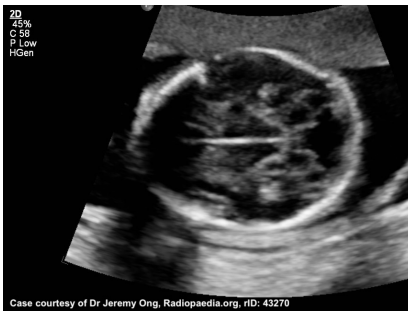
- Lobar: minimal fusion. Absent CSP/CC, fused frontal horns. Rest intact. "Heart-shape" frontal horns
- Semilobar: partial fusion LV and thalami, partial falx, absent CSP/CC. "Butterfly shaped LV"
- Alobar*: most severe form. "No lobes" = No midline separation of hemispheres. Midline brain structures are absent and non-midline are fused. Absence of CC, CSP, 3rd vent, and falx. "Horse-shoe shaped" monoventricle, fused thalami (Image left) FATAL

Associated facial defects

Cyclopia (one eye), Anopthalmia (no eyes), Hypotelorism (closely spaced eyes), Median cleft lip, Cebocephaly (1 nostril and hypotelorism),
Ethmocephaly (no nose, proboscis, hypotelorism)

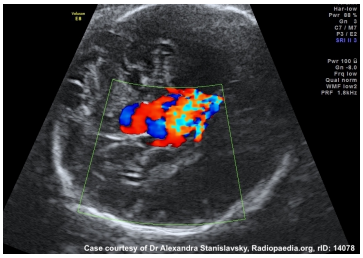
Dandy-Walker Malformation *

Absence or defect of the cerebellar vermis causing 4 ventricle to dilate and cisterna magna enlarge. Cerebellar lobes will be splayed and 4th vent between them. Often associated with other midline brain defects such as agenesis of CC, holopros, ventriculomegaly, and cephaloceles.



Criteria: Cisterna magna >10mm, dilated 4th ventricle, splayed cerebellar lobes "key-hole appearance"

- Dandy-Walker vs Mega cisterna magna
  Enlarged cisterna magna >10mm but no key-hole sign (normal 4th vent)



Vein of Galen aneurysm *

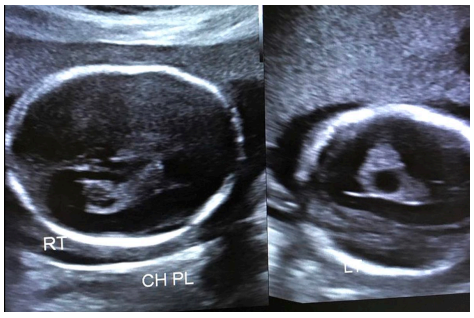
Arteriovenous Malformation. Connection between an artery and vein. Anechoic tubular mass midline brain with turbulent color flow patterns. Assoc. with CHF and **HYDROPS**

Look for the Shape of the Fluid	
Both sides with rim/dangling choroids	**Hydrocephaly**
Fluid filled cranium with no rim	**Hydranencephaly**
Horseshoe	**Alobar Holoprosencephaly**
Butterfly	**Semilobar Holoprosencephaly**
Heart frontal horns	**Lobar Holoprosencephaly**
Key-hole	**Dandy Walker Malformation**
Central tube with color	**Vein of Galen**

Agenesis of Corpus Callosum

CC and CSP are adjacent structures and develop at same time. CSP is documented to confirm presence of CC as well. If the CSP is absent, then CC is most likely absent. Associated with several anomalies such as holopros, DWM, T18, and T13 and others.

Criteria: Absent CSP, colpocephaly (splayed and teardrop shaped LV), sunburst/radial pattern sulci



Choroid Plexus cysts *

Frequently encountered on routine sonos. Usually regress by 3rd trimester. Isolated = normal fetus

Soft marker for T18 Edwards syndrome

Just know the following definitions

- Schizencephaly
  Fluid-filled clefts within cerebrum.
- Porencephaly
  Cyst that communicates with ventricular system caused by hemorrhage, ischemia, vascular occlusion
- Lissencephaly
  "smooth brain". No sulci/gyri within the cortex = Agyria. Only diagnosed 3rd tri.
- Teratoma: Most common intracranial tumor. Same appearance as other teratomas

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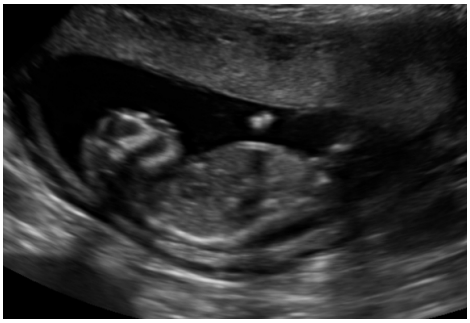
Neural Tube Defects

NTD = neural tube fails to close or form properly. Inc risk: maternal diabetes, valproic acid (seizure med), folate deficiency. ** **Supplement Folate AKA Folic acid to reduce risk**
Anencephaly and spina bifida are most common NTDs.
Open defects: *Elevated **AFP***

Acrania *

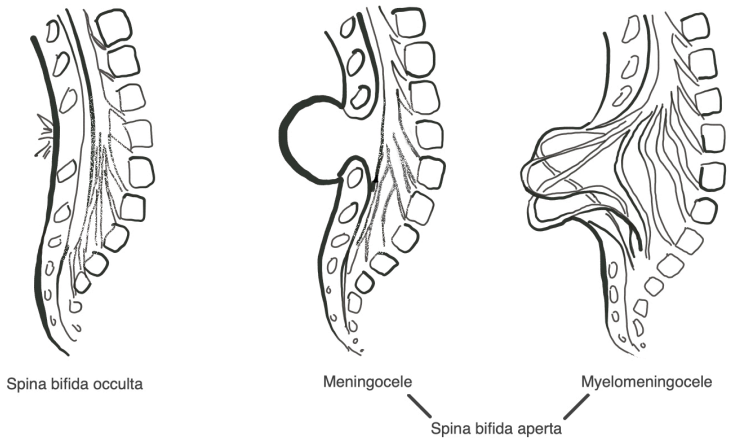
"Without cranium" Absence of cranial vault above the orbits. Can be with or without brain.
Elevated AFP.



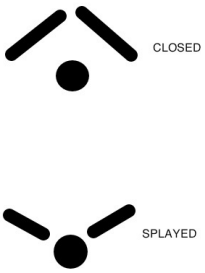
- Anencephaly * (image right) : no cerebral hemispheres.
  In Coronal view: "frog-like" bulging eyes.
- Exencephaly: normal amount of brain tissue/no skull. Will appear as misshapen head with no hyperechoic bone surrounding head

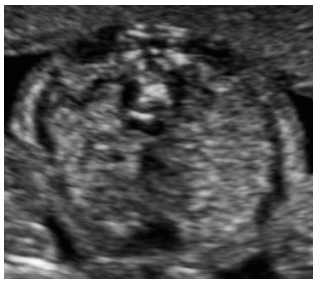
Spina Bifida / Arnold-Chiari II Malformation

Neural tube fails to close and there is a gap between the vertebrae/splaying of the vertebral laminae.
May also be referred to as spinal dysraphism or with terms meningocele/myelomeningocele.
2 types: Occulta (closed) or Aperta (open)



Spina bifida occulta (closed): Covered by skin and no herniation of spinal cord outside of body, defect in vertebrae only. Normal AFP. Postnatal: sacral dimple, lipoma, excessive hair, etc .

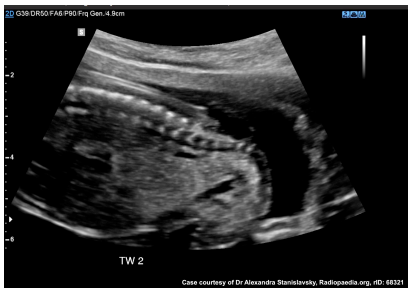




All types of spina bifida: splaying or non-fusion of vertebral laminae in transverse plane.
If open, cystic mass protruding from opening

Spina bifida aperta (open): **Most common spina bifida**. Not covered by skin and result in herniation of spinal contents (AKA cystica). *Elevated AFP.*

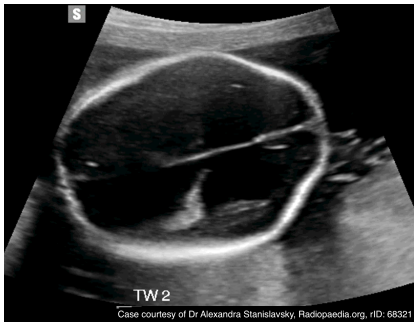
Strongly associated with Arnold-Chiari II Malformation

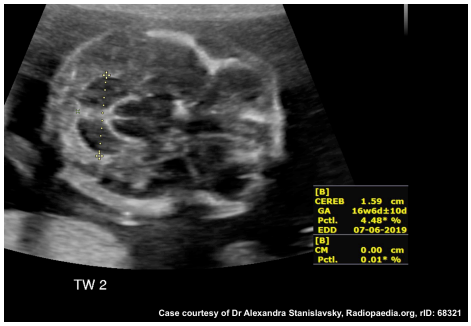


Meningoceles: contain meninges only = cystic appearance
Myelomeningocele: **MOST COMMON**. Contains meninges and nerve roots, more complex in appearance. Most commonly in the lumbosacral area.

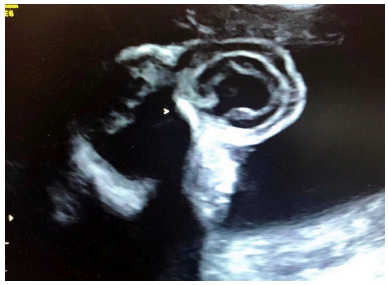
- Arnold-Chiari II Malformation *
  Presence of open spina bifida AKA meningocele or myelomeningocele pulls down on spinal contents causing cranial malformations.

Lemon shaped head
Banana cerebellum
Obliterated cisterna magna





Cephaloceles/Encephalocele
Herniation of intracranial contents through opening in skull. Most commonly occipital location. Abnormal AFP
Associated with **Meckel-Gruber** (occipital cephalocele and PKD)

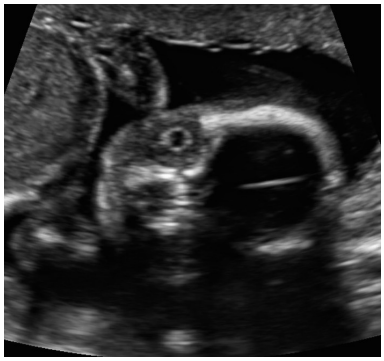


Iniencephaly
"Star-gazer". Hyperextension of neck. Closed NTD. AFP may be normal

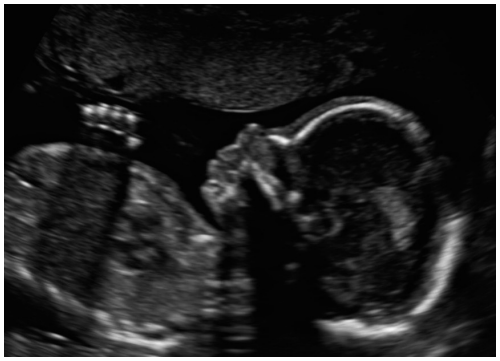
Face and Neck

Normal

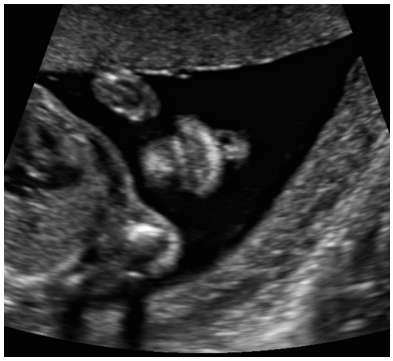
Orbit with lens (Coronal)



Profile (Mid-sagittal)

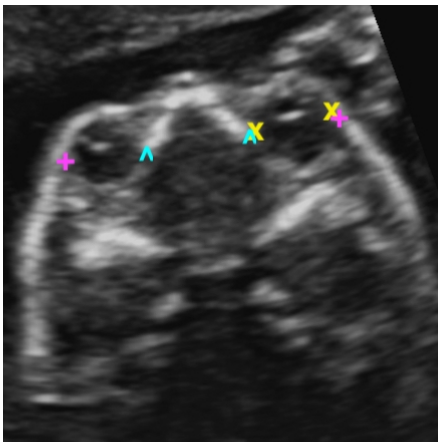


Lips,nose,chin (Coronal)



Measurements (axial plane)

- Ocular diameter: one orbit. Lateral to medial edge of orbit xx
- Interocular: between the 2 eyes. Medial (inner) sides of both ^^
- Binocular: both eyes. Lateral edges of both orbits ++



Abnormal

Orbits

Anopthalmia: no orbits **T13**

Cyclopia: one fused eye **T13**

Hypotelorism: closely spaced eyes (decreased interocular) **T13**

Hypertelorism: far apart (increased interocular) Anterior cephalocele

Micropthalmia: small orbits (decreased ocular)

- Proboscis
  Often seen with eye abnormalities. False nose/ projection replacing or above nose. **T13**

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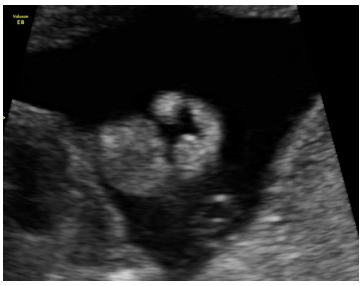
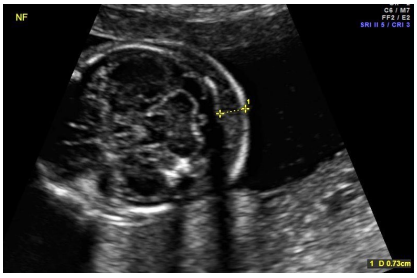
Cleft lip and palate *



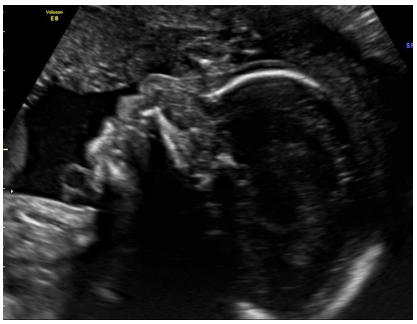
Abnormal/incomplete closure of lip and/or palate. Can be unilateral, bilateral, midline, or eccentric. May be isolated or assoc with holoprosencephaly and T13 (if midline), and amniotic band syndrome (asymmetrical)
Evaluated in coronal view of lips, nose, chin

Nuchal thickening * **T21**



≥6mm measurement of nuchal fold post neck between 18-23 weeks.
Axial view.

Flattened or Absent NB **T21**

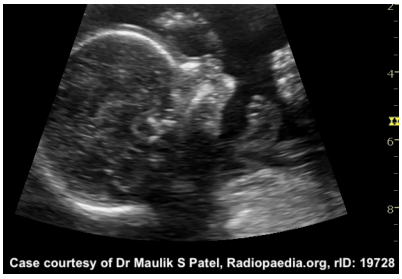


"Soft marker" for T21. May be normal in some individuals based on ethnicity
ML sagittal view

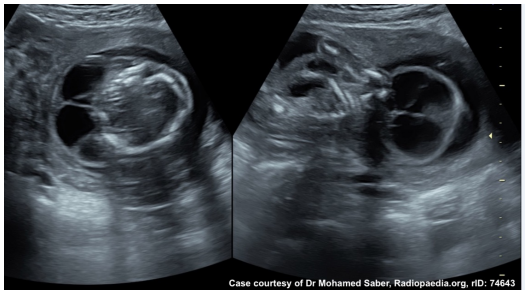
Macroglossia **T21**

Large tongue/protuberance of tongue. ML sagittal view tongue sticking out. If isolated, assoc with **Beckwith-Wiedemann syndrome**

Micrognathia **T18**



Small mandible and recessed chin.
ML sagittal view



Cystic Hygroma * **Turners syndrome**

Abnormal accumulation of lymphatic fluid under the skin. Most common within neck, but may be in axilla.
Often leads to **HYDROPS**
Sono: Cystic mass behind neck with midline septation. In SAG, "bubble appearance". In TRV, cystic with midline septations.

Musculoskeletal

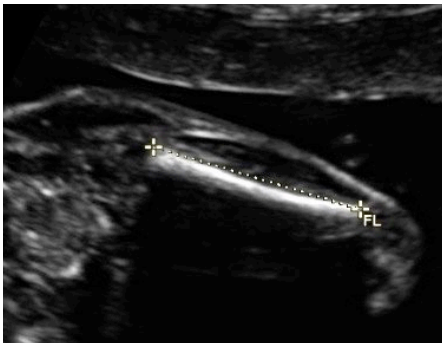
Axial skeleton: cranial and spinal bones. Appendicular skeleton: bones of the limbs and pelvis. Normal bones are hyperechoic with posterior shadowing

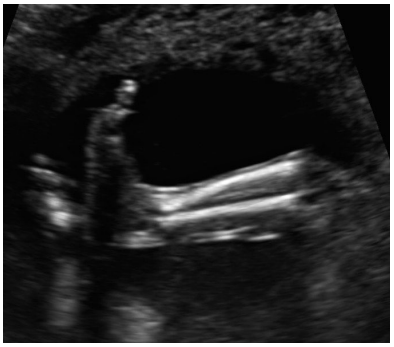
Normal Imaging and Biometry

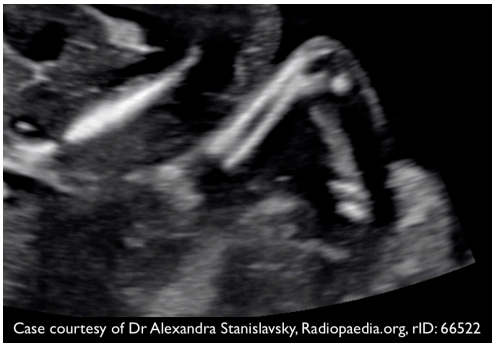
Long bones: femur and humerus (proximal limb). Sagittal plane with beam close or at perpendicular (90 degrees) to axis. Calipers should include entire shaft AKA diaphysis. The epiphysis (ends) should NOT be included in measurement.

Distal limbs:

- Leg: tibia (closer to skin and more medial in coronal view) and fibula. Normal ankle/foot relationship in sagittal to rule out club foot.
- Forearm: radius (thumbside and shorter) and ulna (pinky side, longer, goes to elbow). Hands should open and move freely







Pathology

VACTERL

AKA VATER. Association : at least 3 of these defects makes it the association. So when one of these are detected, careful examination for the others should done.

Vertebral defects
Anorectal atresia
Cardiac defects
Tracheoesophageal fistula
Esophageal atresia
Renal anomalies
Limb defects

Curvature

- Scoliosis: Abnormal curvature laterally or S-shaped spine.
- Kyphosis: Abnormal posterior curvature

Associated with amniotic band, limb body wall complex and VACTERL

Limb-Body Wall complex

AKA Body Stalk anomaly. Fatal condition caused by vascular occlusion, amnion rupture, or embryonic dygenesis. Elevated AFP (abd wall defects). Appears as if front of abdomen is stuck to placental wall due to very short or absent cord. Body wall defects, limb defects, craniofacial defects, marked scoliosis

Skeletal Dysplasias

Autosomal Dominant vs Recessive Defects
- Dominant: only one defective gene is needed
- Recessive: two abnormal genes needed (one from each parent) ALWAYS BAD in fetus

Achondroplasia "Dwarfism"

a- without *-chondro* bone/cartilage *-plasia* growth

Heterozygous achondroplasia: Autosomal dominant. **Most common non-lethal dysplasia.**

Homozygous achondroplasia: Autosomal recessive and fatal.

Findings: Macrocrania, frontal bossing (prominent forehead), trident hand (space between 3rd and 4th digits, rhizomelia (proximal limbs are much shorter than distal. (ie- femur are shorter than tib/fib)

Achondrogenesis

a- without *-chondro* bone/cartilage *-genesis* development/formation

Rare, lethal condition: absent mineralization/ossification of bones, especially noted in pelvis, spine, and cranium. NO shadowing, micromelia, fractures. Polyhydramnios

Osteogenesis imperfecta

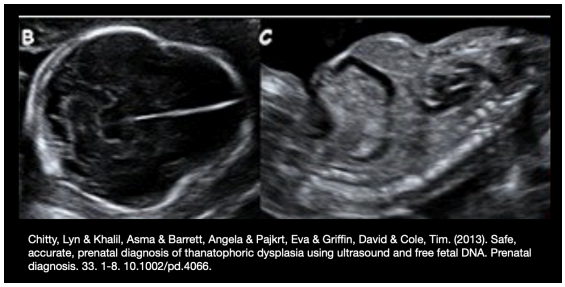
"imperfect bone formation". Brittle bone disease. Results in multiple fractures in utero and under-ossified bones. "soft skull" refers to the ability of compressing cranium with probe pressure.

Type II always fatal, most severe type. Other types are compatible with life and diagnosed after birth

Thanatophoric (death bearing) dysplasia *

Most common lethal dysplasia. Abnormal shaped bone growth. Death secondary to pulmonary hypoplasia

Findings: Cloverleaf skull* (craniosynostosis: premature fusion of the cranial sutures), bell-shaped chest, 'telephone receiver' bowed long bones..



Chitty, Lyn & Khalil, Asma & Barrett, Angela & Pajkrt, Eva & Griffin, David & Cole, Tim. (2013). Safe, accurate, prenatal diagnosis of thanatophoric dysplasia using ultrasound and free fetal DNA. Prenatal diagnosis. 33. 1-8. 10.1002/pd.4066.

Caudal regression syndrome

AKA sacral agenesis. Absence of sacrum and coccyx. Defects in lumbar spine and lower limbs.

Strong association with uncontrolled maternal (pre-gestational) diabetes/diabetes mellitus

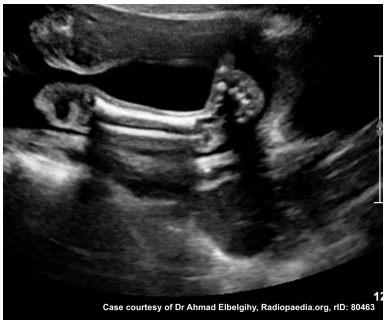
Sirenomelia

Mermaid syndrome. Fusion of lower extremities. Bilateral renal agenesis often in accompanies this disorder. In that case, fatal.

Sacrococcygeal teratoma

Germ cell tumor seen as complex mass extending posteriorly and inferiorly from distal spine. More commonly seen in females. Malignant potential. Not to be confused with myelomeningocele.
** look at posterior fossa for Arnold-Chiari

Abnormal Feet



- Clubfoot * **T13**
  AKA **Talipes equinovarus**. Medial inversion of foot. Bottom of foot and metatarsals appear in the same plane as the tibia and fibula (image right)
- Rockerbottom **T18**
  Rounded bottom of foot. Normal foot/ankle angle.
- Sandal gap **T21**
  Space between big toe and 2nd digit.

Radial ray defect / TAR syndrome Thrombocytopenia Absent Radius

Absent radius, hypoplastic ulna, missing thumb

Digital anomalies -dactyly = digits. Often related to chromosomal defects

- Poly = too many. Associated with **T13**
- Clino = clenched. Associated with **T18**
- Syn = fused. Associated with **triploidy**

Amniotic band syndrome

Sticky bands of amnion result from rupture of the amnion. Fetal parts can be caught and causes amputations and odd facial clefts. May just see the damage or may see fetal limbs stuck in the membrane

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Gastrointestinal

Amniotic fluid production and function

Fetal kidneys produce amniotic fluid. Fetus swallows fluid, passing through GI tract, where absorption takes place. If there is an obstruction or disturbance where fluid cannot be swallowed or absorbed normally, build up of fluid will take place since kidneys are still producing the fluid continually.

GI abnormalities most often result in polyhydramnios.

Polyhydramnios
- Amniotic Fluid Index AFI >24cm
- Deepest Vertical Pocket DVP >8cm

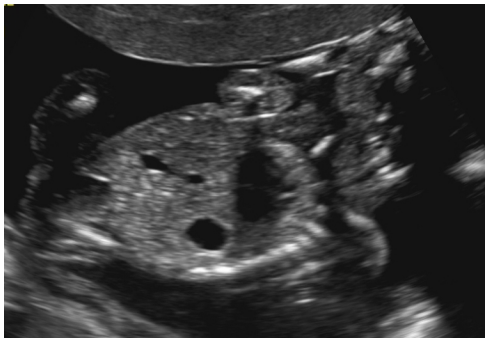
Normalizing the GI tract:

Stomach - inferior to diaphragm. Should empty or fill every 30 minutes.

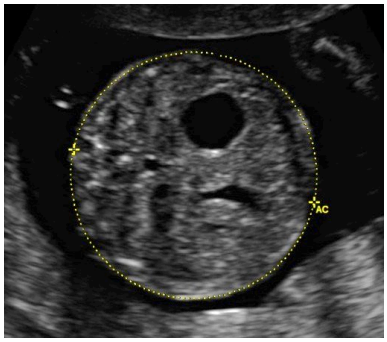
Cord insertion is just inferior to level of the kidneys, should be smooth with no herniations or mass

Bowel - slightly more echogenic than liver

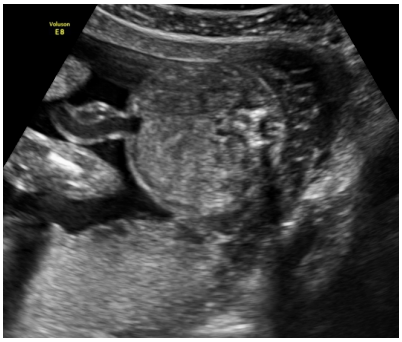
Coronal view
Stomach, Diaphragm, Bowel



Axial view
Stomach / AC



Axial view
Fetal Cord Insertion



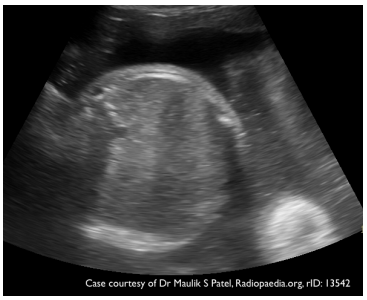
Abnormal

Esophageal atresia *

Absence of a section of the esophagus. Esophagus and trachea form a connection called tracheoesophageal fistula.

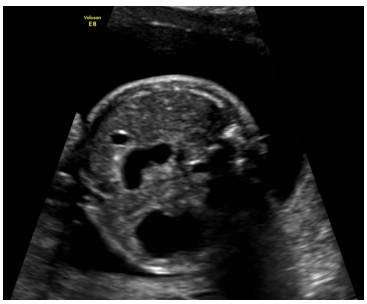
SONO: no visualization of the stomach and polyhydramnios

Associated with VACTERL and **T18**



Duodenal atresia *

"double-bubble" sign demonstrating fluid filled stomach and fluid filled proximal duodenum. Associated with **T21**. *Polyhydramnios*



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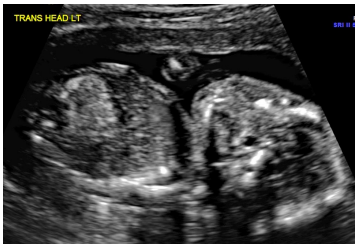
Bowel Obstructions

- Anorectal atresia
  Most common type of colonic atresia that leads to bowel obstruction.
  SONO: Dilated loops of bowel. Look for VACTERL
- Hirschsprung disease
  F*unctional* bowel obstruction caused by the absence of nerves within bowel wall.
  SONO: Dilated loops of bowel.



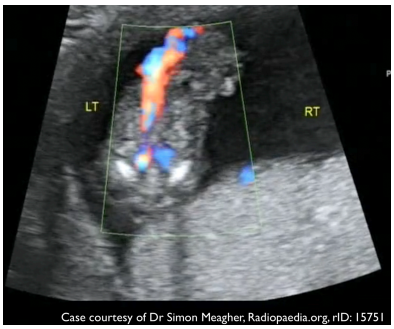
Echogenic Bowel *
Bowel bright as bone (image right). Associated with **T21** and **cystic fibrosis**

Neuroblastoma
Most common malignant abdominal mass in neonates. Adrenal gland tumor. Mass superior to kidney

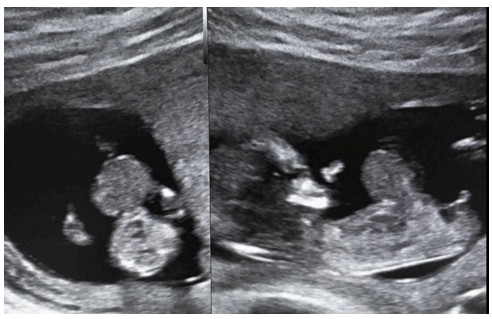
Hepatomegaly
Most common abnormality of the liver. Associated with Beckwith-Wiedemann. Will cause the AC to be larger than expected GA

Abdominal wall defects

- Gastroschisis *
  Herniation of bowel through an opening on right side of cord insertion. Not covered by a membrane or skin. Larger defects may include stomach, and other abdominal organs. Does not have strong association with chromosomal abnormalities, usually isolated finding. Only surgery to correct herniation.. *Highly elevated AFP*



- Omphalocele *
  Persistent herniation of bowel and possibly other organs into the base of the umbilical cord after 12 weeks. Bowel most likely, maybe liver too. Always midline and covered by membrane. *AFP may be normal* since it is closed.
  Very strong association with other abnormalities such as **T18**, **T13**, and Pentalogy of Cantrell as well as others.



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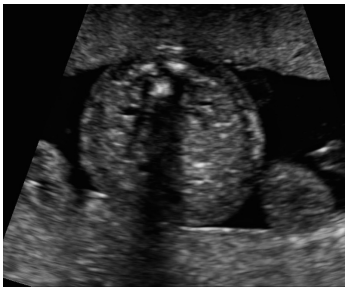
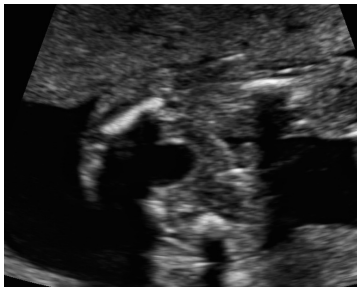
Genitourinary

Fetal kidneys form in pelvis and rise to the renal fossas by 9 wks. (If it does not rise= pelvic kidney). By 10 wks, kidneys are fully functional. 12 weeks, they produce urine. Bladder can be seen by 13 weeks, kidneys by 15. After 16 weeks, the majority of amniotic fluid is made up of fetal urine.
Renal abnormalities are most frequent cause of oligohydramnios (and only 2nd trimester onward).

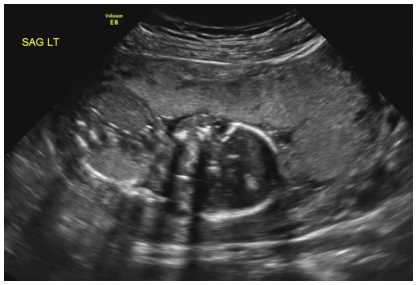
Oligohydramnios
- Amniotic Fluid Index AFI <5cm
- Deepest Vertical Pocket DVP <2cm

AXIAL **Kidneys and Bladder**



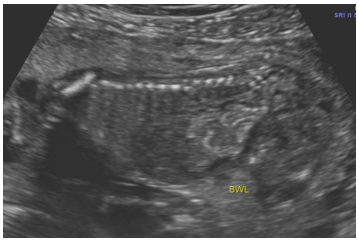


Whether or not the defect will affect the amniotic fluid levels depends on renal function.
As long as there is one functional kidney, there will be normal amniotic fluid and normally visualized bladder. Any conditions that result in bilateral non-functioning will result in no seen bladder and very low or no fluid.



ABNORMAL

Most common renal anomaly: duplicated/double collecting system divided into upper and lower moiety/poles.

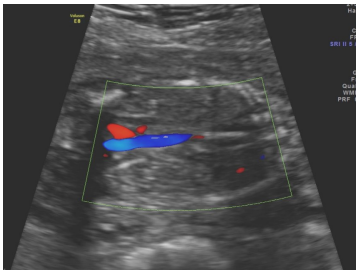


Renal agenesis*

Failure of kidney to form. Unilateral or bilateral. Usually unilateral.

Sonographic signs:

1. "lying down" adrenal sign. Adrenal gland is flattened and parallel to spine (normally sits on top of UP of kidney).
2. Absence of renal artery branches coming off the aorta in a coronal view



Unilateral: fluid and bladder will be normal since present kidney is functional.
Bilateral: NO BLADDER and NO FLUID….. *Potter's syndrome*

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Polycystic Kidney Disease

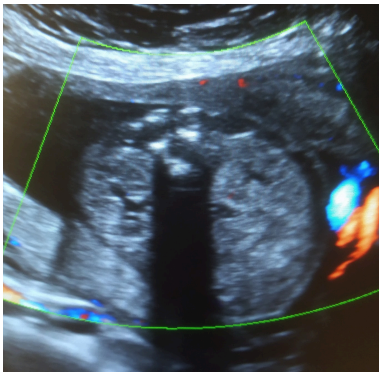
2 kinds: Autosomal Recessive PKD (infantile) and Autosomal Dominant PKD (adult). Both may have same sonographic appearances: bilaterally enlarged, echogenic kidneys. The cysts are not visualized, they are microscopic = Echogenic enlarged appearance.

Difference between the 2 types is FUNCTION.

Infantile (ARPKD) is nonfunctional = no fluid, no bladder and will be fatal. ARPKD is associated with **Meckel-Gruber syndrome (PKD and occipital encephalocele).** ADPKD- Adult version does not affect the function of the kidneys until late adulthood and it not related to other abnormalities

Infantile PKD

Bilat enlarged, echogenic kids
NON-functioning kidneys
Absent bladder
Oligo/anhydramnios
FATAL

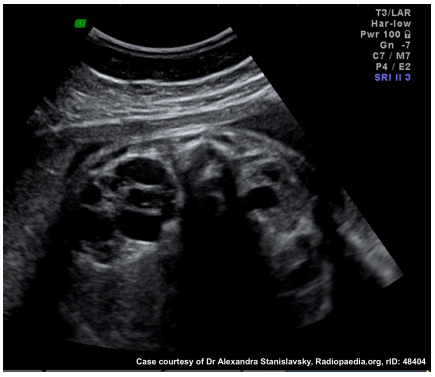


Adult PKD

May be Normal
Bilat enlarged, echogenic kids
Functioning kidneys
Normal bladder/Normal fluid
Cysts develop as adult

Multicystic dysplastic renal disease *

AKA multicystic renal dysplasia.
Can be unilateral (image right) or bilateral.
Large cysts affecting entire kidney. Kidney appears to fill abdomen.
Affected kidney is non-functional.
Bilat = no fluid/no bladder
Unilat = Normal fluid/Normal bladder



Obstructive Cystic Dysplasia

Caused by early renal obstruction. Unilateral or bilateral. Kidney becomes small and echogenic with peripheral renal cysts. Non-functional disease. If bilat = oligohydramnios.

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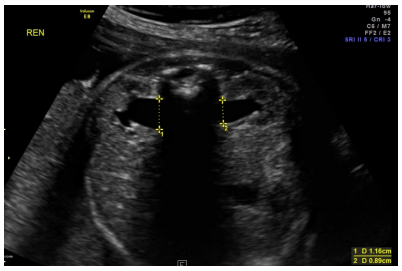
Urinary tract obstructions *

Hydronephrosis is the most common fetal abnormality.

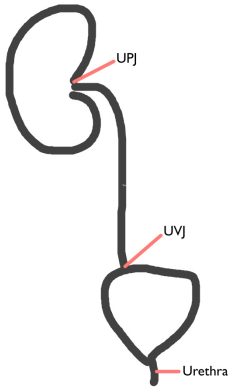
AKA pelvocaliectasis, pelviectasis/pyelectasis, caliectasis (depending on part of kidney that is dilated)

Any obstruction to normal succession of urine flow will result in back up and cause dilatation proximal to obstruction.



Dilatation of the renal pelvis is measured in transverse plane in AP dimension… Pyelectasis is considered:

- >4mm in 2nd trimester
- >7mm in 3rd trimester

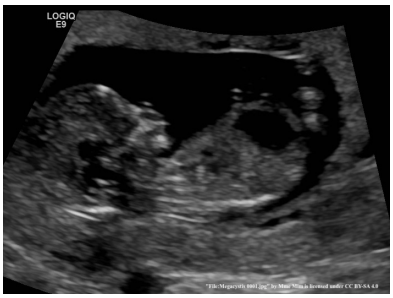


<u>Locations of obstruction:</u>

UPJ - ureteropelvic junction. **Most common cause of hydro.** Pyelectasis only

UVJ - ureterovesicular junction. Least common cause of hydro. Kidneys and ureter

Urethra - bladder outlet obstruction. Everything bladder up dilated



Bladder Outlet Obstruction/Prune Belly Syndrome

Posterior urethral valve causes bladder outlet obstruction in male fetus. Leads to massively dilated bladder. "key-hole" sign : dilated bladder (megacystis) and dil posterior urethra. Dilation of ureters and kidneys will occur eventually.

Triad of Prune Belly (AKA Eagle-Barrett syndrome) : absent abdominal musculature, undescended testis, urinary tract abnormalities.

- Bladder extrophy

Bladder located outside the pelvis. Lower abdominal mass inferior to umbilicus.

May be included with cloacal extrophy which also includes extrophy of intestines and genitalia. Cloaca is everything at the bottom including true pelvis and genitalia

- Abnormal genitalia

<u>Most common cause of ambiguous genitalia is a female with clitoromegaly</u>

Other options in a male: micropenis, hypospadias (abnormal curvature of penis), undescended testis

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Chromosomal Abnormalities and Syndromes

Terminology

Advanced maternal age: 35yo or older at EDD
Karyotyping: analysis of fetal chromosomes
Sequence: single defect leading to development of other defects
Syndrome: group of clinically observable findings that often exist together
Association: nonrandom occurrence of at least 3 associated defects
Anomaly: any structural feature that is abnormal

Aneuploid: any abnormal # of chromosomes
Euploid: normal chromosomes
Diploid: two complete sets of chromosomes. Humans have 23 pairs = 46 chromosomes
Triploid: three complete sets = 69 chromosomes
Monosomy: only one of an individual chromosome. Miss one = 45 chromosomes
Trisomy: three copies of one individual chromosome. One extra = 47 chromosomes
Mosaic: mixed pattern aneuploid

Fetal Karyotyping *Analysis of Chromosomes*

Probe to needle angle should be as close as 90 degrees for constant visualization. Needle will be hyperechoic with reverberation

• Chorionic Villi Sampling

Samples placenta for aspiration of trophoblastic cells. Earliest procedure done TA or TV (depending on placental location) between 10-12 weeks. Fetal loss rate 0.8%

• Amniocentesis

Samples amniotic fluid. 15 weeks onward. US guided TA. Prior to procedure, identify MVP (max vertical pocket), placental position, and fetal position. Fetal loss rate 0.5%

• Cordocentesis

Percutaneous Umbilical Cord Sampling (PUBS). *Samples fetal blood.* After 17weeks. US guidance, samples through umbilical cord near insertion into placenta. Fetal loss rate 0.1%

Trisomy 21 Down's syndrome

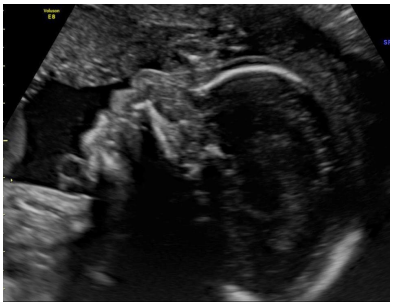
Most common aneuploid. 1 in 800 births.

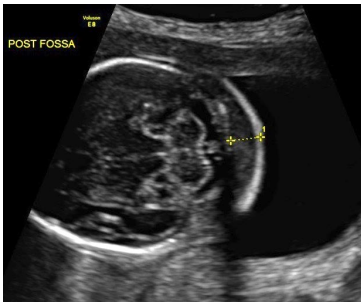
Risk increases greatly with increased maternal age.

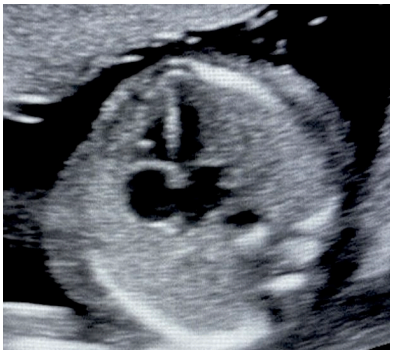
Screening Findings

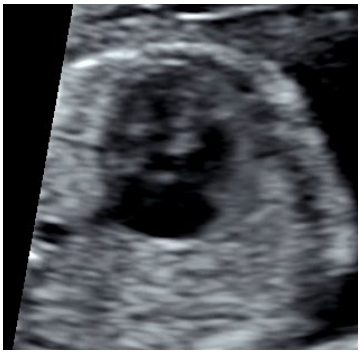
Elevated hCG and inhibin-A

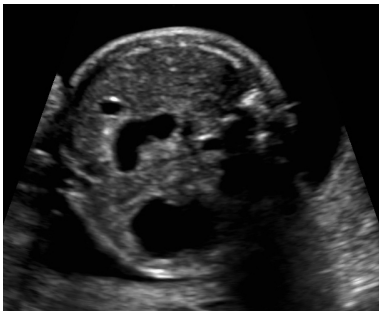
Low AFP, estriol, and PAPP-A

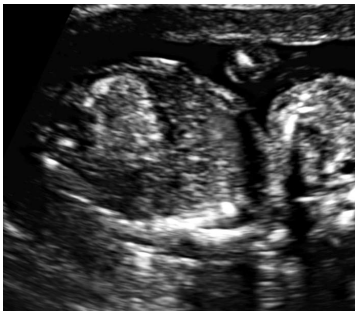










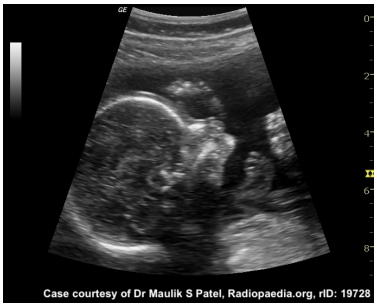


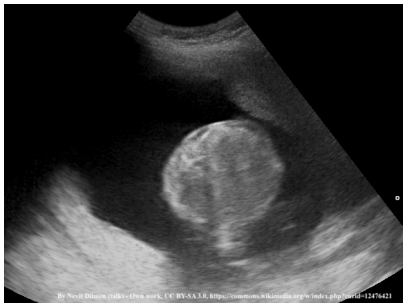
Sonographic Findings

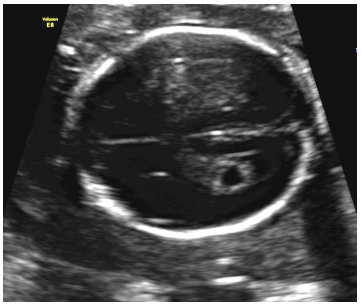
- Absent nasal bone
- Flattened profile
- Thickened NF
- Macroglossia
- Echogenic intracardiac focus EIF
- Cardiac defects: AV Canal (AVSD)
- Duodenal atresia (double-bubble)
- Echogenic bowel (can also be cystic fibrosis)
- Short limbs
- Sandal gap feet

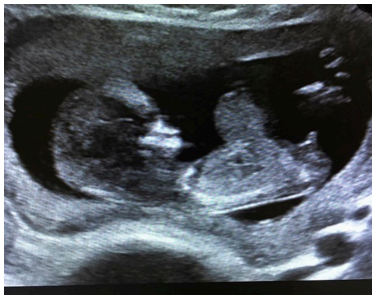
Trisomy 18 Edwards syndrome

Usually fatal









Screening Findings

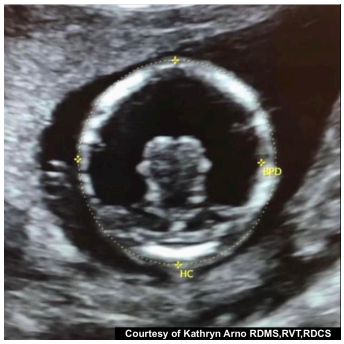
All values decreased

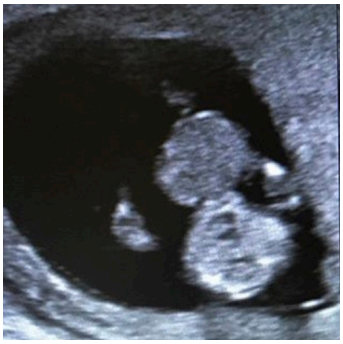
Sonographic Findings

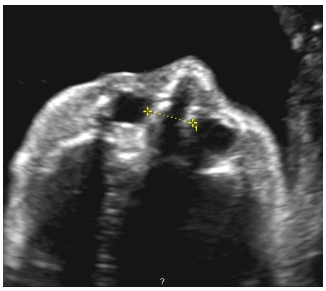
- Choroid plexus cysts
- Strawberry-skull
- Micrognathia
- Omphalocele
- Esophageal atresia
- Clenched hands/clinodactyly
- Rockerbottom feet
- Cardiac defects

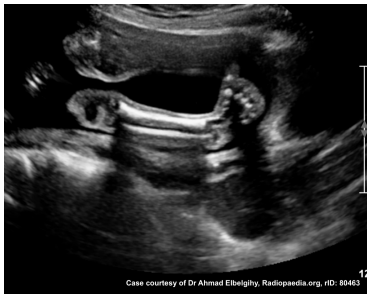
Trisomy 13 Patau's syndrome

Usually fatal









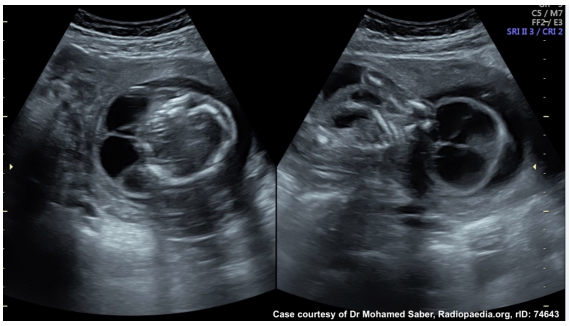
Screening Findings

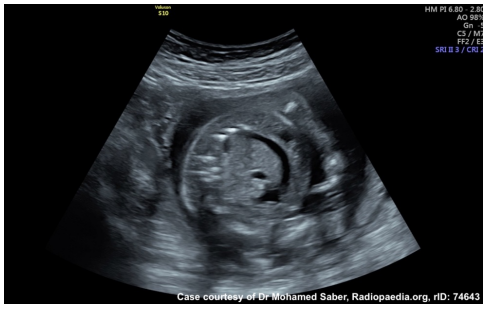
Non-specific

Sonographic Findings

- Holoprosencephaly
- Facial abnormalities
- Microcephaly
- Polydactyly
- Omphalocele
- Cardiac defects (Hypoplastic Lt heart)
- Clubfeet - talipes equinovarus

Monosomy X Turner's syndrome





Screening Findings

Low AFP and estriol

With Hydrops: Low hCG and inhibin A

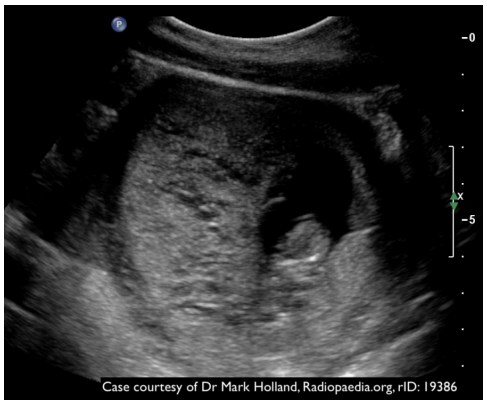
Low PAPP-A

Sonographic Findings

- Females
- Cystic hygroma
- Increased NT
- Renal anomalies (horseshoe/agenesis)
- Cardiac defects
- Nonimmune hydrops

Triploidy

3 sets with total of 69 chromosomes. Usually fatal early.



Screening Findings

High hCG (with molar)

Sonographic Findings

- Partial molar
- Small, low set ears
- Syndactyly (fused digits)
- IUGR
- Cardiac defects
- Theca-Lutein cysts

Additional Fetal Syndromes (all of the following are euploids)

Meckel-Gruber	PKD, encephalocele, microcephaly, polydactyly
Potters	Bilateral renal agenesis, oligo, pulmonary hypoplasia, facial
Beckwith-Wiedemann	Macroglossia, large organs
Pentalogy of Cantrell	Omphalocele, ectopia cordis, sternal and diaphragm defects
VACTERL	Vertebral, Anorectal, Cardiac, Tracheo, Esophageal, Renal, Limb
Amniotic band	Limb amputations, facial clefts, gastroschisis, skeletal defects
Limb Body Wall	Abdominal wall defects, scoliosis, facial, and limb defects
Fetal Alcohol	IUGR, microcephaly, microophthalmos, cardiac defects, hypospadias

The following are less common. Know the basic associations

Kleebattschadel
Cloverleaf skull due to craniosynostosis (premature fusion of sutures)

Holt-Oram / Heart-Arm
Heart and upper extremity malformations

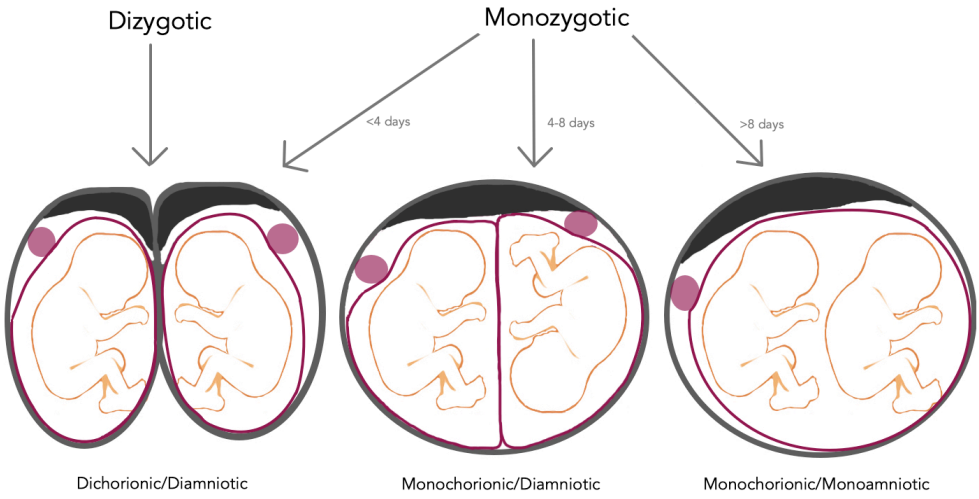
Treacher-Collins / Collar
Ears, mandible, and palate malformations

Multiple Gestations

Complications: Increased risk of preeclampsia, preterm delivery, low birth weight, fetal anomalies, miscarriage, and perinatal death.
Median gestational age of delivery for twins = 36 weeks. Multiple gestations greater than twins are most likely the result of assisted reproduction and have increased risk of complications.

Twinning

Either arise from 2 separate eggs that were fertilized (**dizygotic**) or 1 zygote that splits (**monozygotic**)
Dizygotic is most common: fraternal since they come from separate ovum.



Chorionicity
Chorion forms the gestational sac and placenta. Dichorionic means two of each. Mono means one

Amnionicity
Amnion is the inner membrane and goes with yolk sac.
Diamniotic means there is a membrane between and 2 yolk sacs. Mono means it's all shared

Dizygotic will *always* be dichorionic/diamniotic. That means everything separate and nothing is shared. 2 gestational sacs, 2 chorions, 2 placentas, 2 amnions, 2 yolk sacs

Monozygotic results from single ovum that splits, identical. Time of cleavage or division will determine chorionicity/amnionicity. The earlier the split, the more divided they will be. They can be any of the 3 combinations

Early division <4 days results in dichorionic/diamniotic. 2/2/2/2

Most common division 4-8 days results in monochorionic/diamniotic. 1 single gestational sac, 1 chorion, 1 placenta, 2 amnions, 2 yolk sacs.

Late division >8 days results in monochorionic/monoamniotic. Everything is shared. 1 GS, 1 chorion, 1 placenta, 1 amnion, 1 yolk sac

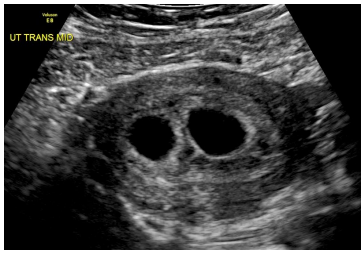
Determining type of twinning

1st trimester

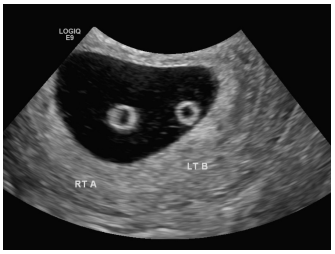
Dichorionic can be seen as two completely separate gestational sacs within the uterine cavity. Dichorionic must also be diamniotic. (Di/Di)

Monochorionic will appear as 1 gestational sac. Count the amnions or yolk sacs to determine amnionicity

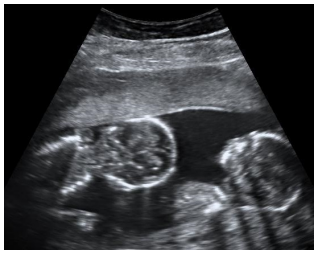
Di/Di



Mono/Di



Mono/Mono

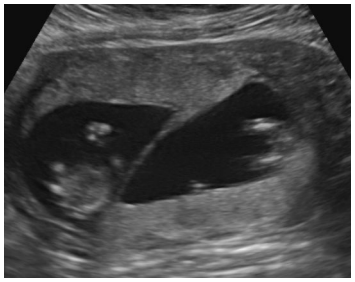


2nd trimester assessment

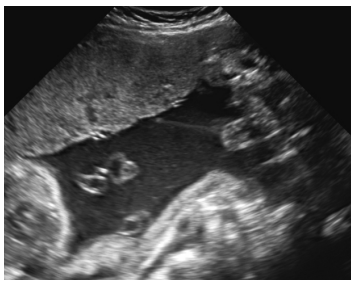
Dichorionic is more obvious if 2 separate placentas are identified.

But the 2 placentas may appear as 1 fused placenta. Most important to look at the junction of the membrane to the placenta

Look for **Twin peak (lambda or delta sign) = Di/Di**



Monochorionic/diamniotic will be seen as thin membrane inserting like a T into placenta which means one shared placenta **T sign**



Twin Complications : monochorionic twins only

Why monochorionic twins only? Monochorionic share one placenta so there's increased risk of fetal shunting and growth issues

Twin to Twin Transfusion Syndrome TTTS *

Fetal shunting through vessels in the placenta. Donor to recipient. Donor twin gives blood to other, eventually suffering from IUGR and anemia. Recipient twin receives too much blood and can suffer from hydrops and CHF due to the overload of blood going to the heart.

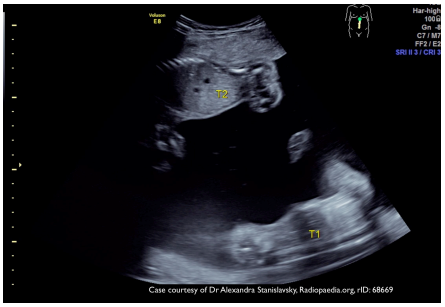
1st initial sonographic indication is discordant fetal growth.

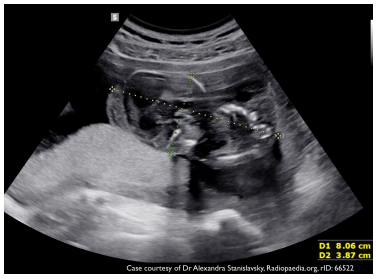
Donor	Recipient
Smaller / IUGR	Larger
Oligohydramnios	Polyhydramnios
Anemia	Hydrops / CHF

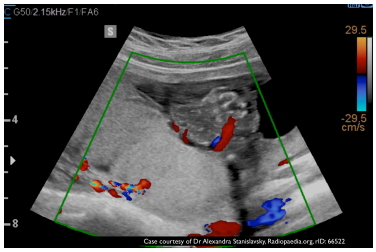
Stuck twin

Most severe type of TTTS. Oligohydramnios is so severe that the donor twin appears to be stuck to the side of the uterine wall

We can determine which is the donor either by the size difference or the sac difference. In this image we can see the fetus "stuck" close to the top of the screen since it has oligohydramnios. The amniotic membrane pulls it closer to the side of the wall. That would be the donor twin.



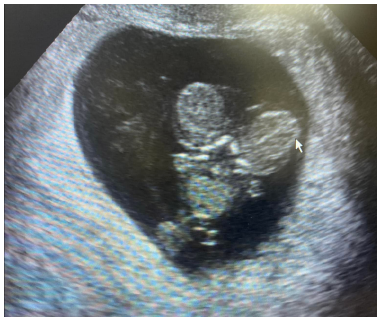




Acardiac twin

AKA TRAP sequence (twin reversed arterial perfusion). Abnormal anastomosis of placental vessels that support the growth of parasitic or acardiac twin.

One normal fetus and an abnormally developed fetus with no heart. The pump, or living, fetus maintains the growth of the parasitic twin. Pump twin has mortality rate of 50% secondary to polyhydramnios and prematurity. Acardiac twin demonstrates absent upper body, absent heart, an hydrops.



Conjoined twins

ONLY monochorionic/monoamniotic. Occurs when zygote splits >13 days. Most common: **thoracopagus (chest: most common)** and omphalopagus (abdomen). 40% chance stillborn.

Twin Demise

Dichorionic twins have greater chance of survival in case one twin demises, especially earlier on in first trimester

- Fetus papyraceus
  Fetal death in 1st trimester and is maintained, not reabsorbed. May eventually become vanishing
- Vanishing twin
  Death of a twin in the early 1st trimester and is reabsorbed = vanishing twin

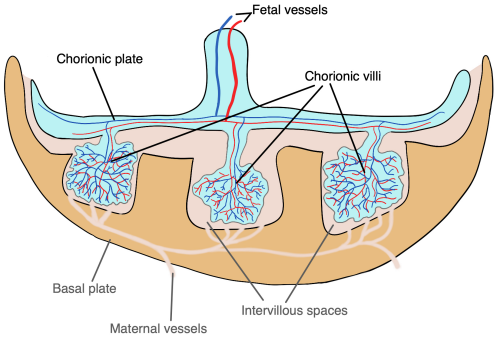
Monochorionic fetal demise of one will often lead to death of other.

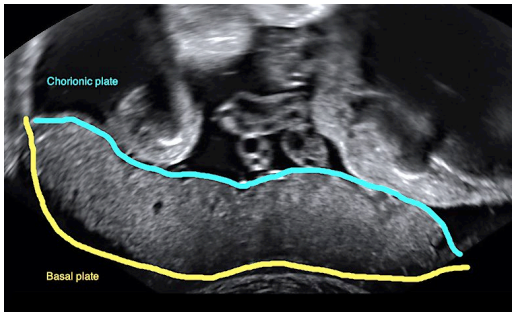
Fetal demise in the 2nd trimester of monochorionic gets can lead to *twin embolization syndrome.* Demised twin begins to breakdown and vascular products can travel through common vascular channels with shared placenta. Central nervous system and kidneys are usually affected.

Placenta

Normal 2-4cm thick. Major function as an excretory organ. Exchanges gases and waste products with nutrients and oxygen. Means of nutrition and respiration

- Maternal side: decidua basalis or basal plate. Maternal vessels enter intervillous spaces where the exchanges occur
- Fetal side: chorion frondosum or chorionic plate which contains extensions called chorionic villi. Functional unit of placenta : lobes of chorionic villi termed cotyledons

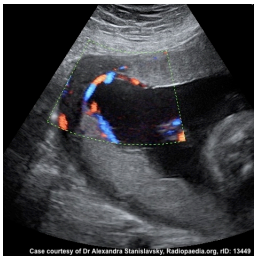




Placental Variants

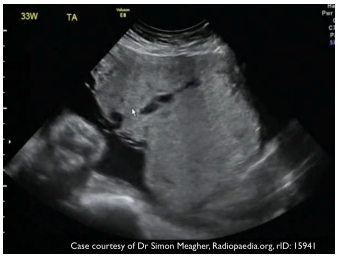
- Bilobed
  2 discs of equal size joined together by an isthmus of placental tissue (tissue connects the 2 lobes).

- Accessory lobe/ succenturiate lobe *
  Additional small lobe separate from main placental mass but connected by vascular connections. No placental tissue connection!



- Circumvallate
  Curled up placental contour appearing as a shelf. Curled edges, do not lay flat or smooth along wall. Inc risk of abnormal placental development and future abruption

- Venous lakes/maternal lakes/placental lakes/lacunae
  Pools of maternal venous blood. Sonolucent areas within placental mass. Will not fill in with color, but can be “swirling” in B-mode



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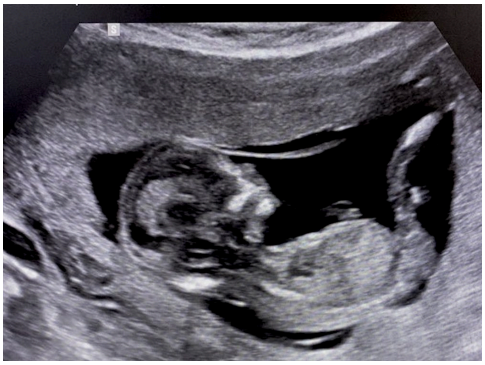
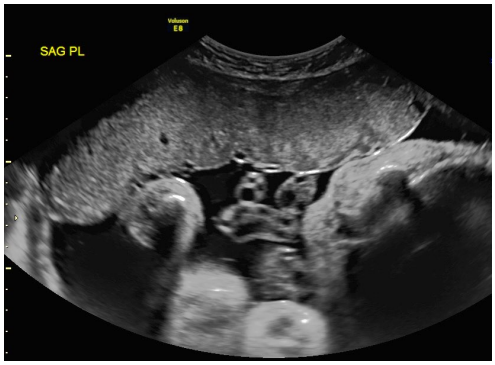
Placental Grading

Placenta should be age appropriate. Advanced maturation of the placenta can be an indication of maternal complications leading to placental insufficiency, and asymmetrical IUGR.

1st trimester Grade 0



2nd trimester Grade 1



Grade 0

1st tri to early 2nd

Homogenous, smooth echotexture. No indentations in chorionic plate. Smooth borders

*Grade 1 **normal for 2nd tri anatomy scan*

2nd tri to early 3rd

Subtle indentations in chorionic plate, small random hyperechoic foci

Grade 2

Late 3rd tri

Larger comma-like indentations alter chorionic plate, larger calcifications in basal plate

Grade 3

Post dates/advanced Complete indentations chorionic to basal plate. Irregular calcifications with shadowing. Related to drug abuse and preeclampsia. May cause IUGR if early gestation

Placental insufficiency: What's next?

- Asymmetrical IUGR: Poor placental health means the baby isn't getting enough oxygen and nutrients and growth will be affected. Brain will shunt more blood to itself
- Dopplers: The arteries feeding the placenta may have increased resistance patterns. Includes uterine and umbilical artery.

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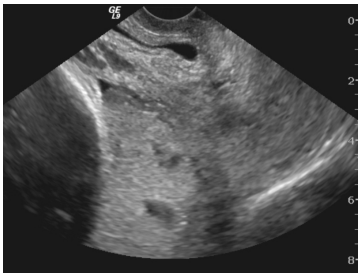
Placenta previa *

Previa = presenting/before. Placenta is implants within the LUS and covers/near to internal os. **Most likely cause of painless vaginal bleeding in 2nd/3rd trimester.** Increased risk with AMA, hx of C-section, multiparity. Can only be diagnosed 20 weeks onward due to possible placental migration.

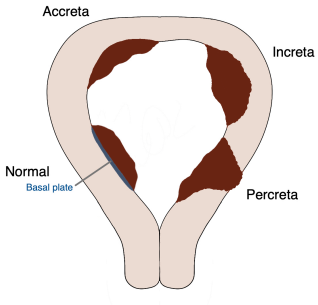


Important regarding technique: Overly distended bladder or LUS contraction may result in false positive. Best to scan with empty bladder or soft touch TV

- Complete: internal os is completely covered by placental tissue
- Marginal: edge of placenta touches internal os
- Low-lying: edge of placenta is within 2 cm of internal os

Accreta

General term for abnormal adherence of placenta to myometrium. Increased risk: Hx of multiple C-sections and/or uterine surgery. Scarring causes the disruption of the basal plate. Special concern for anterior LUS placental location with a history of multiple C-sections.



Sono: Loss of basal plate or myometrial/serosal layer, multiple placental lacunae, and increased peripheral vascularity

- Accreta: adhered to wall. Most common
- Increta: invades myometrium
- Percreta: penetrates through uterus and breach serosal layer

Abruption

Premature separation of placenta from uterine wall. High risk of fetal death. **CRITICAL FINDING**

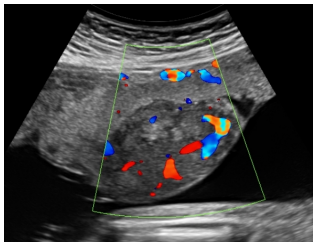
Risk factors: Hypertension, preeclampsia, drug/alcohol abuse, smoking, poor maternal health/nutrition

Clinical : Bleeding, pain, tenderness, trauma, decreased hematocrit

Sono: Hypo or anechoic region between placenta and uterine wall at level of basal plate

- Complete: most severe, entire retroplacental hematoma
- Partial: few centimeters of separation
- Marginal: placental edge, lifting the chorionic membrane from wall

Chorioangioma *



Most common placental tumor. May have elevated hCG. Most common location is adjacent to umbilical cord insertion at placenta. Vascular tumor

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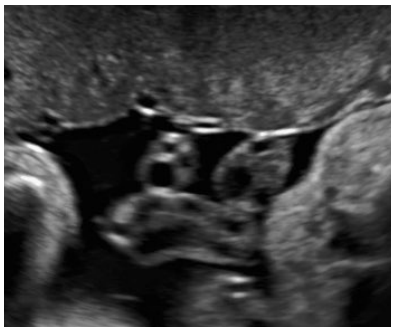
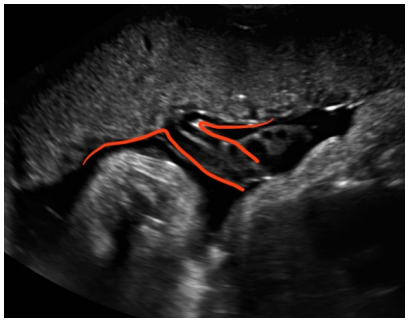


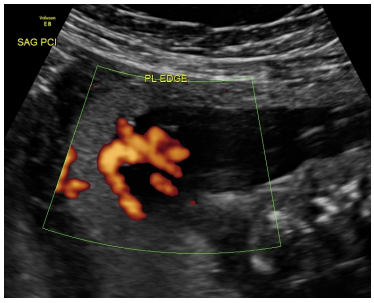
Umbilical Cord

Made up of 2 arteries and 1 vein surrounded by Wharton's jelly. Developed from yolk sac and vitelline duct. Vein carries oxygenated blood to fetus. Arteries carry deoxygenated back to placenta.
Best view : Axial view of 3VC in B-mode

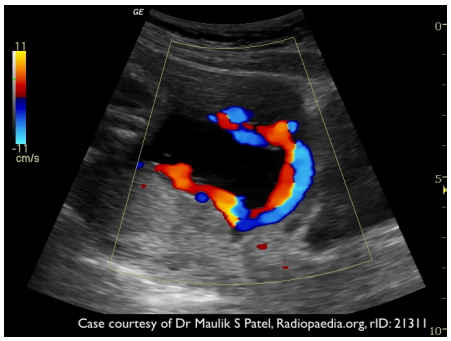


Normal placental cord insertion
Normally inserts in the central part of the placenta. Proper visualization and documentation of PCI is identifying the free floating cord attachment site into the placenta. T shaped appearance as outlined in image right

Abnormal PCI

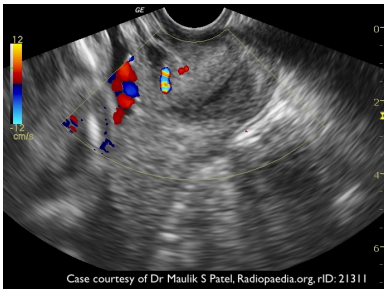


Marginal cord insertion *
AKA battledore placenta. Within 2 cm of edge of the placenta.



Velamentous/membraneous *
Insertion into the membranes beyond the placental edge and insert into side of uterine wall. Vessels must travel to insert into placenta. Vessels may wonder close to and cross internal os = vasa previa. Indication to perform TV to best visualize internal os

Vasa Previa *
Fetal vessels implanted across the internal os. Important for delivery management. May rupture as cervix dilates. Can lead to exsanguination of fetus



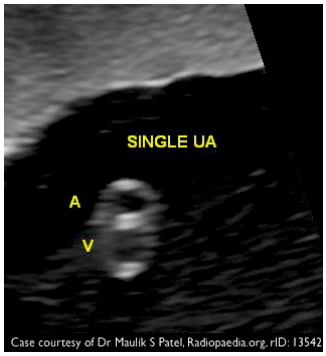
Cord cysts

- Allantoic cyst: cyst of cord adjacent to vessels. Usually near placenta. May be seen as free floating cord with "bubble" inside or adjacent to cord vessels
- Omphalomesenteric cyst: cyst of cord near fetal abdomen (seen at level of abdominal CI)

Hemangioma: most common tumor of cord. Solid, hyperechoic masses. Near placenta

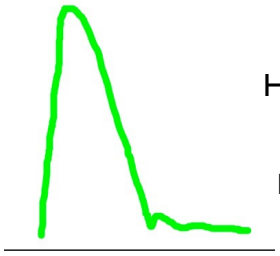
Single umbilical artery (2 vessel cord)

Most likely associated with congenital anomalies: cardiac, omphaloceles, syndromes, etc. Having only 1 umbilical artery does not threaten the wellbeing of the fetus in any way and generally would **not** be related to IUGR, fetal distress, hypoxia, or placental failure. Any association it does have is structural.



Obstetrical Dopplers

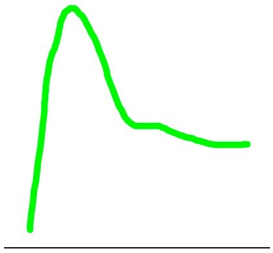
Resistance is determined by the demands of the organ. When an organ requires or wants more blood, the volume flow is increased by lowering the resistance. We can measure resistance by evaluating the change to the diastolic flow. Resistive index, pulsatility index, S/D ratio all measure resistance.



Higher Resistance

Less Diastole

Less volume flow



Lower Resistance

More Diastole

More volume flow

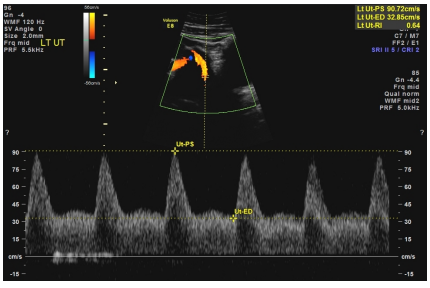
Uteroplacental

Uterine arteries

Supplies uterus and placenta. Gravid uterus now requires high volume flow >> low resistance.

When? Preeclampsia and any at risk for placental complications/ insufficiency. "Early indicator doppler"

Abnormal: Increased resistance/decreased EDV



Umbilical artery

Evaluates **placental resistance** and **fetal well-being**. Why? Placenta is the means of respiration and nutrition. But it is placenta determines the resistance of umbilical artery, since flow is towards the placenta. Increased placental resistance indicates placental insufficiency and as a consequence, fetal IUGR and hypoxia. Placenta is cause of abnormal resistance, but fetus will experience the results.

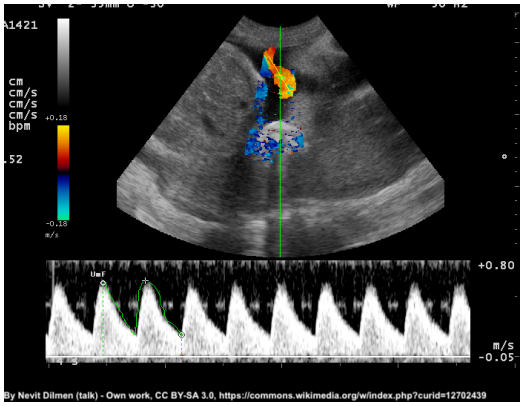
Only fetal cause of an irregular umbilical artery waveform would be arrhythmia (fetal HR reflected by pulsations of umbilical artery). If present, M-mode through an atrium and a ventricle

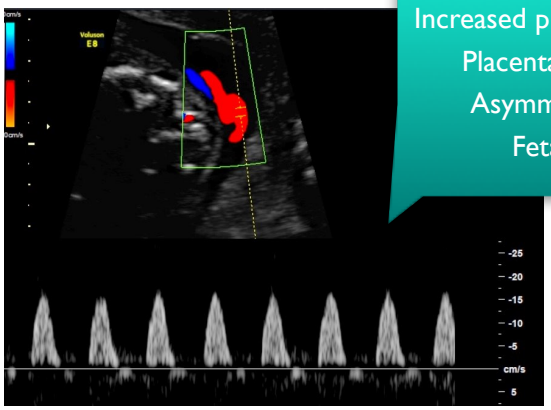
Normal

Low resistance, hi diastolic flow
S/D Ratio <3.0
Resistance decreases with gestational age

Abnormal

Increased resistance,
Decreased diastolic flow / diastolic flow reversal
S/D Ratio >3.0





Increased placental resistance
Placental insufficiency
Asymmetrical IUGR
Fetal hypoxia

Intrauterine Growth Restriction IUGR

EFW below 10th percentile. Biometry measures 2 weeks below expected gestational age. **AC used to evaluate for IUGR.**

Symmetric: entire fetus is evenly small. Usually starts earlier and related to fetal syndrome (ie-Triploidy)

Asymmetric: "brain sparing" head biometry may be WNL. Results in abnormal HC/AC ratio . Generally presents later (2nd trimester and on) and related to maternal complications, placental insufficiency and abnormal dopplers. Also important to then check MCA

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Fetal

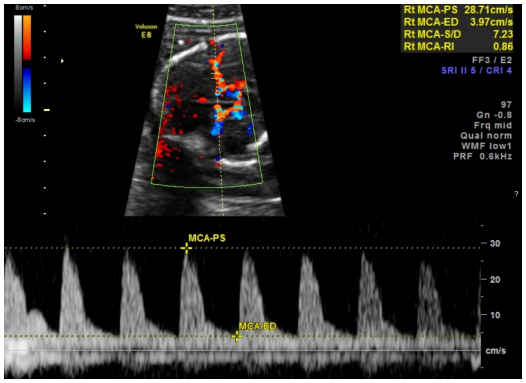
MCA (Middle Cerebral Artery)

Fetal hypoxia or anemia. Evaluate both resistance and velocity

- With asymmetrical IUGR, the brain is not receiving enough blood or oxygen, intracranial shunting takes place by lowering resistance. In other words, the fetus brain takes priority and shunts increased volume of blood to the head to "spare" the brain. Decreasing resistance means increased volume. Increasing supply to brain means the head is able to continue to grow resulting in the asymmetrical growth.
- Anemia reduces hemoglobin, making blood less viscous (thinner). Thinner blood flows faster resulting in increased PSV of the MCA

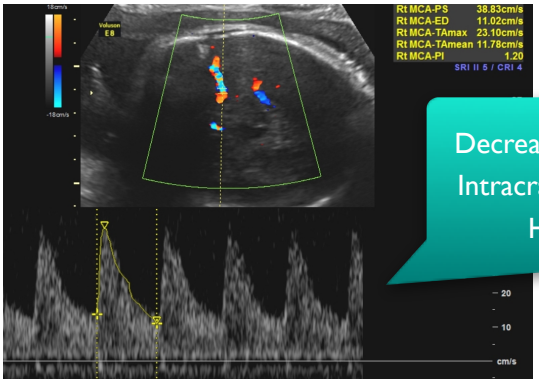
Normal

Higher resistance



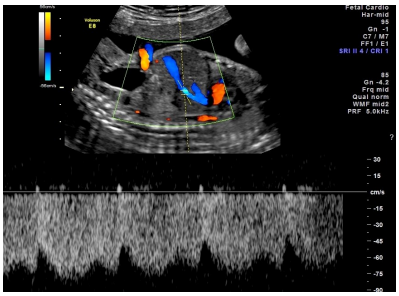
Abnormal

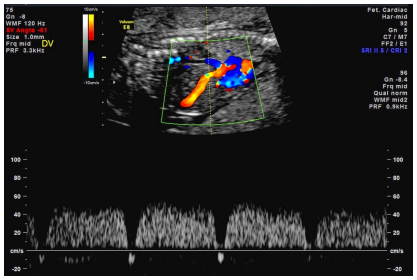
Lower resistance



Umbilical vein / Ductus Venosus

Placenta to fetus. Umbilical vein should be steady, minimally phasic with constant flow towards the fetus. Ductus venosus takes on a more pulsatile waveform since its closer to the heart and waveform reflect atrial contractions. Flow should always stay forward and constant.





Abnormal flow patterns (a-wave flow reversal) in DV indicate increased fetal resistance and carries very poor prognosis

Causes: Only fetal problems such as CHF, hydrops, pulmonary hypoplasia and others that increase fetal resistance.

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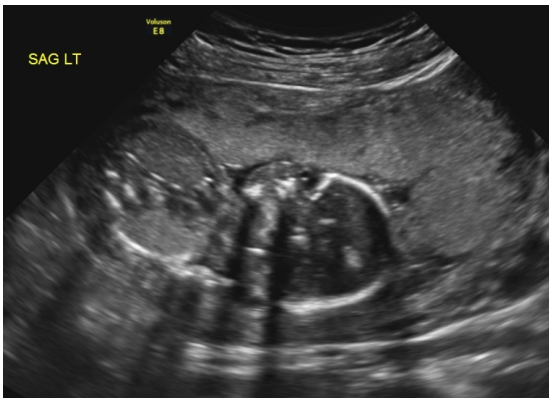
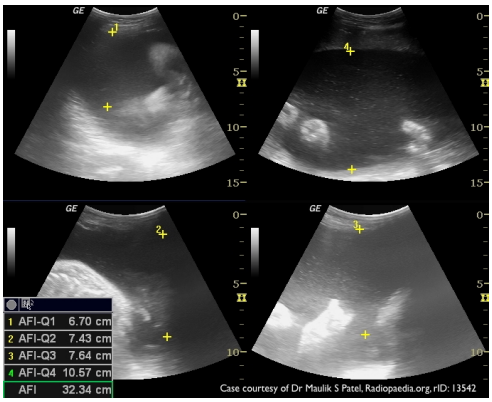
Fetal Environment

Amniotic Fluid

- Amniotic Fluid Index AFI: Dividing the uterus in 4 quadrants and measuring the largest pocket of fluid in each quadrant (excluding fetal parts and cord) and add together. Normal range: 5-24cm. Progressively increases until 28 weeks, then slowly decreases
- Deepest Vertical Pocket DVP: Maximum vertical pocket excluding fetal parts and cord





Oligohydramnios	Polyhydramnios
AFI <5cm / DVP <2cm	AFI >24cm / DVP >8cm
Demise **Renal** **IUGR** **PROM** **Post-dates**	**GI atresia** **Skeletal** **Open defects** **Hydrops** **Cystic hygroma**

Biophysical profile

Total possible score 8/8

- Fetal breathing- at least 30 sec (2)
- Gross fetal movements- 3 gross movements (2)
- Fetal tone- 1 limb or hand flexion/extension (2)
- Amniotic fluid- 1 pocket measuring 2 cm x 1 cm (2)

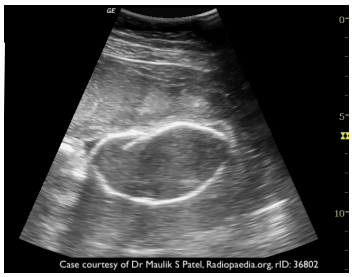
** 2 categories are for fetal movement. NO movement = loss of points from both sections

Lung Maturity

In 2nd to 3rd trimester, **Most accurate measure** is performed by amniocentesis to measure the Lecithin/Sphingomyelin ratio AKA L/S ratio

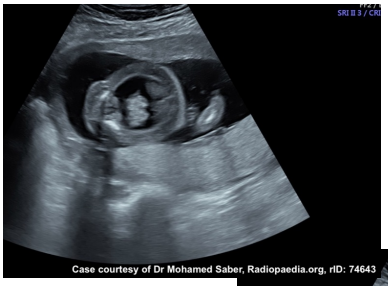
Sonographic evidence of lung maturity: lung increasingly echogenic compared to liver in 3rd trimester

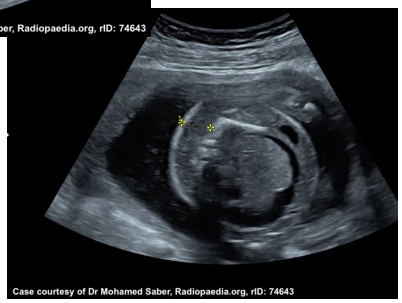
Features of 2nd/3rd trimester fetal demise



- *Spaulding sign** (image right): overlapping skull bones/ collapsing brain
- Roberts sign: Air trapped in abdomen and lungs and increased echogenicity
- Exaggerated curvature of spine

Hydrops





Fluid found in <u>2 fetal cavities</u>.

- Pericardial effusions: around heart
- Pleural effusions: around lungs
- Ascites: abdominal cavity
- Subcutaneous edema/anasarca: edema of the skin (thickness >5mm)

Associated with polyhydramnios and thickened placenta or placentomegaly (>4cm)

Immune hydrops

Only one reason: Rh incompatibility/ Rh isoimmunization

When mother is Rh-negative and 1st pregnancy's fetus is Rh-positive. Antibodies are created during 1st pregnancy. The next pregnancy with Rh-positive fetus, mother's antibodies attacks the fetus. It is as if she is immune to her own baby. Antibodies destroy the fetus' red blood cells leading to fetal anemia and hydrops, this is termed **erythroblastosis fetalis**

Prevention: RhoGAM (Rh immune globulin) at 28 weeks.

Non-immune hydrops

Caused by any other reason for hydrops usually fetal conditions such as cardiovascular, GU, placental abnormalities, fetal syndromes, CCAM, cystic hygroma, fetal neoplasms, chromosomal abnormalities. Pericardial effusions are usually earliest finding

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Maternal Complications

TORCH

Toxoplasmosis, Other infections, Rubella, Cytomegalovirus, Herpes simplex

Group of infections that cross the placenta causing damage to fetus. Sono: calcifications

Thyroid dysfunction (Graves disease / Hashimoto's thyroiditis)

Check for goiter (enlarged thyroid) on fetus

Supine Hypovolemic Syndrome

IVC syndrome. When gravid uterus compresses IVC lowering blood pressure and causing patient to feel dizzy, sweaty, and nauseous.

Turn patient on left side

Preeclampsia and Eclampsia

- Preeclampsia (toxemia) is the presence of pregnancy-induced hypertension and proteinuria. Mother experiences edema in hands, feet, and legs.
- Eclampsia is long-standing, uncontrolled preeclampsia causing headaches and seizures.

Increased risk: Hx of preeclampsia, diabetes, GTD.

May result in placental problems and therefore, IUGR, oligohydramnios, and placental abruption.

*** Always perform dopplers > Uterine arteries and Umbilical artery

HELLP

Hemolysis, Elevated Liver enzymes and Low Platelets

Life-threatening complication requiring delivery. To check maternal liver for hemorrhage/hematomas

Maternal diabetes

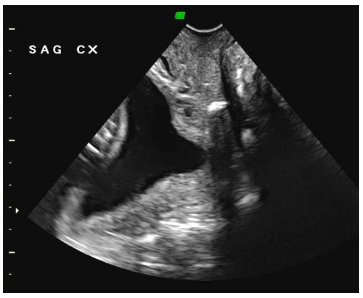
Pregestational diabetes mellitus Type 1 or Type 2 : higher risk of maternal complications of miscarriage and toxemia. Also increased risk of congenital anomalies: heart, skeletal, syndromes, etc

Gestational diabetes is the most common type of diabetes with pregnancy and resolves after birth. Major risk is macrosomia. Also associated with placentomegaly, polyhydramnios. NO fetal defects associated with gestational diabetes

- "Large for Dates" or macrosomia
  EFW greater than 90th percentile. Macrosomia - neonate weighs >4500g

Incompetent cervix

Painless premature dilation of cervix.



Normal at least 3 cm in length (Measurement is taken from internal os to external os). If incompetent, measure closed portion only. The shorter the cervical length, the more likely preterm delivery. The open cervix may funneled or demonstrate bulging membranes

Important regarding technique: an overly distended bladder or contraction can cause a false-negative. If not well vis TA or patient has history, TV is indicated

Treatment: MacDonald and Shirokdar cerclage

Pre-term Labor

Onset of labor before 37 weeks.

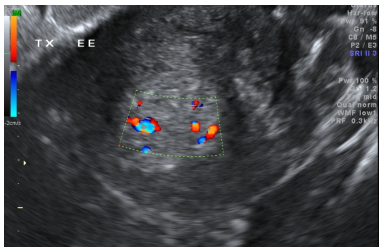
PROM

Premature rupture of membranes prior to the onset of labor. Oligohydramnios

Post Partum

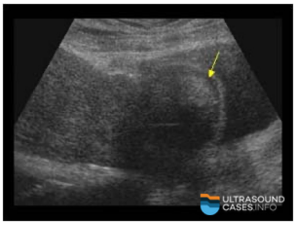
Normal post partum uterus returns to non-gravid size 6-8 weeks after delivery.

- Retained products of conception



Part of placenta may be left behind. C/o post partum vaginal bleeding. Seen as echogenic intracavitary mass within the endometrium or thickened irregular endometrium.

- Bladder flap hematoma



Results from C-section. Complex mass adjacent to scar between LUS and posterior bladder wall (ant CDS)

- Infection/abscess

Fever, increased WBC's/leukocytosis, tenderness. Associated with poor nutrition and hygiene, anemia, C-section, prolonged labor. No specific sonographic findings if no abscess. Abscess: complex fluid collection

Obstetrics/Gynecology Registry Review
Study Guide

Physics Review

- Image optimization based on patient, study type, and findings
- ALARA principle
- Doppler principles

Image Optimization

B-mode scanning technique

- 90 degrees AKA perpendicular imaging angle is best for smooth interfaces such as visualizing a interventricular septum or amniocentesis needle

Transducer frequency choice

- Study type and focus based on indications
- Patient body habitus
    - More superficial the imaging = choose the higher within the frequency range
    - More penetration is needed = lower the frequency

Frequency and Transducer

Transabdominal = Low frequency curved 2-6 MHz

Transvaginal = High frequency 6-8 MHz

ALARA

As Low As Reasonably Achievable

- Reducing acoustic exposure by limiting use of controls that increase output and considering overall scan time
- Monitor output display indices (max thermal index TI 0.7 and mechanical index MI)

Examples of ALARA Guidelines

- Using M-mode for heart rate especially in 1st trimester
- If unable obtain M-mode, cardiac activity should be captured with cine loop
- Exam time should not be extended for non-medical reasons
- Doppler should only be used when clinically indicated and necessary

Obstetrics/Gynecology Registry Review
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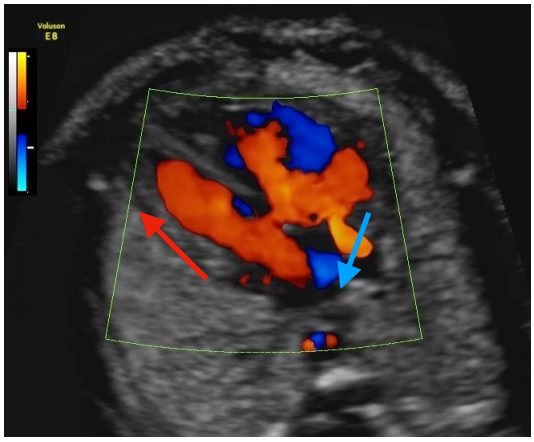
Doppler principles

- Color: Important for determining presence and direction of flow

Determining flow direction

First, look at the scale. The color on top is positive and bottom is negative. Red is positive in this image so it is towards the probe or top of the screen.



In this image taken at the 4CH view, the red areas are flowing towards the probe, meaning into the ventricles. This indicates diastole.

The blue flow is going away from the probe which means blood goes from right atrium to left atrium through the foramen ovale. There's also a VSD as flow moves across the septum into the right ventricle.

How to correctly use Color Doppler

The size of the box should just cover area of interest.

Adjust the scale to fit the type of flow you are evaluating.

Adjust color gain so color fills in vessel but does not 'bleed' out of the vessel/chamber walls.

- PW doppler: Important for quantifying flow velocity and identifying normal perfusion

  Examples: Resistance index, pulsatility index, and S/D ratio important for evaluating normal umbilical artery, uterine artery, and MCA flow patterns

- Power Doppler: Use power doppler when only are interested in presence of flow.
  - Benefits: very sensitive to slow flow
  - Limitation: no direction information

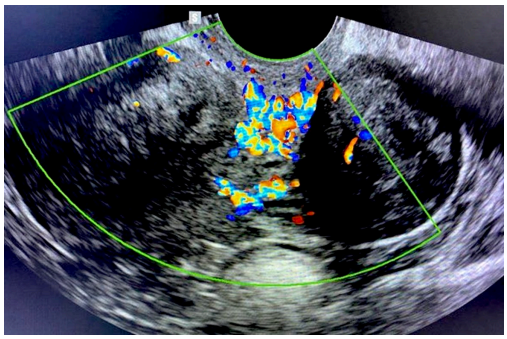
Doppler Optimization

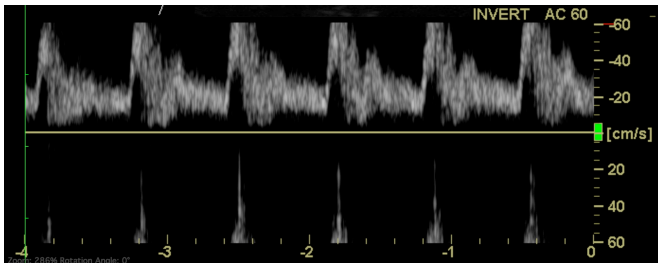
Velocity Scale aka PRF

Needs to match the type of flow you are evaluating

- Decrease the scale = for slower flow or when not sensitive enough
- Increase the scale = when it's aliasing

  Aliasing in color = mosaic pattern. In PW, always must be corrected since the peak cannot be measured accurately





Wall filters and *High Pass filters*

Filters LOW FREQUENCY/HIGH AMPLITUDE.

- Decrease WF = when not sensitive enough

Gain

Fine tuning only! First adjust scale and wall filter appropriately.

- Increase gain to enhance the strength of the doppler signal
- Decrease if bleeding out of vessel

High velocity flow	Low velocity flow
High scale	Low scale / Low wall filter
Uterine artery **Umbilical artery** **Aorta/MPA/MCA**	**Internal parenchymal flow** **R/O ovarian torsion** **Eval for polyp/endo vascularity**

Trisomy Matching "Memory" Game

Cut out along the dotted lines, mix up and spread all tabs facedown. Turn one defect over at a time and place in one of the 3 designated trisomy piles based on which it is associated with

Absent nasal bone	Thickened nuchal fold
Macroglossia	Echogenic intracardiac focus
AV canal	Duodenal atresia
Sandal gap foot	Choroid plexus cysts
Micrognathia	Esophageal atresia
Strawberry skull	Rockerbottom foot
Clinodactyly	Holoprosencephaly
Proboscises	Hypotelorism
Cyclopia	Median cleft lip
Clubfoot	Omphalocele
Polydactyly	Omphalocele