FOGSI FOCUS

ULTRASOUND IN OBSTETRICS & GYNAECOLOGY

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Presidents' Message

Dear FOGSIANS,

It is a great pleasure to present to you FOGSI-FOCUS on Ultrasonography in Obstetrics & Gynaecology. USG is an integral part of Ob. Gyn. Practice. USG for Ob. Gyn. Specialist is like a sthethoscope to a physician. I take this opportunity to salute Late Dr. M.Y. Raval, who as founder chairman of FOGSI Imaging Science Committee put in Herculian efforts to spread the knowledge of USG related Obstetric & Gynaecology amongst fellow FOGSIANS. Dr. Pratap Kumar, myself, Dr. P.K. Shah & Dr. Mandakini Megh as chairpersons of FOGSI Imaging Science Committee, continued to disseminate the knowledge of USG in our field.

This technology should not be misused by any member of FOGSI. FOGSI condemns any method of sex determination that can result into female foeticide. Let us all use this knowledge for betterment of health of all the women of our country. I congratulate all the directors of centres recognized by FOGSI for imparting training in Ob. & Gyn. Ultrasound. I request all the FOGSI members doing USG themselves to follow PCPNDT Act strictly and help our nation bring back sex ratio to normal.

I congratulate Dr. P.K. Shah, Dr. Mandakini Megh & Dr. Nandita Palshetkar for making it possible to bring out FOGSI – FOCUS on Ultrasound. All the contributors have done great job in making this Focus very informative.

EDUCATE, PREVENT & ERADICATE, FOGSIANS ONLY YOU CAN MAKE A DIFFERENCE.

Dr. Narendra Malhotra
President ,**FOGSI**

FOGSI – FOCUS ON ULTRASOUND IN OBSTETRICS & GYNAECOLOGY

Editors : Dr. P. K. Shah,Dr. Mandakini Megh,Dr. Nandita Palshetkar

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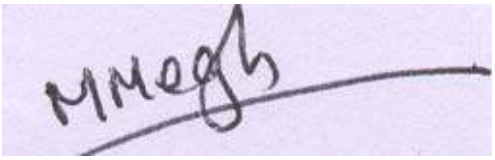
From Editor's Desk



We can not think of Ob. & Gyn. practice now a days without ultrasonography. USG has revolutionized our practice. Technological advances have made 3D & 4D USG available to us. An honest effort has been made to provide precise information on some of the practical aspects of USG in this issue of FOGSI FOCUS. We acknowledge the efforts put in by all the authors. We have made this issue available for all on FOGSI's website, www.fogsi.org.

We thank FOGSI President, Dr. Narendra Malhotra for help, support & constant encouragement in making it possible to publish the FOGSI Focus. We sincerely hope that this focus will help all FOGSI members in improving their knowledge on the subject.

"Beti Bachao, Beti Padhao".


Dr. P.K. Shah Dr. Mandakini Megh Dr. Nandita Palshetkar

TRANSVAGINAL SONOGRAPHY IN INFERTILITY

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The application of transvaginal ultrasound in the evaluation and assessment of the infertile couple is expanding each day. The transvaginal ultrasound picture depicts accurately the pelvic anatomy of the scanned area safely, quickly and reproducibly.

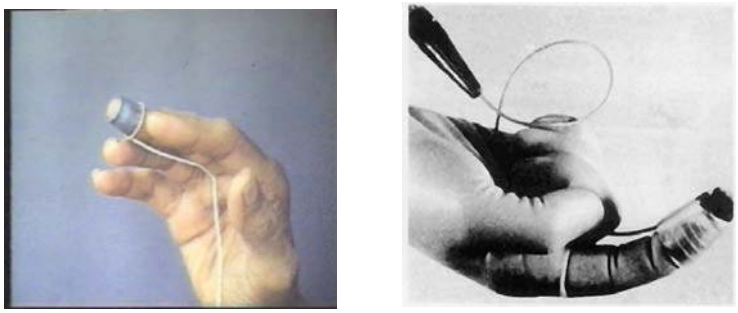
(Fig. 1 : USG Machine & Probes)

The quality of depiction of the pelvic anatomy is dependent on the ultrasound equipment being used and the experience and proficiency of the person performing the scan.

It should be mandatory for the person performing the scan to know about the female endocrinology and be well versed with the causes and management of infertility, specially with the ovulation induction protocols.

Till date there are no known adverse biological effects of Transvaginal ultrasound on the patient, on the oocytes or on the ultrasound operator [1] (A.I.U.M.)

Transvaginal ultrasound today is the modality of choice in evaluating male and female infertility as a first step investigation and should be used by the clinician in the consulting chamber along with pelvic examination. It is like marrying palpation with imaging.

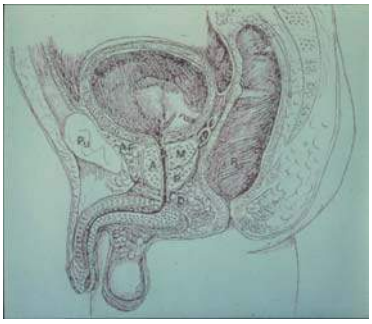


(Fig. 2 : Finger Tip Probe)

Ultrasound Assessment of the Male Partner

Male factor infertility today comprises of almost 40% of the causes in an infertile couple. The modern life style and additives in food have become a major environmental cause of oligo astheno spermia. The function of male genital system encompases the central nervous system (hypothalamus and pitutary), the adrenal glands, the testes, the

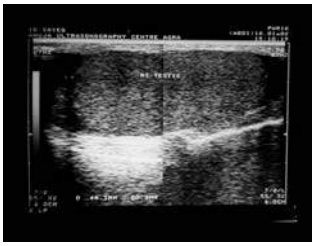
epididymis, the seminal vesicles and the prostate gland. Any malfunctions of any of these may affect the male reproductive capacity.



(Fig. 3 : Male Reproductive System)

In suspected male factor infertility, ultrasound imaging of the ejaculatory system and of the testis is necessary to rule out structural anomalies. Scrotal and transrectal-ultrasound (TRUS) are used in evaluation of the reproductive tract disorders. Color flow imaging is used for assessment of varicocele. 3-D is used for testicular volume and seminal vesicle & prostate evaluation. Computed tomography and endorectal Magnetic Resonance Imaging can also be used.

Scrotal sonography is performed with patient in supine position using a 7.5 – 10 MHz linear probe. This can evaluate the testis for size, shape, hydrocele, benign tumours, atrophy, malignancy, orchitis, torsion, haemorrhage, focal lesions etc. Ultrasound imaging is very sensitive in testicular evaluation. [2][3][4][5]



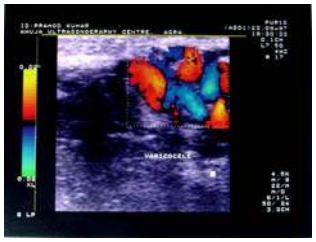


Fig : Testis Fig Varicocle

Transrectal Ultrasonography (TRUS) is an excellent approach for visualising the seminal vesicles, prostate and ejaculatory ducts. With TRUS we can assess obstructions, absence or hypoplasia of seminal vesicle and ejaculatory ducts. TRUS is an excellent screening test for ejaculatory duct pathologies and is indicated in all men with severe oligospermia and a low volume ejaculate. [5]

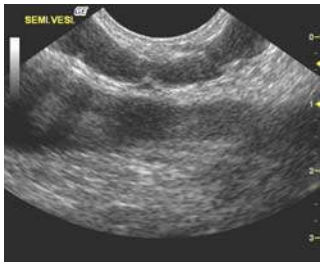


Fig. 4 : TRUS Seminal Vesicle Bow Tie picture

Assessment of the Female Reproductive Tract

The female is responsible for 40% of causes of infertility and contributes in another 20% of mixed causes in the couple. Out of this the ovulatory dysfunction (30%) and tubal factor infertility (25%) are major factors[6]. The female reproductive tract is usually evaluated by the busy gynaecologist by a per speculum and a per vaginum examination and then an HSG is usually ordered and then a transvaginal scan is ordered and later followed with other tests. These usually take time and frustrate the patient and the doctor.

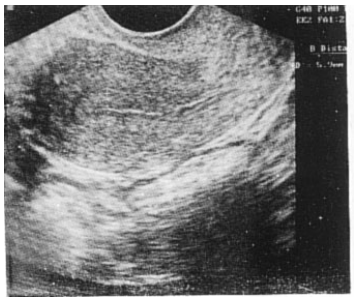
We advocate a Transvaginal scan as the very first visit of the couple by the infertility specialist himself/herself just after a p/s exam, this will enable the clinician to come to a diagnosis on the very first visit regarding problems in vagina, cervix, uterus, endometrium, endometrial cavity, tubes, adnexa, ovaries and general pelvis as a whole. Such an examination helps to decide the further treatment line and actively manage infertility by a single day evaluation test and active management protocol (Rajan, Malhotra) (2000)[7][8].

Vaginal and Cervical Factor Infertility Evaluation :

Vagina and the cervix is the first obstacle that the spermatozoa have to negotiate on their way to reach the oocyte. Vaginal septae, stenosis, vaginismus and coital difficulties are best assessed by a per speculum examination, however TVS helps to locate vaginal cysts and vaginal infiltrations.

Cervix is composed of cervical glands which secrete mucus in response to estrogen stimulation and this secretion assists in passage of sperms. About 5-10% of causes of infertility are due to cervical factors, which may be anatomical or functional abnormalities.

Transvaginal ultrasound can very accurately assess both anatomical and functional problems of the cervix.



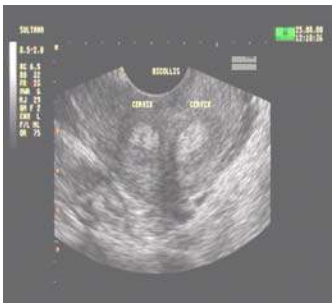
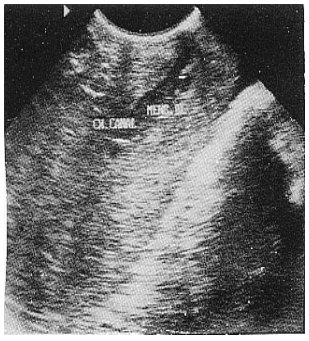


Fig. 5 : Normal Cervix



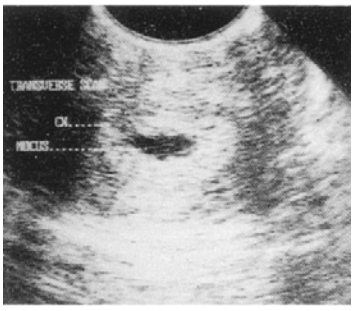
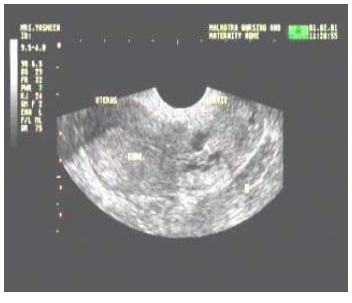


Fig. 6 : Cervical Mucus



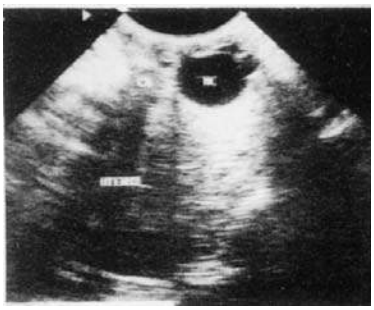


Fig. 7 : Nabothion Cysts

Assessment of Cervicitis, nabothian cysts at internal os, poor cervical mucus, cervical agenesis and cervical stenosis is possible and should be done. Cervical conisation and cervical infections should always be kept in mind and assessed for clinically before a TVS scan.

Uterine Factor Assessment and Evaluation :

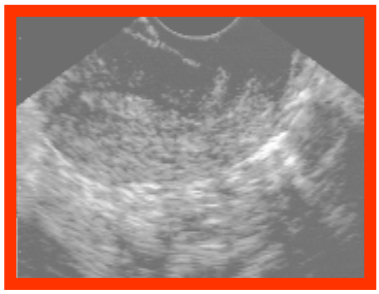
Uterus is the place for embryo implantation and pregnancy continuation. The normal adult uterus is a muscular organ 6-10 cm in length and 3-5 cm in width and has a unique capacity to grow and expand to hold a full term fetus during pregnancy.

Problems of uterus may lie in the musculature (Fibroids, Adenomyosis etc.), in the uterine cavity (congenital uterine malformations, adhesions, uterine cavity polyps, etc.) or problems in the endometrial lining (i.e. inappropriate endometrial growth and secretary transformations in response to progesterone from corpus Luteum).

TVS can accurately assess the uterine factors and with addition of fluid (saline) by sonohysterography the cavity can be accurately studied; with addition of color flow imaging, color doppler studies of uterine artery; power angio the spiral artery and endometrial vascularisation can be evaluated to score the uterus for favourability of implantation (uterine scoring system for Reproduction)(Applebaum, Dalal, Malhotra)

The Uterus

Transvaginal ultrasound examination of the body of uterus is done to observe detailed view of the myometrium and any anomalies, Leiomyomas are one of the most common benign neoplasms in women and have been reported to occur in up to 40 percent of women over the age of 35. A leiomyoma may be suggested by generalized enlargement of the uterus, irregularities in the surface contour, distortion of the endometrial echo, or as areas of hyper or hypoechogenicity compared with the surrounding normal myometrium. Since leiomyomas are composed of smooth muscle cells with acoustic characteristics similar to the surrounding normal uterine tissue they may not be imaged as a separate entity. Uterine leiomyomas do not have a true capsule and there may not be an acoustic interface and therefore no echo resulting from a structural boundary. A submucosal myoma within the uterine cavity may be imaged as an area of increased echogenicity and may be mistaken initially for blood, mucus or a polyp in the uterine cavity. (Fig. Normal Uterus)



(Fig. 8 : Normal Uterus)

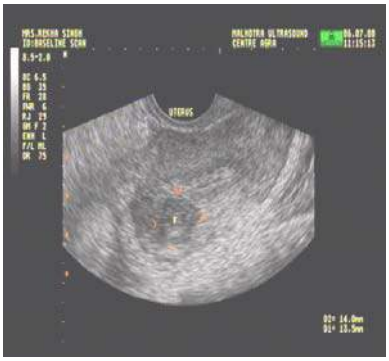
Endometriosis

Endometriosis is a condition where there is ectopic menstruating endometrium, leading to adhesions and/or cyst called as the chocolate cyst. Minor degrees of endometriosis cannot be diagnosed by sonography. The endometrioma cyst wall is generally shaggy and irregular sometimes with septations. These cysts are

homogenous with a low level echo patterns with good through transmission. They have fine stippling pattern filling the whole of the cyst.

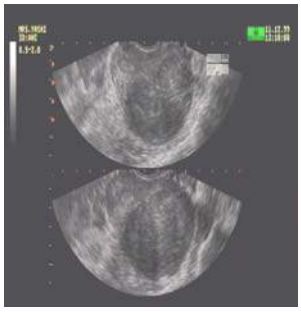
Fibroids

Fibroids can cause infertility by blocking the cervical canal or by blocking the fallopian tubes mechanically. Fibroids pressing over the endometrial cavity can diminishes the available endometrium for implantation and also interferes with the transport of sperms & oocytes. Intramural myomas also increase the uterine irritability and are implicated in causing implantation failures or early pregnancy losses. (Fig. 9 : Fibroid Uterus)



(Fig. 9 : Fibroid Uterus)

Transvaginal ultrasound is the most useful tool for screening for fibroids. The uterus is enlarged with contour deformity (Fig. 10 : Fibroid causing contour problem) and focal masses with different echogenecities (Hypoechoic usually, hyperechoic when calcified and may be isoechoic also).

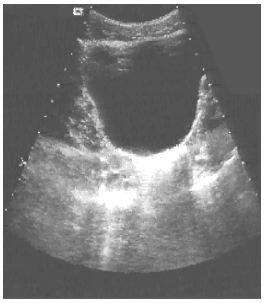


(Fig. 10 : Fibroid Causing Contour Problem)

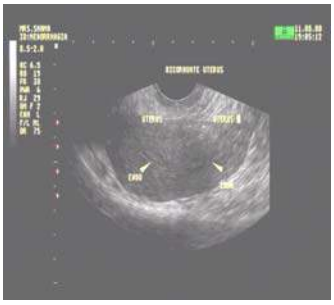
Congenital Anomalies

Congenital anomalies of the uterus occur in about 0.1-0.4% of general population of women and are due to the embryological problems in Mullerian system. Congenital anomalies of the uterus cause infertility and also is a significant cause of recurrent pregnancy loss[9][10]. About 80% of women with congenitally abnormal uterus may have no problems in concieving but anomalies are responsible for almost 20% of recurrent

pregnancy loss and hence should be carefully looked for and treated whenever encountered during infertility evaluation. (Fig. 11 : Bicornuate Uterus) (TAS & TVS)

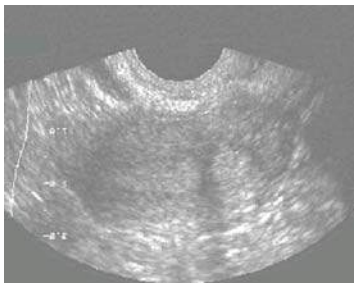


(Fig. 11 : TAS)



(Fig. 11 : TVS)

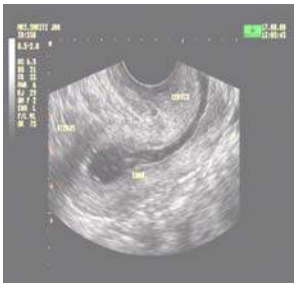
Uterine congenital anomalies can be diagnosed by HSG, TVS, contrast sonohysterography, hysteroscopy and laparoscopy and by a MRI 3D HSG and TVS without saline contrast are the commonest methods. The anomalies which can be diagnosed are bicornuate uterus, unicornuate uterus, intrauterine septa (complete, incomplete or arcuate)[11]. (Fig. 12 : Septate Uterus)

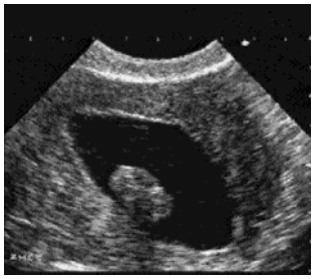


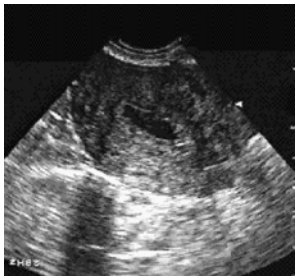
(Fig. 12 : Septate Uterus)

Sonohysterography

Instillation of sterile saline into the uterine cavity under ultrasound guidance (TVS) will let us study the uterine cavity without any radiation exposure and without exposure to contrast media (Fig. 13 : Saline Contrast Sonography) The saline distended cavity is anechoic surrounded by symmetric endometrial lining. Sonohysterography will enable the diagnosis of Asherman's Syndrome or intrauterine adhesions,polyps, submucous fibroids and uterine septa. (Fig. 14 : Polyp Fibroid) (Fig. 15 : Asherman Syndrome)[12]. HYCOSY or contrast hysterosalipingo sonography involves the use of a sonography contrast media (Echovist) (Fig. 16 a & b Echovist). In the future for the better understanding,this may replace the more invasive HSG as a first time investigation of the infertile female.

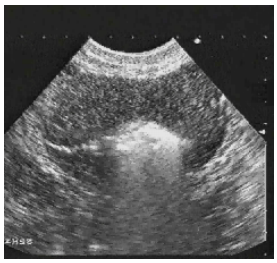






(Fig. 13 : Saline Contrast Sonography)(Fig. 14 : Polyp Fibroid)(Fig. 15 : Asherman Syndrome)

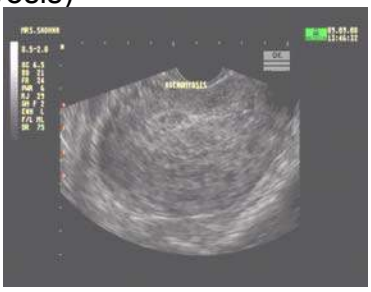
(Fig. 16 a : Echovist)



(Fig. 16 b : Echovist)

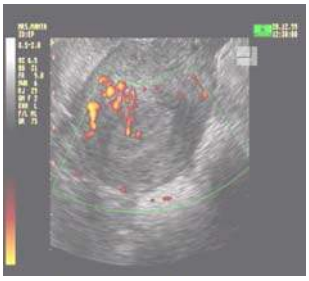
Endometriosis of the Uterus (Adenomyosis)

Endometriosis is a disease in which typically the endometriotic implants are scattered in various extra uterine locations. However sometimes the ectopic endometrium goes into the myometrium and causes adenomyosis. These endometrial tissue start to proliferate inside the myometrium and tends to bleed on progesterone withdrawal during the menstrual cycle thus giving the uterus a typically speckled appearance, resembling 'Salt' & 'Pepper' (Hyperecohic areas and hypoechoic areas). Depending on the extent of lesion and the severity of disease the uterus will appear enlarged and sometimes all of the adenomyosis areas may together look like a fibroid (Adenomyoma). (Fig. 17 : Adenomyosis)



(Fig. 17 : Adenomyosis)

Color Doppler imaging (Fig. 18 : Color in Adenomyosis) helps as the blood flow in these lesions resemble spiral arterial endometrial blood flow pattern while that of the fibroid is single vessel on the periphery. Also the identification of capsule around the mass is seen in leiomyomas while Adenomyomas have no capsules.



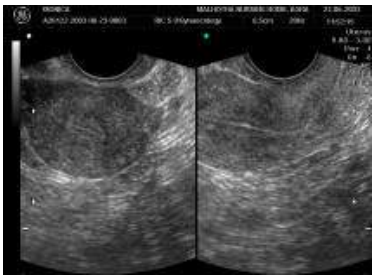
(Fig. 18 : Color in Adenomyosis)

Evaluation of Endometrial Growth

Endometrium is the inner lining of the uterus and has receptors for ovarian hormones and in response to the estradiol from the ovaries (or exogenous), the endometrial lining grows in a typical pattern which is recognisable by TVS. After ovulation (36-48 hr. later) the corpus luteum starts producing progesterone and the endometrial cells and endometrium will now start exhibiting secretary changes which is also well identified by TVS and color Doppler. Endometrial growth correlates well with

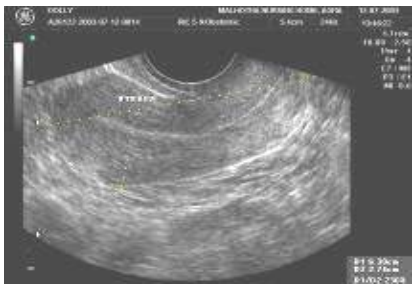
ovarian hormone levels. For ultrasound evaluation of the endometrium, we need to look at endometrial thickness, endometrial pattern and color flow in spiral arteries and endometrial receptivity scoring.

Endometrial thickness is the maximum distance between the echogenic interfaces of the myometrium and the endometrium, measured in a plane through the central longitudinal axis of the uterus. Very easy to obtain the plane and very easy and reproducible to measure (Fig. 19 : Endometrial Measure).



(Fig. 19 : Endometrial Measure)

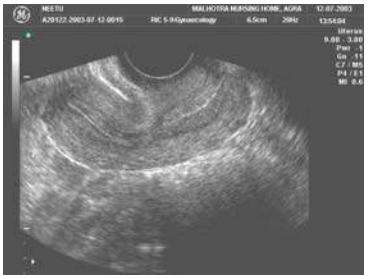
The basal study of endometrial thickness should be started from day 2 or day 3 of Menstrual cycle to look for proper shedding. The endometrium on this day should appear as a thin bright echogenic line or the cavity shows some blood with debris. A thick endometrium on basal scan (Day 2) indicates improper endometrium shedding. (Fig. 20 : Day 2/3 endometrium)



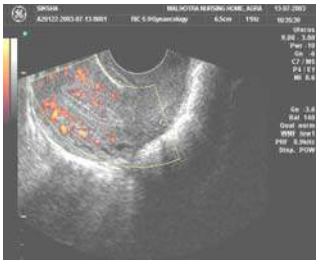
(Fig. 20 : Day 2/3 Endometrium)

The endometrium grows at the rate of 0.5 mm/day in the proliferative phase and 0.1 mm/day in the Luteal phase. A thickness of more than 7mm in the Preovulatory period is associated with higher pregnancy rates [13].

Endometrial Pattern is the relative echogenecity of the endometrium and the myometrium as seen on a longitudinal TV scan. In a typical 3 layer pattern of proliferative phase, (Fig. 21 : Day 9 endometrium) the central line represents the uterine cavity and the outer lines represent the basal layer. Outside this is a hypoechoic interface in between endometrium and myometrium (sometimes described as 5 line endometrium). (Fig. 22 : Periovulatory endo) The hypoechoic area in between the two bright lines represents functional layer of the endometrium [14]. Endometrium growth and pattern can be graded and classified.



(Fig. 21 : Day 9 endometrium)



(Fig. 22 : Periovulatory endo)

1. Four patterns have been described from a fully echogenic endometrium (Grade A) to a district black region surrounding the midline[15].

SMITH'S GRADING

Grade A : Bright Endometrium represents post ovulation or the luteal phase

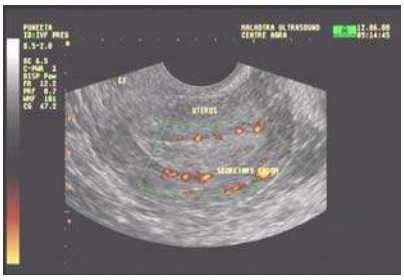
Grade B : Endometrial reflectivity is similar to the myometrium. This characterizes late follicular phase.

Grade C : A solid area of reduced reflectivity appears as a darker area next to the lighter myometrium. This is pattern of mid follicular phase.

Grade D : Echoes are absent in the endometrium, but a bright central echo is seen, described as the triple line.

Late follicular phase endometrial pattern Grade B on the day of hCG is associated with increased pregnancy rates (Smith et al)[15].

Post ovulatory the endometrial echogenecity changes with loss of layers but spiral artery flow increases. (Fig. 25)



(Fig. 25)

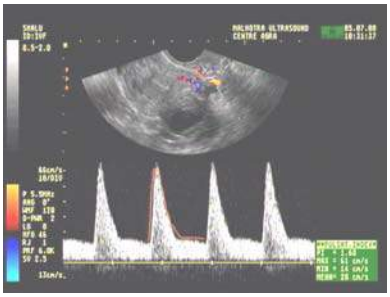
Endometrial Thickness and Menstrual Cycle

Phase	**Appearance**
Menses (day 1-5)	Hypoechoic area is blood. Myometrial contractions are frequent. Thickness < 4 mm.
Early Follicular phase (day 6-10)	Distinct 'triple-line' pattern. Hypoechogenic endometrium. Thickness 7-9 mm.
Late follicular phase (day11 ovulation)	Endometrial appearance similar to myometrium. Thickness 9-12 mm at ovulation
Luteal phase	Bright, fluffy appearance. Absence of triple line. Thickness 10-14 mm.

Now a days, the endometrium pattern is simply described as multilayered or non-multilayered. Serafini[16] has shown that a multilayered pattern to be more predictive of implantation than any other parameter measured.

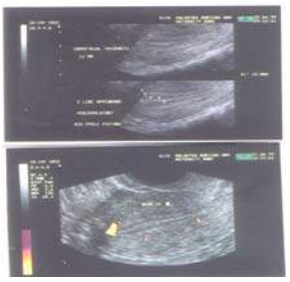
Color flow Doppler of Endometrium and uterine arteries[17] have been extensively studied by Steer et al and they have found that no pregnancy occurred if the P.I. of uterine artery was >3 and there was no spiral artery blood flow in the endometrial zones.

Color Doppler : Addition of color Doppler studies in evaluation of endometrial blood flow patterns and uterine artery flow pattern enabled us to evaluate the physiology of the endometrium. Various scoring systems have been proposed for prediction of implantation by color doppler. The most popular is Appelbaum's USSR [18]. Estrogen produces a vasodilatory effect on the uterine arteries. It has been seen that R.I., P.I. of uterine artery drops with increasing estradiol levels. (Fig. 23 : Uterine A)



(Fig. 23 : Uterine A)

Depending on the vascularisation of the endometrial layers the late proliferative triple line endometrium is divided into four zones [19]. (Fig. 24 : Uterine zones)



(Fig. 24 : Uterine Zones)

Zones

1. 2 mm thick area surrounding zone 2.
2. Hyperechoic outer layer.
3. Hypoechoic inner layer.
4. Endometrial Cavity

Applebaum's USSR Scoring System (UBP)

Ultrasound and the UBP[18]

Ultrasound offers a simple, reliable, reproducible, quick and non-invasive method of assessing the female pelvic region, especially follicular and endometrial growth.

The qualities of the uterus – i.e. the UBP – can be determined using transvaginal colour Doppler sonography (TV-CDS). According to Applebaum,1 certain sonographic qualities of the uterus are noted during the normal mid-cycle. These include :

- Endometrial thickness ≥ 7 mm in greatest anterior-posterior (A-P) dimension (full thickness measured from the myometrial endometrial junction to the endometrial myometrial junction).
- Triple layered ('5-line') endometrial appearance.
- Myometrial contractions causing a 'wave like' motion of the endometrium.
- Homogeneous myometrial echogenicity.
- Uterine artery blood flow < 3.0, as measured by pulsatility index (PI) on Doppler.
- Blood flow within zone 3 (hypoechoic inner layer; see table 1) of the endometrium on colour Doppler.
- Myometrial blood flow internal to the arcuate vessels (seen on gray scale examination).

Table 2. Appelbaum's uterine scoring system for reproduction (USSR)

Parameter	Determination	Score
Endometrial thickness (mm)	< 7 7-9 10-14 > 14	0 2 3 1
Endometrial layering	No layering Hazy 5-line appearance Distinct 5-line appearance	0 1 3
Endometrial motion [no. of myometrial contractions in 2 minutes (real time)]	< 3 ≥ 3	0 3
Myometrial echogenicity	Coarse, inhomogenous Relatively homogenous	1 2
Uterine artery Doppler flow (PI)	2.99-3.0 2.49 < 2	0 1 2
Endometrial blood flow in zone 3	Absent Present, but sparse Present multifocally	0 2 5
Myometrial blood flow (Gray scale)	Absent Present	0 2

Values assume a technically adequate ultrasound examination with no abnormalities of uterine shape or development, no other gross uterine abnormalities (e.g. significant masses), and a normal ovarian cycle (e.g. without evidence of ovarian uterine dys-coordination).[1]

Care to be taken while eliciting and recording uterine biophysical profile by ultrasound.

Ultrasound and the UBP[(18)]

Special care is needed in determining the UBP with ultrasound and colour Doppler imaging; the following guidelines are recommended :

1. If necessary, use both transabdominal ultrasound and TVS to determine the presence of a '5-line' endometrial appearance. Depending on uterine position, a 5-line appearance may be seen on one and not the other (and vice versa).
2. Do not 'rush' the colour Doppler study. Endometrial blood flow is of low velocity; if the sweep through the endometrium is too rapid, flow may not be seen. Additionally, endometrial blood flow is somewhat 'mercurial' – it may seem to 'come and go', and appear in some areas and not in others.
3. Try to make the endometrium as specular (reflective) as possible, using the techniques of manual manipulation of the anatomy and probe pressure to achieve this.
4. Scan endovaginally both coronally and sagittally – there may be a difference in how well the blood flow is imaged.
5. When measuring the endometrium in the A-P dimension, endeavour to do so in the absence of contractions, as these may affect the result. Also whenever possible, obtain the measurement in a standard plane such as when both the endometrial and cervical canals appear continuous.

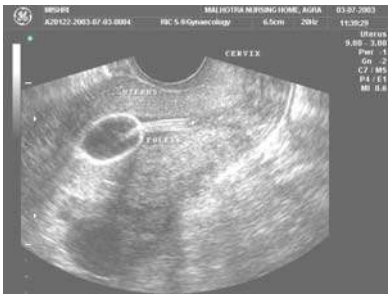
Tubal Evaluation

Normal tubes are isoechoic and are not usually visualised by TVS unless there is fluid contrast. The use of fluid contrast for evaluation of tubes has been described in literature as SION TEST from Mumbai or as SION PROCEDURE of sonosalpingography.

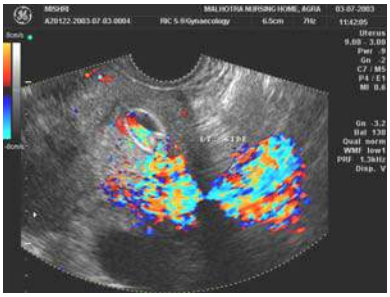
Sonosalpingography

Endosonography as a tool for checking the patency of fallopian tubes was an expected development with great strides taken within the field of Gynaecology. Sonosalpingography also known as Sion Test, used transvaginal sonography to confirm the tubal patency by visualizing the spill of fluid from the fimbrial end of fallopian tubes. Fallopian tubes are isoechoic and cannot be normally seen on Ultrasound unless pathological or fluid surrounds the tubes. We propose to perform this test not as a substitute for Hysterosalpingography or laparoscopy but as an noninvasive, cheap outdoor screening procedure in patients of infertility.

No. 8 Fr. Foley's catheter is put inside the uterus the bulb is inflated with 2 ml of distilled water. Prior to procedure the patient is asked to evacuate the bladder and base line vaginal scan is performed. 20-60 ml of solution containing ciplox, hylase and dexamethasone is taken in 50 ml catheter tip syringe and pushed via foleys catheter and spill is studied from the fimbrial end. (Fig. 26, 27)

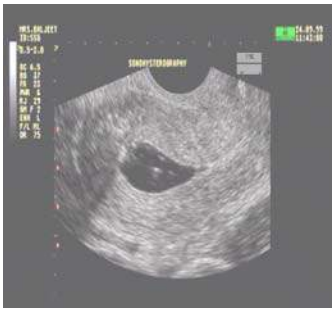


(Fig. 26)



(Fig. 27)

The foley's bulb is then deflated and some saline is pushed slowly to evaluate the uterine cavity as sonohysterography. (Fig. 28)



(Fig. 28)

We have done the Sion Procedure in the patients of suspected pelvic factors. In this we have flooded the pelvis using the same fluid about 200-300 ml, pushed via Foley's catheter and visualized the fallopian tubes.

Sonosalpingography is a good noninvasive screening test for evaluating tubal patency. Sonosalpingography however does not replace the good old Hysterosalpingography in certain specific indications.

Laparoscopy has its additional advantage of having a therapeutic value also.

Sion procedure has an additional advantage of visualising pelvic adhesions & tubo-ovarian mobility.

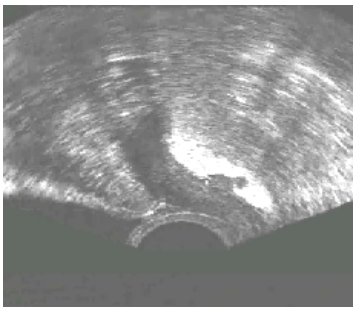
TABLE NO. : COMPARISION OF 3 PROCEDURES

Factors to be assessed	**H.S.G.**	**Laparoscopy**	**Sonography**
Cervix			
Cong. Abnormalities	+	–	+
Cervicities	–	–	+

Uterus			
Cong. Abnormalities	+	Hysteroscopy	+
Myometruim	–	–	++
Endometrium	–	–	++
Tubes			
Morphology	+	+	+
Mobility	–	+	+
Patency	+	+	+ SSG
Ovaries			
Morphology	–	+	+
Follicles	–	+	++
Adhesions	–	++	+
Pouch of douglas	–	+	+
Cost/Time	++	+++	+
Radiation/GA	+	+	–
Therapeutic Value	–	++	+

Hy Co Sy Hysterosalpingo contrast sonography the use of positive contrast media offers much more information rather than the use of sterile saline as negative contrast[19].

Echovist (SHU 45-4; Sharing AG, Berlin) is an ultrasound contrast medium consisting of a suspension of monosaccaride microparticles (50% galactose, 2 µm diameter) in a 20% aqueous solution of galatose. Whenever this immediately reconstituted media is injected in the uterus through a Foley's catheter (No. 8) or a plastic HSG cannula under T.V. scan, the tubal patency and [20] [21] uterine cavity can be accurately studied. (Fig. 29 : Hy-Co-Sy)



(Fig. 29 : Hy-Co-Sy)

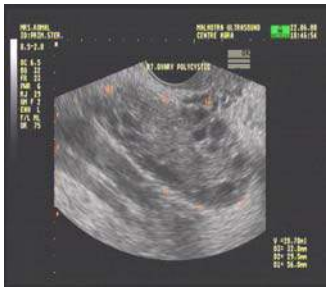
Transvaginal HyCoSy is a diagnostic technique for the investigations and D.D. of uterine cavity pathologies and for tubal patency.

OVARIAN EVALUATION BY ULTRASOUND

Ovary probably is the most frequently scanned organ by ultrasound in an infertile woman. Determination of ovarian status and follicle monitoring are one of the first steps in the evaluation of an infertile woman. It should be kept in mind that a detailed history including menstrual history is very essential for correlating findings of TVS.

Ovarian Study

By ultrasound the ovary is fairly easily recognised usually lying in the ovarian fossa and recognised in front of the iliac vessels when the T.V. probe is placed in the vaginal fornices. Sometimes a bimanual examination improves the image by bringing the ovary nearer to the probe. (Fig. 30 : Normal ovary in front of iliac vessels)

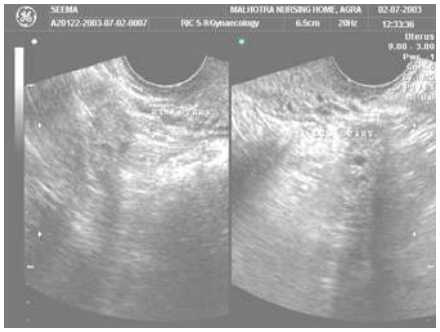


(Fig. 30 : Normal Ovary in Front of Iliac Vessels)

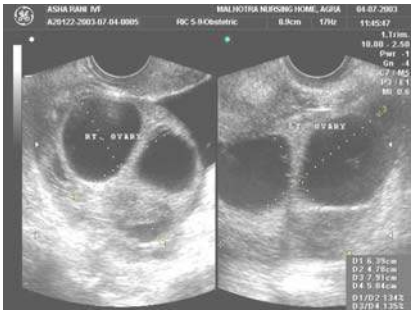
The ovary is imaged for its morphology (Normal, Polycystic or Multicystic), for its abnormalities (cysts, dermoids, endometriomas, tumours etc.), for its follicular growth in ovulation monitoring and for evidence of ovulation and corpus luteum formation and function.

Follicular development

There are often a few follicles (less than 10 mm in diameter) that can be imaged throughout the menstrual cycle and even during menstruation and these preantral follicles are too small to be imaged. Under the influence of follicle stimulation hormone (FSH) released by the anterior pituitary gland in response to pulsatile GnRH during the early part of the menstrual cycle, a few follicles will undergo progressive development. Granulosa cells in developing follicles will secrete increasing amounts of estrogen and follicular fluid, and the follicles will increase in size. As follicular stimulation progresses, one or occasionally two follicles will continue to develop into the dominant follicle(s). Many of the developing follicles will not pass the development stage of 10 to 14 mm diameter before they degenerate. Hackeloer et al[22] noted a linear increase in the size of the dominant follicle through a normal menstrual cycle. Developing follicles destined to ovulate increase in size by 2 mm/day and reach a maximum diameter of 16 to 33 mm before ovulation. Selection of the dominant follicle is thought to occur by cycle days 5 to 7 but is not apparent sonographically until cycle days 8 to 12. Other antral follicles of the developing cohort will generally undergo atresia and will not exceed 14 mm in diameter. However in 5 to 11 percent of natural cycles, two dominant follicles may develop, but they are generally in opposite ovaries. Potential ovulatory follicles will have a diameter of 10.5 mm or greater. (Fig. 31 : Preantral follicles) (Fig. 32 : Dominant follicle)



(Fig. 31 : Preantral Follicles)



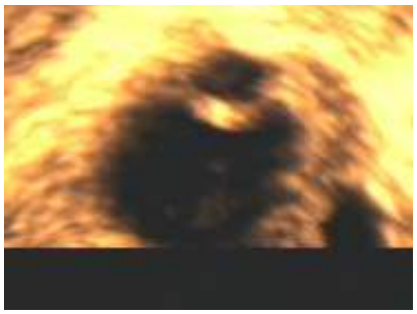
(Fig. 32 : Dominant Follicle)

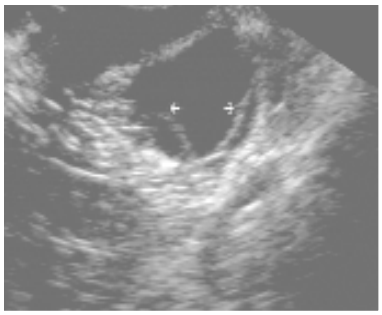
Prediction of Ovulation

A small echogenic mass that is thought to represent the cumulus oophorus may sometimes be noted projecting into the follicle. Visualisation of the cumulus oophorus has been reported in 80 percent of follicles greater than 17 mm in diameter. Ovulation is reported to be within 36 hours of seeing the cumulus. However Zandt-Stastny[23] have challenged this view and have noted that the cumulus is only 100 to 150 μm size and would not be expected to be visualized by sonography. Zandt Stansky et al have suggested that structures imaged in follicles thought to represent the cumulus are artifacts. After the LH surge, the theca tissue becomes hypervascular and edematous and the granulosa cell layer begins to separate from the theca layer. This is appreciated sonographically as a line of decreased reflectivity around the follicle. Picker et al[24] in 1987 suggested that this sonographic sign means impending ovulation within 24 hours. Jaffe and Ben-Aderet[25] in 1984 described the same sign, which they called a double contour and suggested that it occurs a few hours before ovulation. Within 6 to 10 hours before ovulation separation and folding of the granulosa cell layer produces a crenation or irregularity of the lining of the follicle. This has also been suggested as sign of impending ovulation. Unfortunately, despite the fact that there are a number of sonographic signs that have been described to precede ovulation, there is currently no sonographic sign that predicts exactly when ovulation will occur, the signs only give evidence that the time of ovulation is nearing. The mean peak diameter before ovulation reported by Kerin et al[26] was 23.6 + 0.4 mm. However there is considerable differences in the same.

Hence the potential signs of Impending Ovulation are :

- Presence of a dominant follicle (usually more than 16 to 18 mm)
- Anechoic area, double contour, around the follicle (possible ovulation within 24 hrs)
- Separation and folding of the follicle lining (ovulation within 6 to 10 hrs)
- Thickened proliferative endometrium (described later)





(Fig. 33 : Dominant Follicle Showing Cumulus) **(Fig. 34 : Double Contour)**

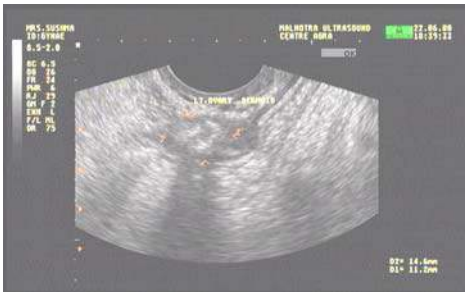
Confirming Ovulation

Sonography does appear to be very reliable in confirming ovulation once ovulation has occurred. Disappearance of the follicle is noted in 91 percent of cases after ovulation and a decrease in follicle size occurs in another 9 percent. Other signs suggesting that ovulation has occurred are the appearance of cul-de-sac fluid, particularly when it was not present in a previous scan, or the development of intrafollicular echoes suggesting the formation of a haemorrhagic corpus luteum.

Ovary in Anovulatory cycles

In an anovulatory cycle, ultrasound imaging of the ovaries will reveal either lack of any follicular development, particularly in the hypogondotropic hypogonadal patient with type I or a few non ovulatory (less than 11 mm) follicles. A dominant follicle larger than 16 mm in diameter will not develop. A cyst may also be associated with anovulation. Anovulation with PCOD will often have enlarged ovaries greater than 8 cm^3 in volume with multiple small subcapsular follicles less than 10 mm in diameter. However, normal sized ovaries do not rule out PCOD. Anovulation can be diagnosed when serial scans do not show development of a follicle. A mature corpus luteum is noted sonographically in about 50 percent of patients after ovulation. If pregnancy does not occur the corpus luteum generally degenerates and disappears just before menstruation. Corpus luteum cysts may be 4 to 6 cms in diamter and occasionally even large but are more commonly 2.5 to 3 cms in diamter. They may persist for 4 to 12 weeks and may be responsible for supressing normal follicular development until they resolve.

(Fig. 35 : Ovary lacking any follicles)



(Fig. 35 : Ovary Lacking Any Follicles)

In PCOD the ovaries are increased in size. The mean volume of the ovary is 12.5 cms^3 with a range from 6 to 30 cms^3. The classical anatomic criteria are not present in all patients with clinical or endocrine findings suggestive of PCOD. Therefore, an ultrasound showing ovarian enlargement can help make the diagnosis, but a normal ultrasound examination with normal size ovaries does not rule out PCOD. If the clinical

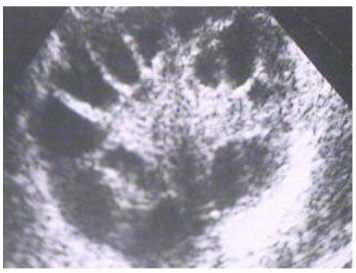
or biochemical abnormalities characteristic of the syndrome are present. Ultrasound may also suggest the diagnosis of PCOD in a patient with normal sized ovaries and the clinical and or endocrine criteria of PCOD by confirming anovulation :

Enlarged ovary (more than 8 cms).

Multiple small cysts (0.2-0.6) (Fig. 36).

Anovulation (lack of follicular development) (Fig. 35).

Resting or follicular endometrium (Fig. 20).



(Fig. 36)

Endometrium

The endometrial cavity should be visualizable as a separate entity within the uterus in virtually all menstruating patients. The endometrial cavity is generally centrally located in the uterus. The cyclic histologic changes and changes in thickening of the endometrium with hormonal stimulation are well known. This cyclic changes of the endometrium can be imaged using transvaginal ultrasound during the different phases of the menstrual cycle. The hormonal and ovulatory status of the patients can be assessed by evaluating the endometrial patterns.

Sakamoto[6] in 1985, described the characteristic sonographic images noted through the menstrual cycle. The proliferative endometrium is characterized by (a) the presence of a well defined three line sign, (b) a hypoechogenic functional layer, and (c) a minimal or absent posterior acoustic enhancement. The three line sign is formed by the central hyperechoic reflection representing the endometrial cavity and the additional hyperechoic reflection representing the thin developing layer of endometrium. There is also a surrounding hypoechoic halo. During the luteal phase, the endometrium is hyperechoic, with posterior enhancement and absence of the three line sign and halo.

Early proliferative phase : The anechoic central echo noted during early menses is replaced by a hyperechoic central line and the endometrium begins to thicken, forming the three line sign. The general hypoechogenic character of the functional layer of the proliferative endometrium is thought to be related to the simple configuration of the glands and blood vessels. The outer lines represent the endometrium and the interface between the endometrium and myometrium. These outer lines may thicken as the estrogen stimulation increases and the follicular phase progresses and blends into a thickened hyperechoic endometrium during the secretory phase. In the follicular phase, the halo which is about 2 mm thick and surrounds the endometrium, is present. There is no posterior enhancement. A follicular phase endometrium greater than 6 mm thick has been associated with a serum estradiol level over 200 pg/ml and a developing follicle greater in diameter.

Late proliferative phase : There is continued thickening of the endometrial echo complex in the late proliferative phase. The halo is still present. The endometrial complex is still imaged as three parallel lines, but the outer lines may begin to thicken. The total endometrial thickness increases and may reach 109 mm or greater in total thickness. There is no posterior enhancement. Cervical mucus may occasionally be imaged as hypoechoic density in the endocervix near the time of ovulation.

Luteal phase : In the luteal phase the endometrium is thickened and is imaged as a homogeneous hyperechoic density with posterior enhancement and loss of the surrounding halo. The three line sign is gone. The rate of increase of thickness slows and the endometrial echo complex soon achieves its greater anterior posterior dimension. The echogenicity of the endometrium becomes hyperechoic after coiling and lengthening of the endometrial glands and the production of mucus and increased tortuosity of the glands and blood vessels. Interestingly, acoustic enhancement is usually associated with cystic or fluid filled structure that are hypoechoic, yet is characteristically seen posterior to the luteinized endometrium. Acoustic enhancement occurring posterior to a hyperechoic structure is unusual and is thought to be related to the increased vascularity of the endometrium. Posterior enhancement is assessed in the anterior posterior pelvic (AP-Pelvic) image plane, since some posterior enhancement may be noted in a trans pelvic (T-Pelvic) plane even during mid to late follicular phases. Although it is not known why enhancement is noted in the T-Pelvic plane, one explanation endometrium as small anechoic areas may be noted in the endometrium. These probably represent areas of hemorrhage into the endometrium with endometrial degeneration and herald onset of vaginal bleeding.

Minimally stimulated or single line endometrium : Patients with low estrogen or excess androgen have generally have a single line endometrium similar to a late menstrual endometrium. Care must be taken when interpreting or reporting such measurements as to whether full thickness of both layers of endometrium or only one layer is being used. Since it is generally simpler just to measure the full endometrial thickness this is usually the measurement reported. The average thickness of the proliferative endometrium to be 8.4 mm + 2.2 mm while the secretory endometrium is 9.6 + 3.4 mm.

Endometrial motion : The endometrium can be seen to move during real time ultrasonographic imaging. This movement can be quite impressive when first seen.

ROLE OF TRANSVAGINAL COLOR DOPPLER IN INFERTILITY

The advent of transvaginal Color Doppler Sonology has added a new dimension to the diagnosis and treatment of infertile female. Color Doppler innovation is a unique non-invasive technology to investigate the circulation with organs like uterus and ovaries. Dynamic changes occur almost every day of the menstrual cycle in a reproductively active female. These events are picked up very well by transvaginal Color Doppler and definite conclusions can be drawn regarding the diagnosis, prognosis and treatment of infertile patients. As the vaginal probe lies close to the organs of interest various vessels supplying these structures can be studied in detail like the uterine artery, ovarian artery and their branches.

Study of Menstrual Cycle by Color Doppler

It is very important to study the whole of the menstrual cycle by transvaginal color Doppler during the evaluation of infertility. It provides vital information about follicular dynamics like blood flow to the growing follicle, the vascular supply of the endometrium and corpus luteum vascularization which are very important for a successful outcome in terms of pregnancy.

Changes in the ovary

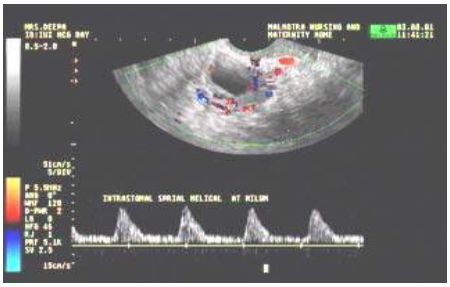
The ovaries are situated on either side of the uterus and measure about 2.2 to 5.5 cms in length, 1.5 to 2.0 cms in width and 1.5 to 3.0 cms in depth and are recognized by the presence of follicle of different sizes. The blood supply is by ovarian artery via the infundibulo pelvic ligament and ovarian branch of the uterine artery. There is

anastamosis between the two sources of blood supply. The primary and secondary branches of the ovarian artery grow along with the development of the follicle. Dominant follicle within the ovary can be recognized by transvaginal color Doppler by day 8th or 10th of the cycle by a ring of angiogenesis around it, when compared to the subordinate follicles which do not demonstrate this. These vessels become more abundant and prominent as the follicle grows to about 20-24 mm in size. (Fig. Follicular blood flow vascularity)

The Phases are as follows

Early follicular (Day 5-7). Late follicular (Day 11-13), Early luteal (Day 15-17) and late luteal (Day 26-28). In general the index values are high in the early part of menstrual cycle and fall as ovulation approaches. According to KURJAK et al the RI in the early proliferative phase is 0.54 +/û 0.04 and declines the day before ovulation when it is about 0.44 +/û 0.04 (9).

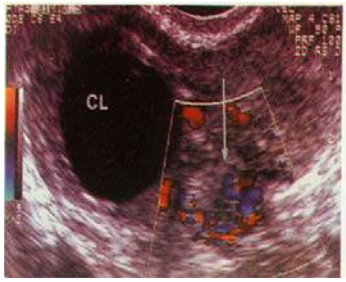
This is the best time for administration of surrogate HCG. A marked increase in peak systolic velocity with a relatively constant RI of 0.44 +/û 0.04 in the proliferative phase and starts decreasing as ovulation approaches. It starts to fall a day before ovulation and the lowest value of 0.84 +/û 0.04 is seen on the day 18 of the menstrual cycle that is around the time of the implantion window. (Fig. 37 : R. I. of ovarian a.)



(Fig. 37 : R. I. of Ovarian a.)

Luteal Phase Changes in Ovarian Vascularity

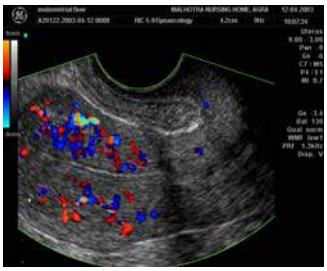
The functional capacity of the corpus luteum is assessed by the low impedance flow and the abundance of vessels around it. Mature corpus luteum is a highly vascularized structure with a low RI of 0.44 +/û 0.04. In patients with corpus luteum defeciency the vascularity is not optimal and the RI is raised to around 0.59, with decreased diastolic flow. If pregnancy occurs then low RI of 0.50 continues. (Fig. 38 : R. I. in Luteal phase)



(Fig. 38 : R. I. in Luteal Phase)

Secretory Changes in the Endometium

Michael Applebaum in his study with transvaginal color Doppler divided the endometrium and periendometrial areas into 4 zones. In the study conducted by him no pregnancy was reported in IVF patients unless vascularity was demonstrated in Zone III or with in Zone III or IV prior to transfer. (Fig. 39 : Vascularity of endometrium)



(Fig. 39 : Vascularity of Endometrium)

Doppler Assessment of Uterine and Ovarian Flow in Infertility and IVF

GOSWAMY ET AL found absent diastolic flow in infertility patients and with severe problems and even reversal of diastolic flow.

Role of Transvaginal Color Doppler in other Conditions Associated with Infertility

Luteinized Unruptured Follicle

This condition is recognized by serial ultrasonography to monitor the growth of follicle, with failure to see expected changes at the time of ovulation.

The typical blood flow pattern seen in the corpus luteum is absent.

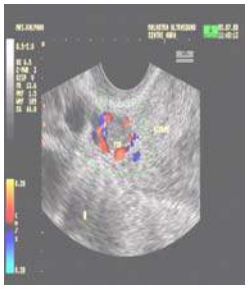
Luteal Phase Defect

This is due to decreased vascularisation of corpus luteum. The three to seven fold increase in blood supply is necessary to deliver, the steroid precursors to ovary and removal of progestrone as shown in experimental animals.

An increasing corpus luteum resistance index indicates less chances of embryo survival, specially within first 8 weeks of pregnancy.

Fibroid

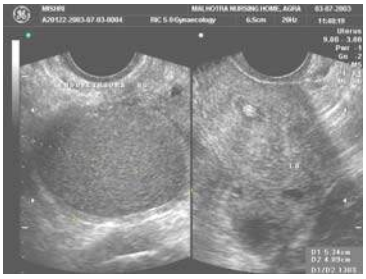
To Define the borders of fibroid color Doppler is of real help as the vascular supply at the periphery of the leiomyoma can be delineated very well. Good vascularity denotes a favourable response to GnRH if used before laparoscopic surgery. (Fig. 40 : Vascularity in a fibroid)



(Fig. 40 : Vascularity in a Fibroid)

Endometriosis

On Gray scale scan endometrioma is seen as a homogeneously echogenic intraovarian mass. Color Doppler may demonstrate the flow around and not within the endometriotic cyst. (Fig. 41 : Blood flow to endometrioma)



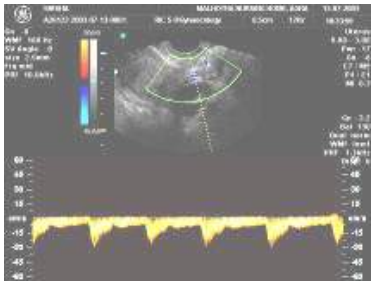
(Fig. 41 : Blood Flow to Endometrioma)

Tubal Causes

During active phase of PID low impedance blood flow signals are usually detected and after effective antibiotic therapy flow tends to return to normal. In the absence of this change surgery is indicated.

Polycystic Ovarian Disease (PCOD)

Contrary to the normal ovarian blood flow, which is seen around the growing follicle, PCOD subjects show abundantly vascularized stroma. Waveforms obtained from the ovarian tissue showed a mean resistance index of 0.54 without cyclical change between repeated examinations. (Fig. 42 : Blood flow in PCO)



(Fig. 42 : Blood Flow in PCO)

Uterine Factor

The possibility of decreased uterine blood flow may be associated with infertility as already discussed in proceeding paragraphs. GOSWAMY ET AL depicted in their study that uterine artery indices which were high in failed IVF cases improved after the patients were put on oral oestogen therapy and pregnancy rate improved when compared to those who did not get this treatment.

Color Doppler and its Contribution Towards in Vitro Fertilization

During stimulation protocols color Doppler ultrasound has its greatest contribution in monitoring follicular development and guiding oocyte harvesting procedures. The use of color Doppler ultrasound can occasionally be of help as it avoids accidental puncture of iliac vessels and also vessels on the surface of ovary.

Avoidance of Ovarian Hyper Stimulation Syndrome (OHSS)

In a stimulated cycle resistance of the intraovarian vessels measured by transvaginal color doppler correlates well with number of follicles, that is those with more than 15 mm size. This correlation exists even during the early follicular phase, when follicular recruitment and development have just started. This suggest that vascularization of the follicles may play a role in their maturation from early follicular phase onwards. This study in the early follicular phase can prevent OHSS.

Optimal Conditions for Embryo Transfer

As shown in a recent work by CAMPBELL it is possible to calculate the probability of pregnancy by using PI values of uterine artery on the day EMBRYO TRANSFER. Highest probability of pregnancy was predicted for patients who had medium values for PI. Those with high PI had failure rate upto 35%. In other words the lower the PI value more the chance of pregnancy. Steer et. al. have shown that if P.I. is > 3 before E.T. no pregnancy results.

ULTRASOUND GUIDED ASSISTED REPRODUCTION TECHNIQUES

Historical Review

The ultrasound guided oocyte aspiration was initially peformed transabdominally through the full bladder, or directly, through the anterior abdominal wall. Subsequently, transvesical and pereurethral approaches were developed. However, the first description of oocyte collection with Transvaginal transducer was described by Wikland in 1985.

VAGINOSONOGRAPHIC FOLLICULAR ASPIRATIONS

Introduction

The impact of Transvaginal ultrasound has been tremendous since its inception, as it has enabled the operator to visualise pelvic organ as a ôclose-upö shot. Also, the high resolutions it offers and the consequent high definition image, allows for better and perfect diagnosis of the pathology, than with the Transabdominal ultrasound. Having said this, it is also important to note, at the stage, that the operator has to be well-versed with the orientation and the planes of Transvaginal ultrasound to achieve the above.

The ability to diagnose conditions earlier and better than with Transabdominal Papproach has put Vaginosonography in an enviable position for Interventional procedures to be carried out on the pelvic organs. Thus, the Transvaginal transducer coupled with the biopsy guide, has become of late a crucial weapon in the armamentarium of the Interventional ultrasonologist. Whereas with Transabdominal ultrasound intervention is mainly a free-hand technique, with Transvaginal ultrasound, it

becomes a guided procedure where in the operator can be confident that the needle will follow the path charted out by the software generated biopsy line on the monitor. Also, unlike the former, Transvaginal ultrasound-guided procedures, have the advantage of easy accessibility to the pelvic organs in the needle path.

The dramatic entry of Interventional Transvaginal sonography was made primarily for the purpose of oocyte retrieval in the Assisted reproduction programmes, which was hither to done via. laparoscopy. Hence, Transvaginal sonography, besides diagnosis, also started playing an important role, in the therapeutic field.

VAGINOSONOGRAPHIC PUNCTURE PROCEDURES

Assisted Reproduction Techniques

- In-vitro fertilisation and Embryo transfer. (IVF & ET)
- Gamete Intra-fallopian transfer. (GIFT) & (SIFT)
- Intra-follicular Insemination. (IFI)
- Direct intraperitoneal insemination. (DIPI).
- Peritoneal oocyte sperm transfer (POST).

Patient Preparation

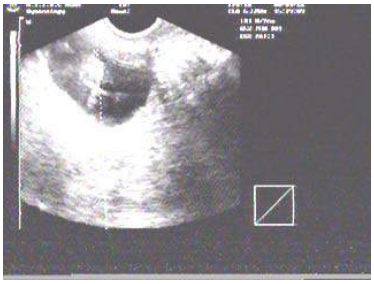
Short General anaesthesia with Ketamine or Pentothal is the preferred mode for Transvaginal sonography-guided aspiration. Sometimes, when there are few follicles, we administer intravenous analgesia, as the punctures are few and procedure is short.

Procedure

Patient is placed in the lithitomy position. Vagina is cleansed with Betadine solution and this is then rinsed thoroughly with Normal saline so that no trace of Betadine remains. Intravenous antibiotic is given intraoperatively.

Transvaginal transducer is cleaned with Absolute alcohol solution and then with Normal saline. It is then covered with a disposable, sterile plastic sheath, same as that used for the Endoscopic video camera. Biopsy guide, which is washed with Normal saline, is attached to the Transducer. The assembly is introduced within the vagina. The software-generated biopsy guideline on the monitor is lined up with the follicle to be punctured. Guage 16/17 Biopsy guide is used for these procedures. The Cook's IVF Ovum aspiration needle (Guage 17) with the tubing and the bung is used for puncture. The bung is introduced into the Falcon test-tube, the other end of tubing is connected to suctions apparatus with foot-controlled device. Suction is adjusted at 100 mm Hg.

Under ultrasound guidance, the needle is introduced into the follicle and fluid is aspirated by applying the suction. A single channel needle is used, curetting of the follicle is done at the end of the aspiration and when the follicle has completely collapsed, the needle guide is realigned to the next follicle and procedure repeated. All the follicles of one ovary are aspirated with a single puncture. If need be, the needle is flushed with culture medium, on completion of one ovary or when there is a bloody tap. After, all follicles are aspirated, including the intermediate ones, the transducer is removed and the guide detached. The vagina is swabbed with normal saline and bleeding from the puncture site looked for. Then, the vaginal transducer is reintroduced and the pelvic area is scanned for active hemorrhage. (Fig. 43 : Ovum pick up)



(Fig. 43 : Ovum Pick Up)

Complication

1. Vaginal puncture hemorrhage. This may result after the needle is removed. Usually vaginal packing and direct pressure upon the puncture site stops the flow and nothing else seems to be necessary.
2. Hemorrhage within the pelvis and/or ovarian follicles. This may be detected by post-procedural ultrasound. However, we have not encountered any catastrophic bleeding into the ovary or from its surface. Careful monitoring is essential.
3. Vascular injuries Internal iliac vessel puntrure is a major complication and laparotomy may have to be done to arrest the hemorrhage.
4. Pelvic infections occur even after the administration of antibiotics.

Embro Transfer

Usually, transcervical embryo transfer is undertaken as a blind procedure. Very rarely, when the cervix is stenosed or tortuous, the embryos are transferred through the myometrium. (SURGICAL EMBRYO TRANSFER-SET)

The patient positioning and initial workup is same as was described for oocyte aspiration. The guideline is aligned with the Endometrial cavity just below the fundus. A 25 cm. 19 gauge needle primed with culture medium is passed through the biopsy guide and then into the uterus. The needle traverses throught the myometrium and then in positioned into the cavity. Injected culture medium is seen transiently separating the anterior and posterior surface of the endometrial cavity. A long embryo transfer catheter loaded with the embryos at its tip, if then passed down the needle into the endometrial cavity. The embryos are injected and the catheter and needle withdrawn. In animals, this procedure has been shown to lead to pregnancy in 70% cases, but the success in humans is very low.

Direct Intraperitoneal Insemination (DIPI) and Peritoneal Oocyte sperm transfer

Direct Intraperitoneal insemination, introduced in 1986, has been used to treat couples with unexplained infertility, cervical mucus hostility, oligospermia, failed donor insemination and women who have ovulated prior to egg collection. The patient is superovulated with CC/HMG or LHRH analogue and HMG.However, the cycle is aborted, or the patient is given alternative of oocyte aspiration followed by embryo transfer or GIFT or POST. DIPI is performed 36 hours after administration of HCG. Patient is placed in lithotomy position, vagina is cleansed with Betadine/Normal saline

and vaginal probe with biopsy guide is inserted. A pool of free fluid is identified and a gauge 18 needle is passed along the guide and into this pool with a single rapid thrust. The patient is usually sedated intravenously. Aspiration of fluid confirms that needle is in position and then, the washed semen sample is injected. We have had success rate of 15% in 25 selected cases for DIPI. But, this procedure does not have a role in the routine treatment of infertility.

Mason and colleagues placed both sperms and oocytes in the pouch of Douglas reasoning that most patients with unexplained infertility will have gamete abnormalities rather than a problem with ovum pick-up by the tubes.

In this technique, Peritoneal oocyte and sperm transfer (POST), the patients are superovulated as in IVF cycles. Follicles are aspirated by the Transvaginal route and when all follicles have been aspirated, the needle is introduced under Vaginal ultrasound guidance, into the pouch of Douglas which is repeatedly rinsed with the culture medium until the aspirated is clear. A long embryo transfer catheter, loaded with three eggs and 4 million prepared sperms, is passed along the needle and its contents injected into the pouch on Douglas. We have done 10 cases of POST in various set-ups and have had a cumulative pregnancy rate of 30% per procedure. This technique is simpler to perform than IVF or GIFT, it has two distinct advantages over DIPI in that the egg release from the follicle is guaranteed and the spare eggs may be inseminated to confirm the fertilising ability,and then transferred.

Direct Intra-Follicular Insemination

This procedure was carried out in a Colombian study wherein the follicles were not aspirated, but the prepared sperm sample was injected directly into the unruptured follicle 34-36 hours after administration of hCG. The study includes a limited population and has shown to have no major complication or sequelae. The pregnancy rate was 20% in this study. However, the possibility of an ectopic gestation (Ovarian/tubal) is there and we must consider this procedure only as a seldom alternative to IUI or GIFT when the patient does not desire intervention.

Ovarian Hyperstimulation is more commonly seen in recent times due to the universal prevalence of Assisted reproduction treatment. Controlled ovarian stimulation with GnRH and HMG/FSH is desired for IVF/GIFT/IUI cycle. However, sometimes, even with careful and close monitoring these may go haywire and result in the hyperstimulated ovaries. It can be classified into Mild, Moderate and severe degree, depending upon the associated clinical and sonographic findings. The presence of free fluid/Ascites is a common feature with Ovaries contained large cysts and measuring 7 cms. in diameter or above. Vaginal sonography is necessary to provide a constant monitoring for these cysts, as well as for free fluid estimation. Vaginosonography-guided aspiration of the free fluid from the pouch of Douglas with the patient in reverse trendelenburg position is considered as a mode of treatment, and should be carried out in cases of Mild to Moderate degrees of hyperstimulation. The ovaries are very vascular and should not be touched but treated conservatively. If the cysts persist after 2 or 3 cycles then they may be aspirated under Vaginal sonography guidance. (Fig. 44 : OHSS).

Prevention

When the Ovaries are stimulated with ovulation induction hormones in an Assisted reproduction cycle, monitoring of the follicular growth with hormonal assays is done on a daily basis. If there are multiple intermediate follicles in either or both ovaries and/or if the serum E2 level when 3 or more follicles reach dominant size of 18 mm is more than 3500 pg./ml. then the hCC administration is withdrawn. This would prevent the blow-up of these stimulated ovaries and thereby reduce the potential for ovarian hyperstimulation. Alternatively, these follicles could be aspirated without prior hCG administration and the oocytes collected.

In managing ART Pregnancies and their complications, transvaginal ultrasound is an important tool especially in the diagnosis of early ART viable pregnancies and their number and implantation site. Bleeding and other complications of ART pregnancies are best managed under ultrasound guidance.

Complications like ectopic pregnancy, hetrotrophic pregnancy and high order multiple pregnancy also essentially need to be diagnosed and closely monitored by transvaginal ultrasound scan. (Fig. 45 : Ectopic pregnancy) (Fig. 46 : High order multiple pregnancy)

Conclusion

Ultrasound today has revolutionised the practice of infertility. To practise infertility and ART without a transvaginal scan is unthinkable in this modern era of technology. The addition of color has given us a good insight to the physiology of female pelvis. Color doppler today helps in prediction of success and complications. The addition of interventional procedures have simplified ART to an out patient procedure and prevented major operations in cases of ectopic pregnancy and ovarian cysts. 3-D has now given us a new dimension of volume estimation and sculpture like images.

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ULTRASONOGRAPHY OF THE OVARIES AND FALLOPIAN TUBES

DR. NARENDRA MALHOTRA
DR SAKSHI TOMAR

High-resolution transvaginal sonography (TVS) has been widely available since mid 1980 and has gained acceptance as an integral part of gynaecologic and easily obstetric sonography examinations. In many ultrasound laboratories, the standard examination of female pelvis consists of transvesical-transabdominal(TAS) combined with TVS and in some cases transvaginal colour flow Doppler (TVCFD).

Ovaries

Ovaries are ellipsoidal in shape, position is variable especially in multiparous women.In nulliparous females ,ovaries are situated in the ovarian fossa (also known as fossa of Waldeyer). The ovarian fossa is situated on the lateral pelvic wall and is bounded anteriorly by the obliterated umbilical artery , ureter and internal iliac artery posteriorly and the external iliac vein superiorly. Ovarian lesions are a cause of great concern because of their malignant potential and the limited ability to distinguish accurately between benign and malignant tumors prior to surgery.

Fallopian Tubes

These originate from the lateral uterine angles towards their respective ovaries.These measure approximately 7-12 cm in length and are a few mm wide. Normal fallopian tubes are not visualized by ultrasound unless abnormal or surrounded by fluid.

OVARIAN SONOGRAPHY

The frequent anomaly of the ovaries recognized by ultrasound is the polycystic ovaries. The most common type of cystic adnexal lesions are functional ovarian cysts.

Classification Of Ovarian Lesion:

Benign Cystic Lesion of Ovarian and Paraovarian Structures

- Functional Cysts

a. Follicular cysts
b. Corpus luteum cysts
c. Corpus luteum of pregnancy
d. Theca luteum cysts

- Surface epithelial inclusion cysts
- Rete cysts
- Lutein cysts

- Ovarian hyperstimulation syndrome(OHSS)
- Polycystic Ovarian Syndrome
- Ovarian Remnant Syndrome
- Neonatal Ovarian Cysts
- Paratubal, paraovarian cysts
- Endometriosis and Endometriomas
- Pelvic inflammatory disease
- Peritoneal inclusion cysts

Ovarian Vascular Lesions

- Ovarian torsion
- Ovarian oedema
- Ovarian vein thrombosis

Ovarian Neoplasms

- Suface epithelial stromal tumours
  a. Serous tumours
  b. Mucinous tumours
  c. Epidermoid
  d. Clear cell tumours
  e. Transitional cell(Brenner's) tumours
- Germ Cell Tumours
  a. Mature cystic teratomas (Ovarian Dermoid Cysts)
  b. Mature solid teratoma
  c. Immature teratoma
  d. Dysgerminoma
  e. Yolk sac tumours
- Sex Cord – stromal tumours
  a. Fibroma
  b. Thecoma
  c. Granulosa cell tumours
- Metastatic tumours

Benign Cystic Lesion of Ovarian and Paraovarian Structures

Functional Cysts

- Follicular Cysts: Thin walled, unilocular 3-8cm in size. Usually regress spontaneously.
- Corpus Luteum Cysts: Commonly complicated by haemorrhage. Thick walled with echogenic contents.
- Corpus Luteum of Pregnency: Corpus luteum of pregnancy may become enlarged and cystic. Needs follow up ultrasound and monitoring

- Theca Lutein Cysts: Usually multilocular results from overstimulation by high levels of circulating human chorionic gonadotrophin (hCG) in trophoblastic disease

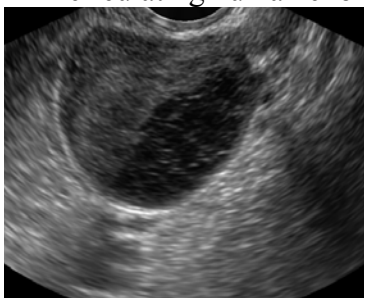

Fig : Transvaginal scan demonstrating a luteinized unruptured follicle with hemorrhagic phenomenon.

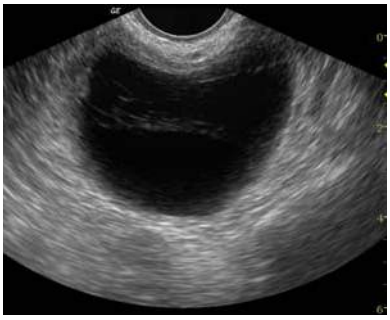

Fig : Transvaginal scan demonstrating a Haemorrhagic Cyst with internal reticular appearance

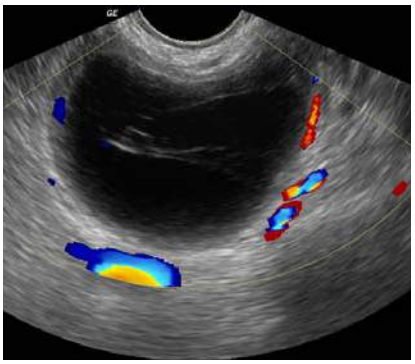

Fig : Transvaginal color doppler scan, same patient showing pericystic flow

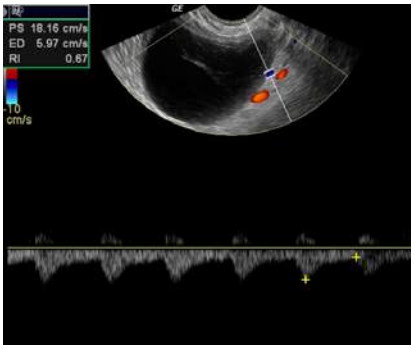

Fig : Transvaginal color Doppler scan. Same patient, spectral shows moderate high signal resistance (RI= 0.67)

Surface Epithelial Inclusion Cysts

These result from cortical invaginations of ovarian surface epithelium. Mostly seen in postmenopausal women , usually multiple.

Rete Cysts

Rare origin, located within ovarian hilum. Indistinguishable from other simple cysts

Lutein Cyst

Ovarian enlargement resulting from the presence of multiple luteinised follicle cysts, secondary to hCG stimulation. Self limiting condition .

USG: Bilaterally enlarged ovaries containing multiple cysts. Cysts may be simple or have haemorrhagic contents

Ovarian Hyperstimulation Syndrome

Seen in women undergoing ovulation induction ,after administration of gonadotrophins followed by hCG or rarely clomiphene alone

USG: Mild to moderate OHSS is characterized by cystic ovarian enlargement (<5 cm in diameter) and a small amount of pelvic fluid. Severe OHSS is characterised by cystic enlargement with abdominal distension and discomfort or pain with or without nausea and vomiting or diarrhoea. Ascites and pleural effusion are seen

Polycystic Ovarian Syndrome

Seen in 16 to 22 per cent of women in their reproductive years and in upto 50 per cent of women presenting to infertility clinics.

USG: Typical polycystic pattern is defined by the presence of 10 or more cysts measuring 2-18 mm in diameter in a single place arranged peripherally around an increased amount of central stroma (Garland sign or Necklace sign)

Greater ovarian stromal blood flow velocity and lower impedance have been demonstrated in women with PCO. The impedance of uterine arteries has been demonstrated to be increased.

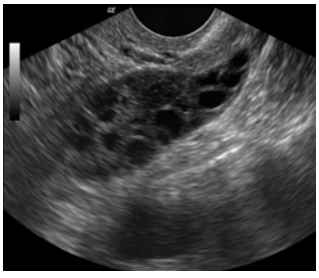

Fig : Transvaginal sonogram of the polycystic ovaries showing small cysts scattered throughout the entire ovarian parenchyma.

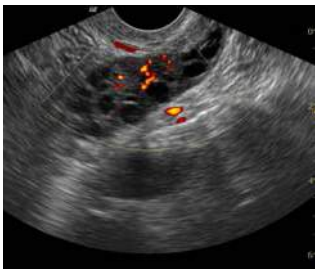

Fig : Transvaginal Color Doppler Scan. Same patient, doppler depicts blood flow signals from enlarged ovarian stroma

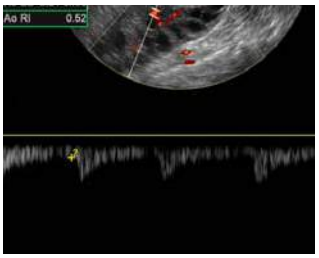

Fig :Transvaginal Color Doppler Scan. Same patient, spectral shows moderate resistance (RI= 0.52) to blood flow.

Ovarian Remnant Syndrome

It is a complication of oophorectomy in patients with distorted anatomy resulting from adhesions and endometriosis, making surgical dissection diffcult; residual ovarian tissue may form a cystic or complex mass

Paratubal ,Parovarian Cysts

These arise from the mesonephric and paramesonephric structures Most common in third and fourth decade, may be complicated by haemorrhage , torsion or rupture

Endometriosis

It is the presence of endometrial tissue outside the endometrium and myometrium.Ovaries uterine ligaments, rectovaginal septum ,cul-de-sac and pelvic peritoneum are the most common sites.

USG: Endometriomas have a variety of appearances ranging from an anechoic cyst to a cyst containing diffuse low level echoes with or without solid components to a solid appearing mass. Presence of a fluid-fluid level , punctuate or linear bright echogenic foci in the wall of the cyst favours the diagnosis of endometrioma. Pericystic vascular location at the level of the ovarian hilus is typical of endometrioma.

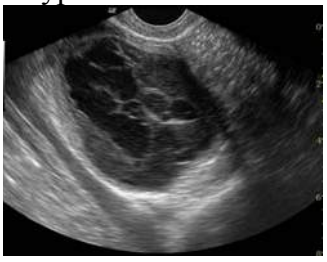

Fig : Transvaginal scan demonstrating a multilocular endometriotic cyst

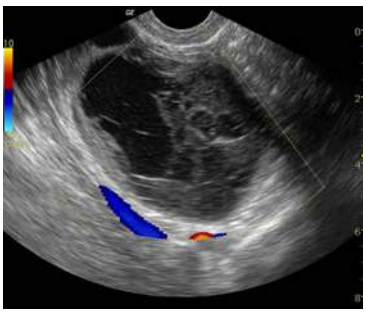

Fig : Transvaginal Color Doppler scan. Same patient, no vascularity is noted

Pelvic Inflammatory Disease

Ovarian involvement in PID is almost always secondary to salpingitis. Sonographic findings may be normal early in the disease course . Timor Tritsch et al described the sonographic findings as:

- Thickening of tube wall >5mm
- 'Cog wheel sign`: Cogwheel shaped structure seen in cross section of tube in acute salpingitis.
- Incomplete septa correlating with folds or kinds in dilated tube.
- Beads-on-a-string`sign: Hyperechoic mural nodules within fluid tube representing flattened and fibrotic endosalpingeal folds.
- Tubo ovarian complex: Ovary cannot be separated from the tube by pushing with the vaginal probe.
- Tubo-ovarian abscess: Conglomerate mass or fluid collection.
- Cul-de-sac fluid.

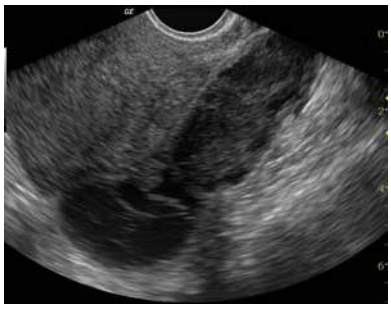

Fig : Transvaginal scan : Complex adnexal mass containing a fluid filled dilated tube and attached ovary.

Fig : Transvaginal Scan of a pseudoseptate, dilated, tortuous tubular fluid filled structure (pyosalpinx) in the adnexa. Ovary was seen seperately

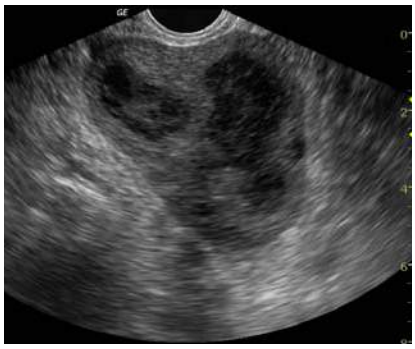

Fig : Transvaginal Scan of a complex adnexal mass that could be interpreted as a tuboovarian abscess

Peritoneal Inclusion Cysts

These are formed by trapping of fluid (which is normally produced by active ovaries) within peritoneal adhesions. A history of trauma, abdominal surgery , PID or endometriosis is common. USG shows the ovary surrounded by septations and fluid and lies inside or in the wall of a large ovoid or irregular anechoic cyst.

Ovarian Vascular Lesions

Ovarian Torsion

Usually caused by ovarian (particularly dermoid) and parovarian cysts. Sonographic appearance depends on the duration, degree of torsion and any associated intra-ovarian mass. USG shows a cystic , solid or complex mass with or without pelvic fluid, thickening of wall. These findings however are non-specific. Enlargement or ovary with absent or markedly diminished ovarian flow is a specific finding.

Massive Ovarian oedema

Accumulation of edema fluid within ovarian stroma, most likely due to torsion of the ovary. A definitive diagnosis of massive ovarian oedema cannot be made on preoperative imaging but should be considered in the differential diagnosis of a solid extrauterine mass in the appropriate clinical setting.

Ovarian Venous Thrombosis

Occurs most often postpartum, may also follow pelvic operations, pelvic trauma-sonographically thrombosed vein appears as an anechoic to hypoechoic tubular mass extending superiorly from the adnexa with absence flow on Doppler. A perivenous phlegmon with increased vascularity may be seen.

Ovarian Neoplasms

Surface Epithelial Stromal Tumours

Serous Tumours:

Benign serous cystadenomas appear as sharply marginated, anechoic masses that may be large and are usually unilocular. Internal thin walled septations and papillary projections may be seen (in borderline tumours)

Serous cystadenocarcinomas are usually multilocular, containing multiple papillary projection and septations, echogenic material is occasionally seen within the loculi.

Mucinous Tumours

Sonographically, mucinous cystadenomas have thicker and more numerous septations and frequently contain fine, gravity-dependent echoes produced by thick contents.

Mucinous cystadenocarcinomas usually appear as large, multiloculated cystic lesions containing echogenic material and papillary excrescences.

Pseudomyxoma peritonei is by far the most common manifestation of atypically proliferating mucinous tumours.

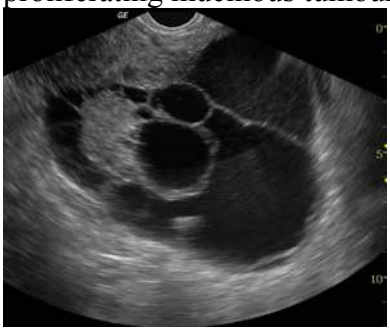

Fig : Transvaginal scan showing a multilocular ovarian tumor presenting thick septa and echogenic content.

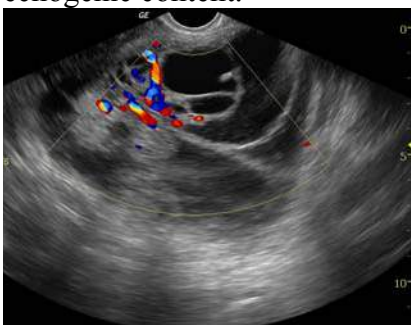

Fig : Transvaginal color doppler scan. Same patient demontrates vascularity within the thick septa.

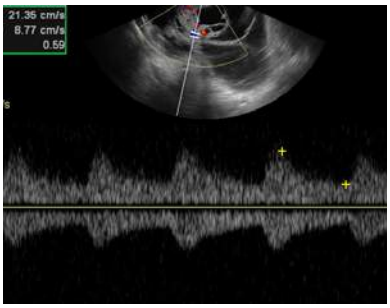

Fig : Transvaginal color doppler scan. Same patient, spectral shows moderate high signal resistance (RI= 0.59) depicted from a thick septa suggest the benign nature of the lesion. The tumor was diagnosed as a mucinous cystadenoma .

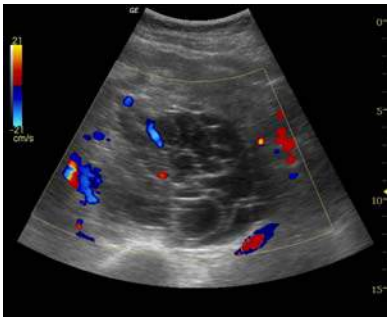
Fig : Transabdominal Scan showing a complex adnexal mass with thick septae. Vascularity is seen within the septa.

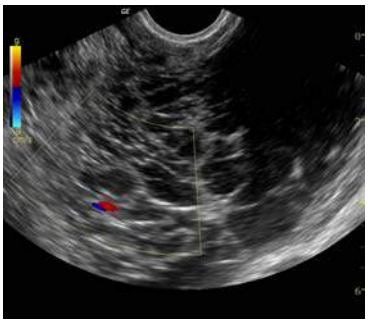
Fig : Same patient, Transvaginal color doppler scan.

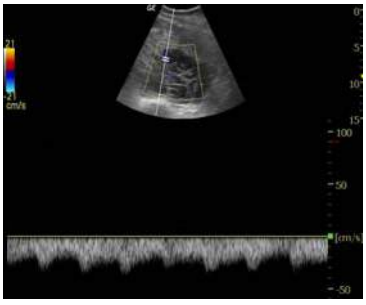
Fig : Transabdominal Sonogram. Same patient, spectral shows low resistance, high diastolic flow. Histology confirmed a malignant tumor.

Endometrioid Tumours

USG : These tumours are seen as cystic masses containing papillary projection.

Clear Cell Tumours

Sonographic features of clear cell tumours are non-specific, usually seen as complex, predominantly cystic masses.

Transitional Cell Tumours (Brenner's Tumour)

These are usually small (1-2 cm), hypoechoic and solid. Extensive calcification may be seen cystic areas are unusual.

Germ Cell Tumours

1. Mature cystic teratomas (Ovarian Dermoid) : USG features include the presence of regional diffuse bright echoes with or without posterior acoustic shadowing, hyperechoic lines or dots, shadowing echodensity and a fluid level.
2. Immature teratomas : These are rare malignant tumours, usually large and predominantly solid.
3. Struma Ovari : This term is used for tumours (Mature cystic teratomas) containing thyroid tissue.
4. Dysgerminoma : Sonographically, the presence of a solid ovarian mass with a multilobulated appearance separated by fibrovascular septa, is highly suggestive.
5. Yolk sac tumours : Similar in appearance to that of Dysgerminomas.

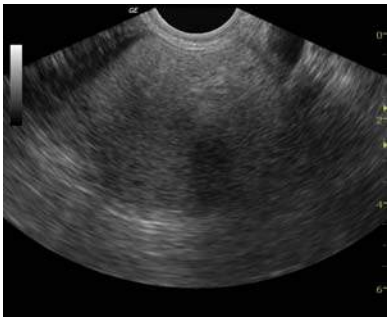

Fig : Transvaginal scan of an adnexal mass showing an echodense pattern with faint posterior shadowing. A Dermoid Cyst.

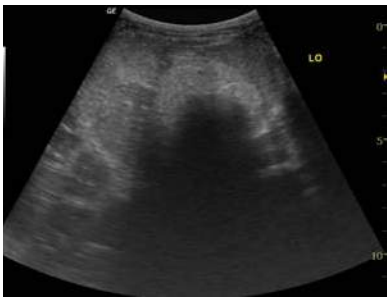

Fig : Transvaginal scan of an adnexal mass showing dense echopattern with an echogenic tubercle and posterior shadowing.

Sex Cord-Stromal Tumours

1. Fibromas : Meig's Syndrome complicates about 1 percent of ovarian fibromas and is defined as ascites and pleural effusion accompanying a fibrous ovarian fumour usually a fibroma. Two typical appearances have been described sonographically. The first has features similar to that of uterine fibroid. Second appearance is that of a hypoechoic mass with substantial attenuation.
2. Thecoma : Sonographically, these tumours are similar in appearance to fibromas.
3. Granulosa cell tumours : Sonographically small tumours are predominantly solid, having an echogenicity similar to that to fibroids. Large ones resemble cyst adenomas and are multiloculated and cystic.
4. Sertoli-leydig Cell Tumours : Similar in appearance to granulosa cell tumours.

Metastaic Tumours

Tumour spread to ovaries is by several routes :

1.Direct spread:
2.Through the lumen of fallopian tube onto the surface of ovary .
3.Distant site metastatic deposits via blood and lymphatics.
4.Trans-coelomic dissemination with surface implantation.

USG: Bilateral ovarian enlargement by solid masses is highly suggestive These masses may contain hypoechoic areas that represent cystic degeneration or necosis.

Ovarian Lymphoma: Solid hypoechoic masses.

Evaluation of an Ovarian Mass

1. Morphologic parameters: Benign tumours are usually unilocular, with thin walls & no septa. Malignant tumours are multilocular, have thick or irregular walls or septa, poorly defined borders, mural nodules, solid components and echogenic elements.
2. Doppler parameters: Neovascularity is a feature of malignant tumours and is characterized by low impedance, high velocity flow. Most authors used a cut off for malignancy of less than 0.4 for RI and 1.0 for PI.
3. Colour flow mapping: Peripheral vascularisation appears to be more common in benign tumours, whereas malignant tumours tend to have more centrally located vessels.

   Arrangement of vessels. Is also a helpful discriminator because benign masses tend to have regularly spaced vessels, whereas malignant tumours demonstrate random distribution of vessels.

   Absence of diastolic notch has been associated with malignant tumours.

USG in first trimester

Dr. Rajat Kr. Ray

Ultrasonography,particularly transvaginal sonography,has become the method of choice in examination & evaluation of first trimester of pregnancy.

Indications:-

- Location :-intrauterine or ectopic
- Viability:-whether alive or not?
- Singleton or multiple pregnancy
- Estimation of gestational age
- Probability of consequent demise
- Presence of any associated pathology like fibroids & ovarian tumour
- Evaluation of nuchal translucency & nasal bone screening at ≥ 11weeks

First trimester can be divided into following phases

- Pre & peri-ovulation.
- Cenceptus phase :- 3rd – 5th weeks
- Embryonic phase :- 6th – 10thweeks
- Fetal phase:-≥ 11weeks

With high resolution transvaginal sonography,a tiny gestational sac(GS) with a size of 2-3mm can be detected between 4w1d to 4w3d,which is the first definitive sonographic fiinding of early pregnancy.It is perceived as a small fluid collection sorrounded completely by an echogenic rim,which exceeds echogenicity of myometrium.The position of a normal GS is in the mid to upper uterus.As the sac implants into the decidua,it should be adjacent to the linear central cavity echo complex(intradecidual sign). As the sac enlarges, it gradually deforms the central cavity echo complex giving rise to sonographic appearance of double decidual sac sign, which consists of two echogenic concentric lines surrounding a portion of gestational sac,formed by decidua capsularis & decidua parietalis.Its size increases by 1mm/day.

The first anatomical structure identified within the GS is the secondary yolk sac(YS).By TVS,it can be always seen by 5.5 weeks,when the mean sac diameter(MSD) is 8mm,but by TAS,it will be demonstrated by 7 weeks when the MSD is 20mm.Its size increases between 5-10weeks.It appears as spherical in shape with a well-defined echogenic rim & sonolucent center

Max diameter obtained is 5-6mm (CRL-30-45mm).It is considered abnormal,when:-(1)size is <2mm or >6mm.(2) is calcified,(3) is double,(4)appears wrinkled before 10 weeks.

Embryonic disc is detected initially as a subtle area of focal thickening along the periphery of yolk sac. Threshold of detection is when it measures 1-2mm in length,which corresponds between 5 & 6 weeks & an MSD between 5 & 12mm.As the human eye is sensitive to motion,cardiac activity can occasionally be seen before the embryo itself is identified.Cardiac activity should always be detected when CRL measures 5mm by TVS (MSD of 16mm)& 9mm by TAS(MSD of 25mm).During first trimester cardiac rates vary with GA.By 5-6 weeks,it is 100-115 bpm(beats per minute),which increases to 160-170 bpm

by 9 weeks.If the FHR is <100 bpm,the embryo may result in demise or may develop congenital anomaly.
Amniotic membrane can be visualized by TVS by 5.5wks,but it is routinely detectable when CRL is ≥ 7mm.From this age onwards,amniotic sac diameter & CRL increases by 1mm/day.It is visible as a thin rounded structure encircling the embryo.It is considered abnormal when(a) it is clearly seen or thickness& echogenicity equals that of YS,(b)there is enlarged amniotic cavity related to CRL (Mean Amniotic Sac Dimeter-CRL=0-1mm),or (c) there is an empty amnion with GS >16mm.

Spine can be seen as parallel echogenic lines by 7-8wks.Limb buds can be demonstrated starting from 7^{th} week.Upper limb buds appear earlier followed by lower limb buds.Feet & hands are completely developed by end of 10^{th} week,giving a human-like appearance. fingers & toes are detected in 11^{th} week. Discreet movements can be seen by end of 8^{th} week.By 9^{th} week,trunk begins to elongate & straighten & midgut herniation into umbilical cord is prominent,which occurs at the beginning of 8^{th} week. Midgut should return inside abdomen by end of 11th week & its base should not be >7mm in diameter.Umbilical cord first appears as a linear structure from fetus to chorion frondosum by 8th week.Placenta can be detected by 9^{th} week as a thickening of the hyperechoic rim of tissue around the gestational sac.Sometimes cyst is seen in umbilical cord by 8th week& disappears by 12th week.They are usually singular,closer to fetus with a mean size of 5.2mm & has a normal outcome.Developing rhombencephalon appears as a cystic space in the posterior cranium between 7^{th} & 9^{th} week. By 11-12 weeks, fingers,toes & spine are more clearly visible. Lateral ventricles reach inner skull,choroid plexus fills completely. More accurate BPD measurement can be done,face becomes indistinctly visible, stomach can be visualized in 93%,bladder is visible in 50% cases, in about 50% cases placenta partly or completely occupy lower pole.
If a patient presents with bleeding & a history of amenorrhoea,it may be due to (1)threatened abortion:- living embryo with long & closed cervix,(2)incomplete abortion:-echogenic & vascularised mass within uterine cavity,(3)blighted ovum:-gestational sac of > 16mm (TVS)with no embryo,(4)embryonic demise:-embryo of >5mm with no cardiac activity(TVS),(5)ectopic pregnancy:-empty uterine cavity with evidence of pregnancy outside cavity, or(6)gestational trophoblastic disease:-cavity filled with mole cysts.Intrauterine collection of blood is found in some cases.Prognosis is good if cardiac activity & scan results are normal & poor if there is progressively larger haematoma, advanced maternal age, associated with severe bleeding & pain.
Assessment of gestational age is most accurate in first trimester,as biologic variation in fetal size is minimal.The first structure measured for this purpose is gestational sac.Most authorities use mean sac diameter(MSD) measurement for this purpose,which is the mean of length,width & antero-posterior diameters.The next landmark is yolk sac,the presence of which without an embryo corresponds to 5.5weeks.Once the embryo becomes visible,measurement of choice becomes crown-rump length,which is the greatest embryonic length measured by placing the calipers at the head & rump of the fetus.Gestational age estimated has a maximal error of 3-7 days.
With continued technological improvements,many gross anomalies can now be detected in late first trimester; like large encephaloceles, holoprosencephaly, ectopia cordis, conjoined twins,anencephaly etc.After completion of 10weeks,screening for chromosomal anomalies

can be undertaken by measurement of nuchal translucency,nasal bone,ductus venosus flow,tricuspid regurgitation etc.
First trimester is also the best time to detect any associated pathologies like corpus luteal cysts,other cysts like dermoid & myomas,which are associated with increased loss rate.

Conclusion

USG examination has become the "golden standard" in follow-up of the development & complications of early pregnancy.The essential aim of an early pregnancy scan is not only to diagnose a pregnancy,but also to differentiate between a normal & abnormal pregnancy.

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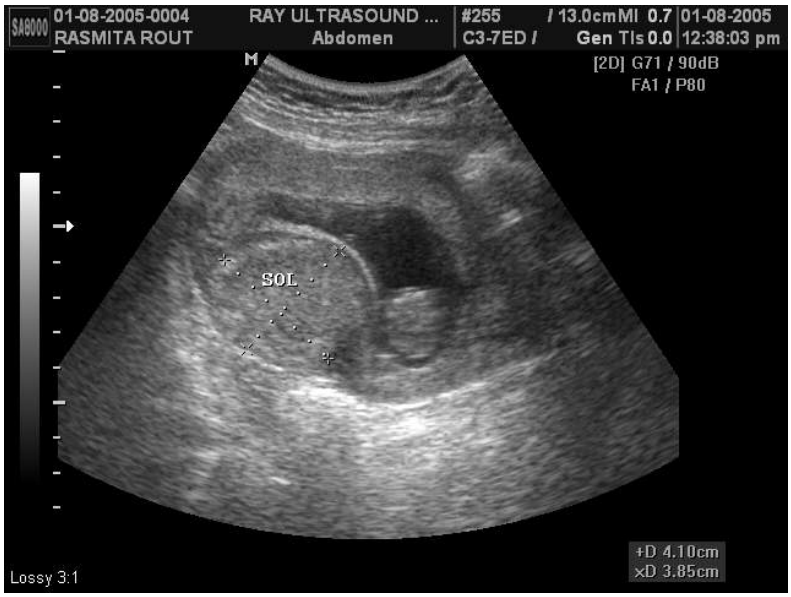

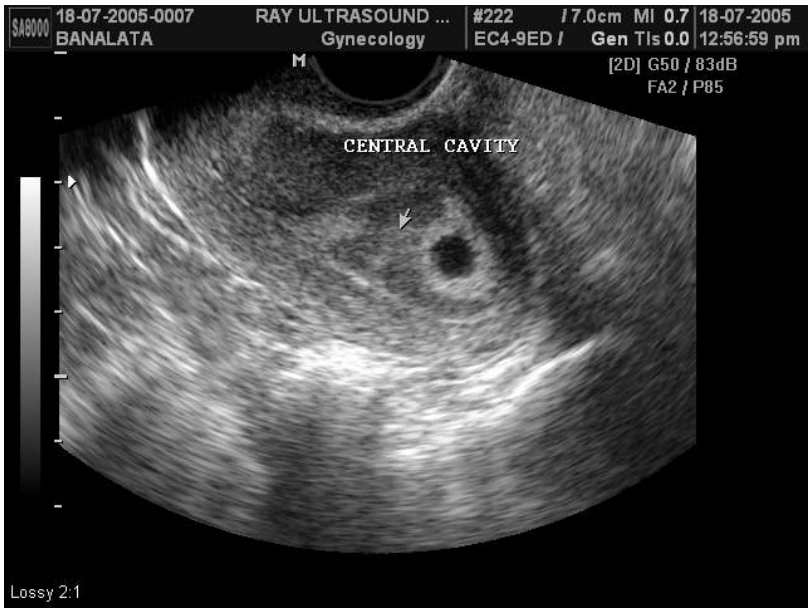
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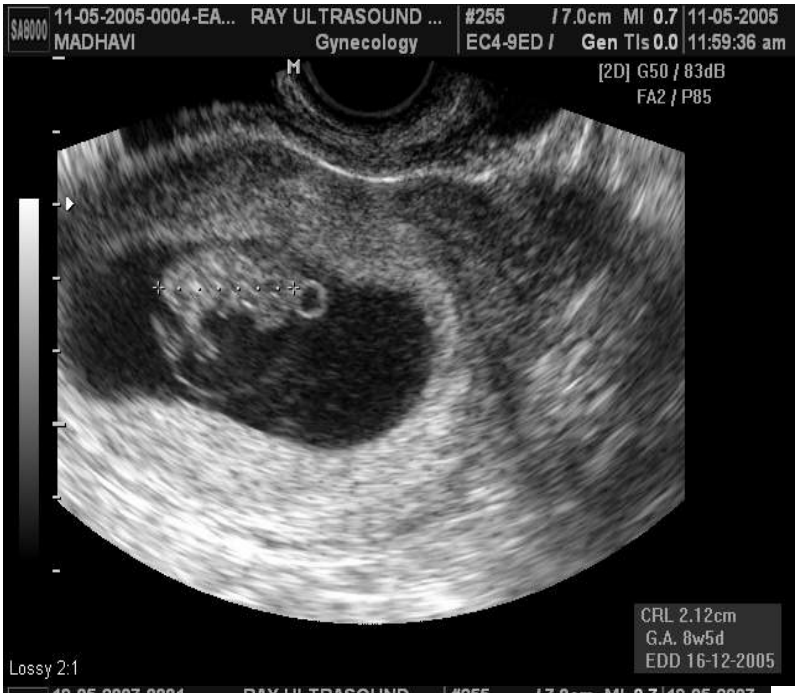
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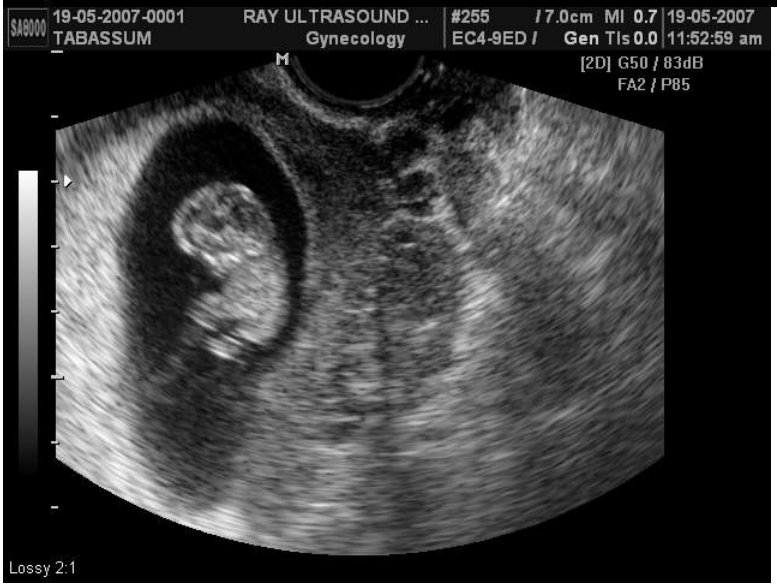
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11 -14 weeks Genetic Scan

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Introduction :

One of the primary goals of prenatal surveillance is to screen for & to detect congenital anomalies. Accurate & early prenatal diagnosis & timely termination of pregnancy reduces P.N.M. The optimal gestation to scan for most of developmental abnormalities : 18-22 wks. But it can be done at any stage of pregnancy. Sensitivity of USG to detect congenital abnormalities depends on expertise, gestational age, resolution of equipment. Sensitivity : 75-90% for CNS, 23-26% for cardiac .Variation in detection due to maternal obesity, abdominal scars, foetal position, oligohydramnios, foetal movements & foetal positions.

Whom to Scan

- Advanced maternal age
- Previous birth of a malformed fetus
- Family history of a malformed fetus
- Consanguinity
- Exposure to drugs/radiation
- Maternal Diabetes Mellitus
- Bad obstetric history
- Bleeding in early pregnancy

Sonographic findings in 1st Trimester requiring detailed abnormality scan

- **Oligoamniotic sac**
- **Embryonic bradycardia**
- **Abnormal yolk sac**
- **Increased nuchal translucency**
- **One identified abnormality**
- **Dates size discrepancy at 9-12 weeks**

Non sonographic findings requiring detailed abnormality scan

- **Abnormal results from a CVS/amniocentesis**

- **Abnormal immunoglobulin profile**
- **Abnormal triple test/increased alpha-fetoprotein /abnormal PAPP**

UNFORTUNATELY MOST FETAL ABNORMALITIES ARE DETECTED IN LOW RISK PREGNANCIES

Normal Sonoembryology

• Parameter	Seen first at
• G.S.	4.2-5 wks
• Y.S.	5-5.3 wks
• Embryonic pole	5.2 wks
• Cardiac activity	5.3 wks
• Limb buds	8.0 wks
• F.M.	8.0 wks
• Bowel herniation	9-11 wks
• Kidneys	10 wks
• Details of skull,spine	10 wks
• Stomach bubble	11 wks
• Cardiac config.	12 wks
• U. bladder	12 wks

All the fetuses which have an abnormality in their genetic configuration are bound to show some structural abnormalities and about last 12 -15 years the awareness for detecting such fetuses has increased.

If Sonographic markers for fetal anomalies are taken into consideration with other sonographic findings, 50 – 70 % of Trisomy 21, 80% of trisomy 18, 90 % of Trisomy 13 fetuses will show positive Sonogram.

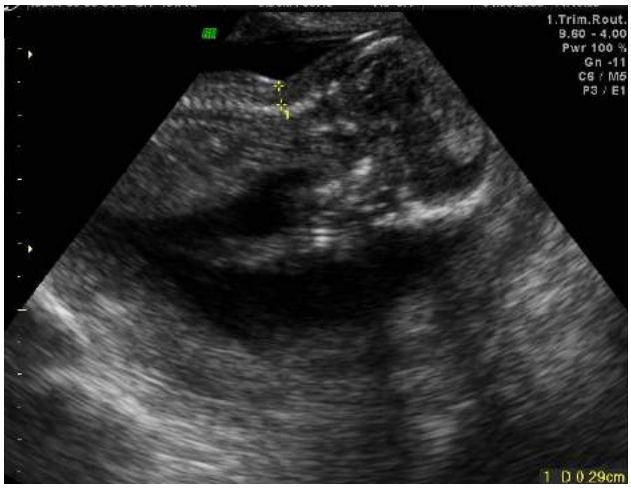
A targetted scan was therefore designed to pick up these abnormalities and was named as 'Genetic sonogram'.

Genetic scan

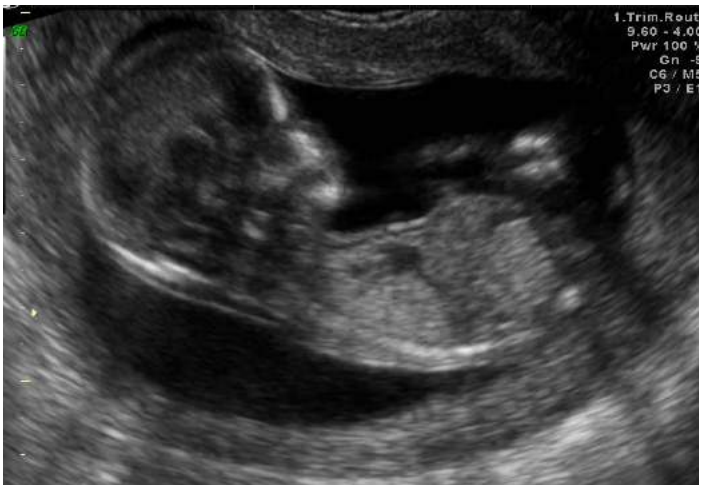
By definition, a genetic sonogram is defined as an ultrasound that can modify the *a priori* risk of fetal aneuploidy, typically based on a panel of ultrasound markers.

This scan is done for detection of

- **Chromosomal markers & major structural anomalies in aneuploidies.**



Most indicative markers are
A.Nuchal translucency
B.Ductus venosus flow
C.Nasal bone length



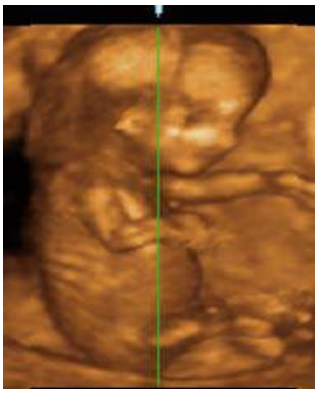
Nuchal Translucemy

Genetic scan 11-14 weeks

A. Nuchal Translucency :Translucency between the skin and soft tissues posterior to the cervical spine. Normal variations: 10 -12 weeks : 1.5 to 2.5 mm, 12 – 14 weeks : 2 – 3 mm , Nuchal translucency increases by 0.6mm on extension & reduces upto 0.4mm on flexion of the fetal neck. Cord round neck – false increase in NT by 0.8mm Measurements above and below the cord may be different Nuchal translucency increases by 17% every week.

Measuring Nuchal translucency

When CRL is between 36 and 80mm. (10.3 – 13.6wks). In true sagittal section. With maximum magnification – 75% of the screen should be covered. (caliper 0.1mm). Neutral position- neither extension, nor flexion of the head.Mean of three values. Interobserver and intraobserver error of < 0.5 %.



Causes of increased nuchal translucency

- Cardiac abnormalities & overload in Twin to Twin transfusion
- Venous congestion(amniotic membrane rupture diaphragmatic hernia)
- Delayed development of lymphatic drainage
- Neuromuscular defects leading to impaired lymphatic drainage.
- Congenital infections
- Anemia and hypoproteinemia
- Chromosomal anomalies –altered extracellular matrix

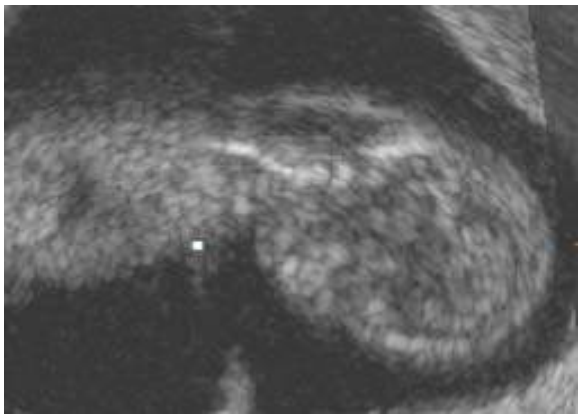
NT is increased in 75 - 80 % of fetuses with trisomy 21 between 11 – 14 weeks, but 5 - 10 % of healthy fetuses also show this sign. (also increased in Trisomy 13, Trisomy 18, triploidy, Turner's and Klinefelter's syndrome). Likely hood ratio for trisomy 21 with Increased NT is 13.

The fetal medicine foundation multicentre project report :

- Assessment of risk by combination of maternal age and nuchal translucency followed by invasive diagnostic testing for 5% of pregnancies with screen positive result and selective termination of affected fetuses, would reduce the livebirth incidence of trisomy 21 by 75%. Snijders RJM , et al.,Lancet 1998:352: 343 –346.

Sensitivity of nuchal translucency

- Nuchal translucency only picks up Downs' syndrome in 85% of high risk patients and in 75% of low risk patients false neg:6%
- Maternal age + NT + NB + Serum markers : 97% sensitivity with false negative 5%



The prevalence of the cardiac defects increased from <3% at nuchal translucency of < 3mm at 14 weeks, to 15% if it was >5.4mm.
Zosmer N., et al, Br. J. Obstet Gyneacol 1999;106: 829-833

Nuchal Translucency

Increased NT : look for chromosomal defects and multiple structural defects of heart and abdomen. Spontaneous resolution does not exclude chromosomal defects. Normal karyotype does not exclude structural abnormalities

Limitation

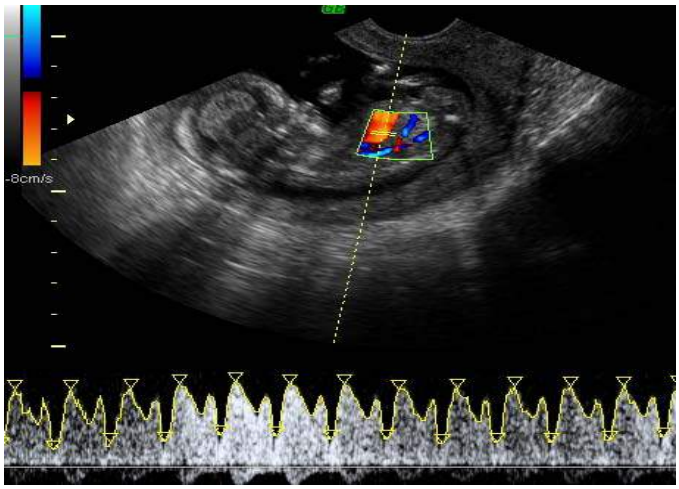
If normal nuchal translucency is considered as THE sign for a normal embryo, chances are there to miss 48% of abnormal babies, so detailed structural scan is always a must.

The most recent proposed protocol is that a Nuchal Translucency > 3mm is an indication for genetic counselling.

Anomaly scan 11 – 14 weeks

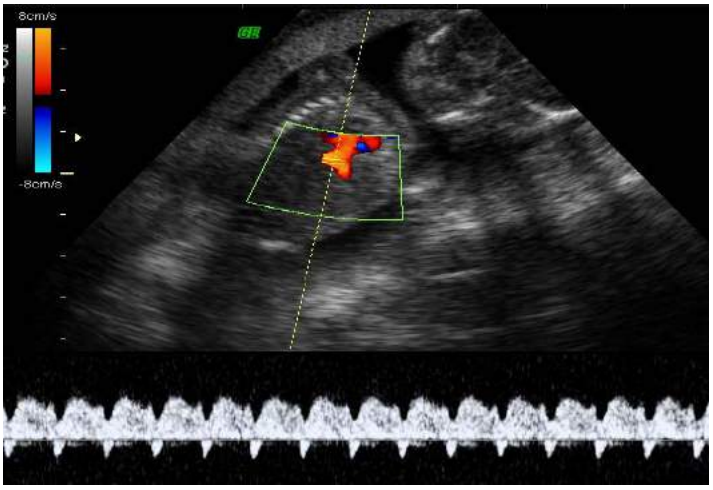
B. Ductus venosus flow: Origin from the portal sinus. Visualized in midsagittal longitudinal section or oblique transverse section through upper abdomen. Measured at the narrowest part (2mm) at the inlet in the umbilical sinus – 60° doppler angle. Common error is sampling hepatic vein for DV . Continuous forward triphasic flow. Ductus Venosus flow velocity is approximately 3 times higher than the flow velocity in the umbilical vein or inferior vena cava.

Normal



Ductus Venosus

Abnormal



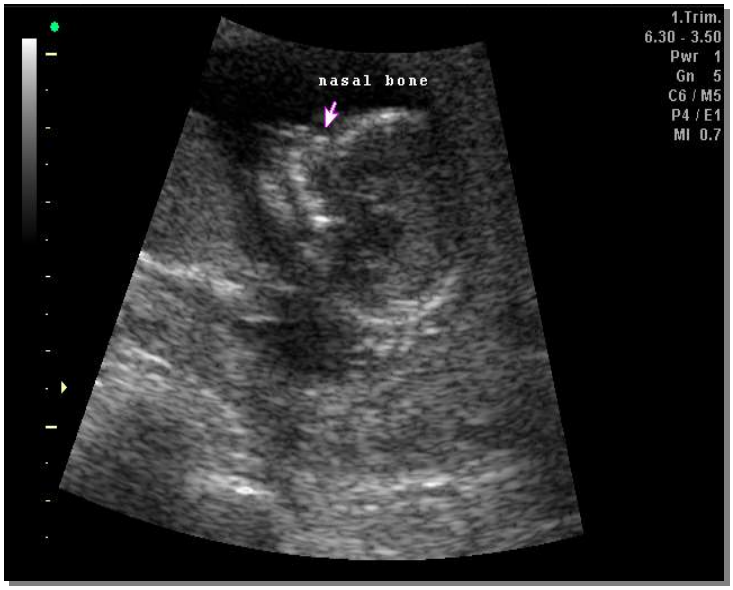
Ductus Venosus

Normal values – ductus venosus

Reversed diastolic flow – chromosomal or cardiac anomalies/ immature sphincter of DV in first trimester.Absent / inverted ACV has been seen in about 70 – 90 % with chromosomal anomalies. Abnormal flow in 40% fetuses with increased NT.

3. Nasal Bone

Intramembranous ossification first appears at CRL of 42 mm and increase linearly with gestation.Measured when CRL is 65 – 74 mm.11wks – 3mm doubles at 14 weeks. The bone is to be measured and not the skin – Tilt the probe for confirmation. Beam should be at 45°/ 135° angle to the line drawn from the front of the chin of the fetus and perpendicular to the nasal bone.



Nasal bone

Interpretation - Nasal bone

60 - 73% of Downs' syndrome fetuses have absent / hypoplastic nasal bone in 1st trimester. 0.5% of normal patients have absent nasal bone. With absent nasal bone the risk of Down's syndrome increases by 146 folds.Nasal bone length steady in Downs' syndrome from 11 –14 weeks.

Sensitivity of various parameters for Down's Syndrome

- Nuchal translucency – 80.4%
- Nasal Bone – 58.7%
- Ductus venosus flow – 93.5%
- All three combined – 94% with likelihood ratio for negative test only 0.08%.

Other markers

1.Cystic hygroma
2.Omphalocoele
3.Megacystitis
4.Growth delay
5.Heart rate variation
6.Umbilical cord abnormalities
7.↑ umbilical artery PI
8.Renal pyelectasis

9.CPC
10.Wide iliac angle

1. **Cystic Hygroma**

Nuchal cystic hygroma is a nuchal swelling, made of two symmetrical cavities, completely seperated by nuchal ligament with or without internal trabeculae. Simple nuchal oedema Cardiac defects or trisomies. Septated extensive usually Turner's Syndrome. Fetal karyotyping is must – 47 – 73% of cytogenic anomalies. In 1/3rd fetuses with nonimmune hydrops. Resolves in 37% of fetuses in 2nd trimester.May progress to lymphangiectasis – fatal .More chances of Turner's if in 2nd trimester and more likely to be fatal. Also seen with Trisomy 21, 18, 13 and Klinefelter's syndrome.

Milder cases of cystic hygroma

- Better prognosis
- Surgical correction after birth is possible
- Bony abnormalities of mandible, occipital bone and vertebrae may persist
- Facial palsy may persist

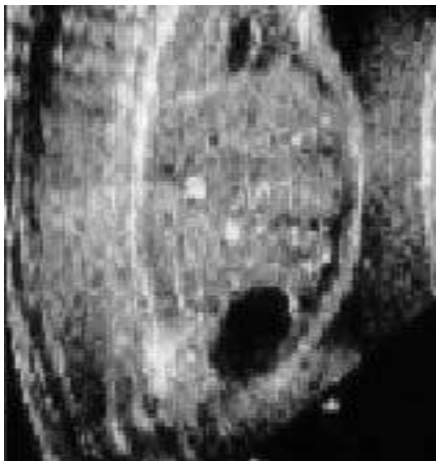
2.**Omphalocoele**

- Physiological from 8th to 12th weeks
- Abnormal if persists after 12th week –Trisomy 18
- Liver containing or bowel containing
- Even before 12th week, abnormal
- if has a neck of more than 7 mm
- Larger than or of the size of the abdomen
- Herniation of the liver – no chromosomal anomalies
- Bowel containing : Hyperechoic to the liver: 35 – 58% with aneuploidies
- Nonchromosomal syndromes like Turner's syndrome or uniparental disomies.
- 50% of bowel containing omphalocoeles are associated with cardiac anomalies.
- Prevalence of omphalocoele is 10 times higher in fetuses with increased nuchal translucency.

3. **Megacystitis**

- Bladder is visualized when CRL is 67mm.
- Normal emptying time cycle is 30 – 155 minutes.
- Megacystis – vertically measures > 6 mm or bladder diameter/CRL > 10%
- 8 -12 mm – mild to moderate
- > 17 mm severe
- Extent of megacystitis can be correlated to the risk of aneuploidy 25 % with moderate and 10 % with severe, at 10 -14 weeks.

- Risk is significant when due to obstructive uropathy and associated with other abnormalities.



Megacystitis

4. Growth delay

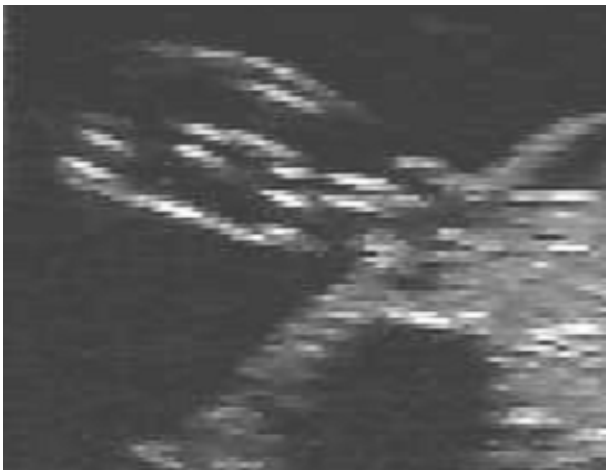
Difference in CRL of > 7mm of normal – three times higher risk of aneuploidy. Trisomy 18 – severe early onset growth restriction.Comparatively less in Trisomy 13 and 21.

5. Heart rate variation

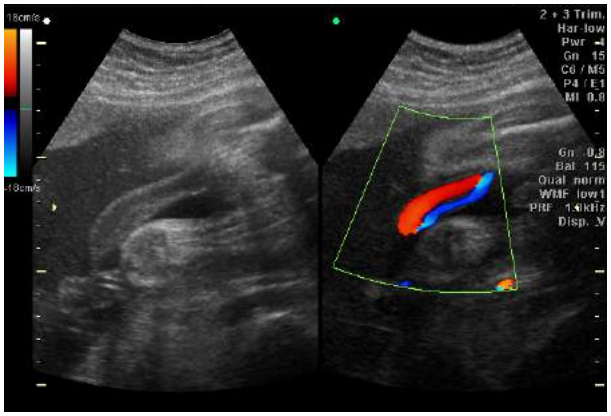
5th week – slow like peristalsis- 60-80/ mt. End of 5th week – 100/ mt. End of 6th week – 105 – 130 / mt
9 weeks – 160 – 170 / mt, then 120 – 160/mt. Tachycardia : Trisomy 21, 13, Monosomy X. Bradycardia : Trisomy 18 and triploidy

6. Umbilical cord abnormalities

Umbilical cord diameter above 95th percentile.Single umbilical artery seen in 1 % of pregnancies and of that 1 – 10 % have aneuploidies. Umbilical cord cyst- transient –not significant unless persist in 2nd / 3rd trimester.



Umbilical cord

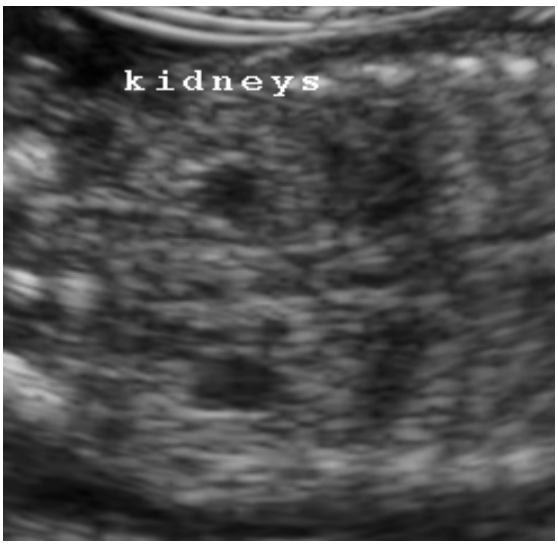


7. Umbilical Artery flow

High uterine artery PI or reversed end diastolic flow at 10 –14 weeks is associated with chromosomal anomalies.
Large prospective studies still needed to be proved.

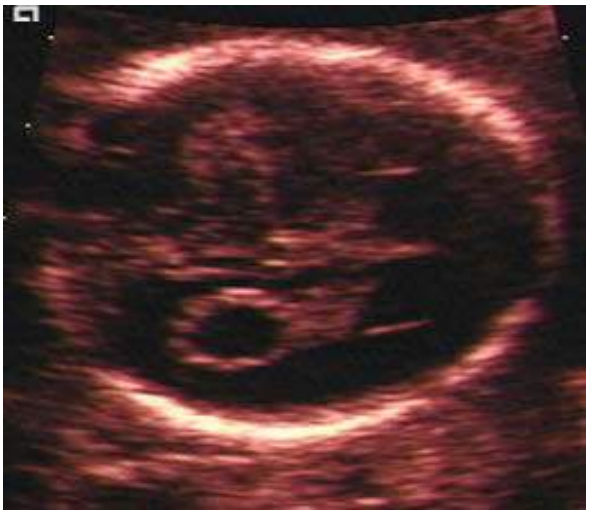
8. Renal pyelectasis

Renal pelvis of > 3mm at 14 – 15 weeks is pyelectasis. If bilateral, increases the risk for trisomy 21 by 1.5 – 2.0 folds.Is seen in 17 – 25 % of Trisomy 21, but 2.1 -2.8 % of normal fetuses.

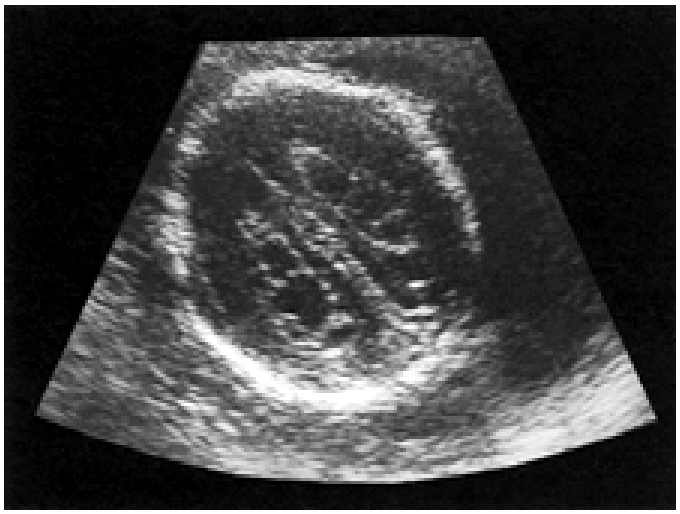


9.Choroid plexus cyst (CPC)

Choroid plexus is visualised at 8 weeks. Are transient and disappear at 20 – 23 weeks. Choriod plexus cysts
1% of normal and 50% of trisomy 18 fetuses have CPC. Usually 1 -2 mm in first trimester.Isolated and transient – nonsignificant.



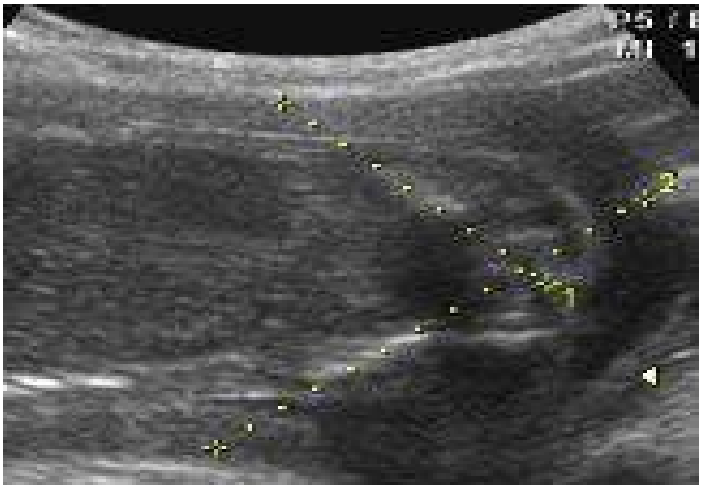
Choroids Plexus Cyst



Choroid Plexus Cyst

10. Fetal iliac angle

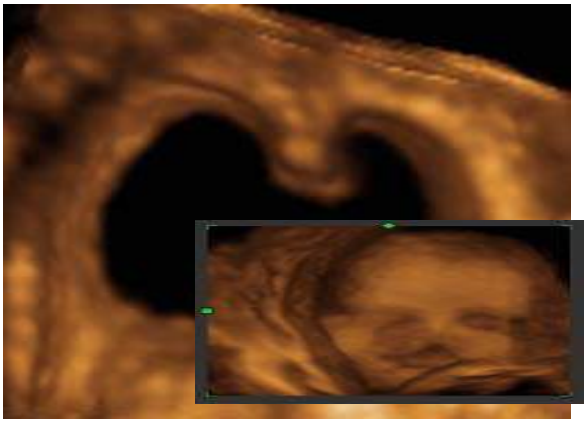
Used in second trimester. May be used in first trimester. Values need to be proved.

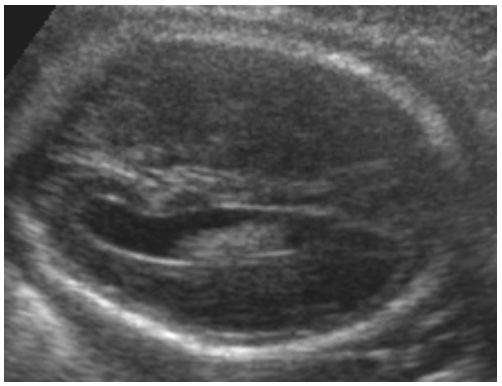


Foetal iliac angle

Structural abnormalities

Holoprosencephaly, Ventriculomegaly, Facial cleft, Micrognathia.





Risk of aneuploidy is higher when

Multiple markers , large gestational sac, associated cardiac anomaly or duodenal atresia, history of previous abnormal child ,high maternal age.

Limitations of USG

Not useful for single gene disorders like thallasemia or muscular dystrophy.

Conclusions :

A detailed, well performed anomaly scan can reduce the rate of invasive testing and less number of abnormal births.

TRANSVAGINAL ULTRASONOGRAPHY IN ECTOPIC EXTRAUTERINE PREGNANCY

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AGRA

The incidence of ectopic pregnancy has markedly increased in the last two decades and even in the USA, 6 to 11 percent of all maternal deaths are due to ectopic pregnancy (leading cause of death in the first trimester of pregnancy). The increasing incidence of ectopic pregnancy has been attributed to an increase in pelvic inflammatory disease, use of IUCDs, fallopian tube surgery and also our improved ability to diagnose an ectopic pregnancy.

Because of the variability of symptoms and morphologic presentations, ectopic pregnancy is still difficult to diagnose at times. In our country, usually the presentation is of classical ruptured ectopic (60%); another 25 percent cases present with chronic ectopic and only 15 per cent cases present to us before rupture. It is these 15 percent in which conservative surgical management and non-surgical management may have a role.

Transabdominal sonography (TAS) has proved disappointing in the diagnosis of ectopic pregnancy. Transvaginal sonography (TVS) cannot eliminate all problems but offers great advantage in positively diagnosing extrauterine gestation. The place of ultrasound in the overall approach to the patient with suspected ectopic pregnancy (EP) has undergone significant evolution, since, diagnosis of ectopic pregnancy was first described sonographically about 25 years ago. Initially, ultrasound was used to directly visualise ectopic masses. Later, recognition of the non-specificity of sonographic adnexal findings prompted the use of ultrasound to primarily exclude patients with intrauterine pregnancy. Today with transvaginal ultrasound we can not only identify the ectopic mass, but also characterise it for therapeutic planning.

Sonographic findings of ectopic pregnancy by transvaginal sonography are listed below:

A. Intrauterine Findings

1. Empty uterus with thickened endometrium.
2. Central hypoechoic area.
3. A sac like structure (pseudogestational sac).
4. Very rarely a concurrent intrauterine pregnancy.

Axioms

a. Best evidence against an ectopic is the presence of intrauterine pregnancy.

b. Absence of a detectable intrauterine pregnancy in pregnant women without symptoms significantly increases the likelihood of an ectopic.

c. Consider ectopic whenever intrauterine contents do not resemble a normal intrauterine pregnancy.

B. Extrauterine Findings

1. Adnexal masses
2. Living extrauterine embryo in an unruptured sac
3. Cul-de-sac findings.

C. Other Ectopic Pregnancies

1. Heterotropic pregnancy (co-existing intra and extrauterine)
2. Interstitial pregnancy.
3. Rudimentary horn pregnancy.
4. Ovarian pregnancy.
5. Cervical pregnancy.
6. Abdominal (peritoneal pregnancy).

OVERVIEW OF SONOGRAPHIC SIGNS

The sonographic work up of possible ectopic pregnancy includes both negative ad positive signs.

1. Negative signs

Intrauterine pregnancy

i. Living intrauterine embryo
ii. Gestational sac with yolk sac
iii. Double decidual sac sign.

2. False negative signs

Intrauterine pseudogestational sac.

a. Empty uterus sign
b. Free pelvic and abdominal fluid.

4. Direct positive signs

a. Adnexal pregnancy
   i. Living embryo
   ii. `Tubal or adnexal ring
   iii. Complex or solid mass.
b. Abdominal pregnancy.

1. Negative Sonographic Signs

Intrauterine Pregnancy

When considering an ectopic pregnancy, the first branch in the sonographic decision tree is whether or not there is evidence of an intrauterine pregnancy essentially ruling out the possibility of an ectopic pregnancy of one in 30,000 pregnancies. However, based on the currently increased incidence of ectopic pregnancy in general, heterotropic pregnancy is now estimated to occur as frequently as 1:10,000 pregnancies.

One strategy to deal with this problem is not to confirm intrauterine pregnancy until an embryo with cardiac activity is demonstrated, although this can delay diagnosis for 1 to 2 weeks. Actually, embryonic development can be recognised even earlier, before demonstration of the embryo itself, through demonstration of a yolk sac. The yolk sac is consistently demonstrable in normal intrauterine pregnancy by transabdominal sonography in a gestational sac of 2 cm in diameter and by transvaginal sonography in a gestational sac of 0.6 to 0.8 cm.

2. False Negative Sonographic Signs

Intrauterine Pseudogestational Sacs

Pseudogestational sacs are intrauterine fluid sacs surrounded by decidual reaction that simulate the sonographic appearance of true gestational sacs and are found in 8 to 29 per cent of patients with ectopic pregnancies. Identification of the double decidual sac sign or of embryonic development often is helpful in differentiating pseudogestational sacs from normal intrauterine gestational sacs, but may prove

problematic in distinguishing pseudogestational sacs from the sacs of failed or failing intrauterine pregnancy. Colour Doppler sonography has provided us with new diagnostic information to differentiate pseudogestational sacs from gestational sacs of intrauterine pregnancy, both normal and failing.

3. Indirect Positive Signs

a. Empty uterus Sign and the Discriminatory Zone

Demonstration of an empty uterus in a patient known to be pregnant, is one of the oldest signs of ectopic pregnancy. After introduction of the highly sensitive radioimmunoassay HCG test, hormonal pregnancy testing became positive two weeks prior to sonographic demonstration of the intrauterine gestational sac. Application of the empty uterus sign in conjunction with a positive a radioimmunoassay pregnancy test, therefore, risked misdiagnosing normal intrauterine pregnancy as ectopic pregnancy in the two weeks prior to visualisation of the normal sac.

Non-visualisation of an intrauterine sac, together with a serum HCG above the discriminatory zone, indicated an abnormal pregnancy, either a spontaneous abortion or an ectopic pregnancy. As sonographic resolution has improved the discriminatory zone has continued to fall, now at 10,000 mi,u./ml (IRP) with transvaginal sonography.

b. Free Pelvic and Abdominal Fluid

Pelvic fluid may fulfil two functions in sonographic diagnosis; as a primary, though indirect, sonographic sign of ectopic pregnancy, and as a supporting sign, increasing the specificity of the other adnexal findings. Pelvic fluid functions as a relatively specific sign of ectopic pregnancy only when it is found in large volume or, when it is echogenic. As a primary sign, echogenic fluid (representing haemoperitoneum) indicates ectopic pregnancy with a specificity of 96 per cent and with a sensitivity of 25 to 59 percent.

4. Direct Positive Signs

a. Adnexal pregnancy

i.Live extrauterine embryo. Demonstration of a live adnexal embryo is the most specific sonographic sign. The sensitivity of this sign, however, has increased over the years, probably due to earlier presentation and diagnosis and due to the superior resolution of transvaginal sonography.

ii. Tubal or adnexal ring. The sac, termed an adnexal or tubal ring because of its location has a characteristic appearance with no live embryo within. As in the case of the intrauterine gestational sac the tubal ring consists of an anechoeic central fluid sac and an echogenic thick rim. Tubal rings without live embryo (with or without yolk sacs) are seen in 28 to 44 percent of ectopic pregnancies via transvaginal scanning. The potential exists, however, for similar sonographic appearances to cause tubal rings and corpus luteum cysts to be confused with the another.

iii. Complex or solid masses. Complex or solid adnexal masses are found with transvaginal sonography in 22 to 45 per cent of cases of ectopic pregnancies. It is imperative to pay close attention to the ovaries during the evaluation of all adnexal masses for possible ectopic pregnancy. failure to demonstrate a distinct ipsilateral ovary in addition to the visualised mass may indicate that the mass is ovarian in origin.

b. Abdominal Pregnancy

Sonographic diagnosis of abdominal pregnancy presents a challenge. The diagnosis may be missed 25 percent of the time or more, and often is delayed until late in pregnancy.

COLOUR AND PULSED DOPPLER SONOGRAPHY FOR ECTOPIC PREGNANCY

Color and pulsed Doppler ultrasound adds physiologic information to the sonographic work up of possible ectopic pregnancy, thereby, increasing the sonographic diagnostic sensitivity for ectopic pregnancy, normal early intrauterine pregnancy, and failed early intrauterine pregnancy. Diagnostic sensitivity increases from 71 to 87 per cent for ectopic pregnancy, from 24 to 59 per cent for ectopic pregnancy,

from 24 to 59 per cent for failed intrauterine pregnancy, and from 62 to 82 percent for all diagnoses when transvaginal sonography was augmented with color Doppler sonography. The combined use of colour and pulsed Doppler and two dimensional imaging solves the following sonographic problems in the ectopic pregnancy work up.

1. Differentiation of pseudogestational and gestational sac.
2. Differentiation of the empty uterus sign into either intrauterine pregnancy (Normal and abnormal) or no intrauterine pregnancy (with a greatly increased chance of ectopic pregnancy).
3. Identification of the site of the corpus luteum.
4. Assistance in detection and confirmation of the nature of adnexal ectopic pregnancies.

ULTRASOUND GUIDED CONSERVATIVE MANAGEMENT OF ECTOPIC PREGNANCY

Conservative therapy for ectopic can be :

i. Expectant
ii. Systemic methotrexate
iii. Local injection of methotrexate

i. Expectant

It (i.e. no methotrexate, no surgery) the following criteria to be met for this line of management.

1. Decreasing HCG levels
2. Minimal symptoms.
3. Adnexal mass □ 5 cm.
4. Colour Doppler evaluation for trophoblastic flow. Spontaneous resolution is common especially when close watch is kept on pregnancies with ART procedures and colour flow studies are carried out.

ii. Local Injection of Methotrexate

It can be tried in very early detected ectopics and avoids all the systemic side effects of methotrexate and it acts on the trophoblastic growth which is seen to gradually regress and can be monitored by color Doppler flow studies and serially decreasing βHCG levels. If a live embryo is seen, KCl is first-injected in the fetal heart.

iii. Systemic Methotrexate

Systemic methotrexate has also been effectively used especially if proficiency in interventional ultrasound is lacking and the criteria used are:

1. Adnexal mass □ 3.5 cm. in greatest diameter.
2. Serum HCG < 1000 IU/L.
3. No sign of live ectopic pregnancy.
4. Little or no free intra peritoneal fluid.
5. Vital signs are stable.

Single dose of methotrexate intra muscular gives a success rate of 94.1 percent (Gross et al).

CONCLUSION

Ultrasound, specially TVS is today the hall mark of diagnosis and early management of ectopic pregnancy. Color flow improves the diagnostic capability.

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USG in Gestational Trophoblastic Neoplasia

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GESTATIONAL TROPHOBLASTIC DISEASE (GTD) / NEOPLASIA

Is a manifestation of an aberrant fertilization that leads to unrestrained, proliferation of trophoblastic tissue which may sometime result in an invasive neoplasm.

Clinical classification of trophoblastic diseases[1]

I. Hydatidiform mole
   A. Total hydatidiform mole
      (1) non-invasive total mole
      (2) invasive total mole
   B. Partial hydatidiform mole
      (1) non-invasive partial mole
      (2) invasive partial mole
II. Choriocarcinoma
III. Persistent trophoblastic disease

Every disease from I to III is further divided into metastatic or non-metastatic disease.

NCI / NIH clinical classification[1]

1. Benign GTD
   A. Complete hydatidiform mole
   B. Partial hydatidiform mole
2. Malignant GTD

A. Non-metastatic GTD

B. Metastatic GTD

1. Good prognosis, low risk - absence of any risk factor
2. Poor prognosis, high risk - presence of any risk factor

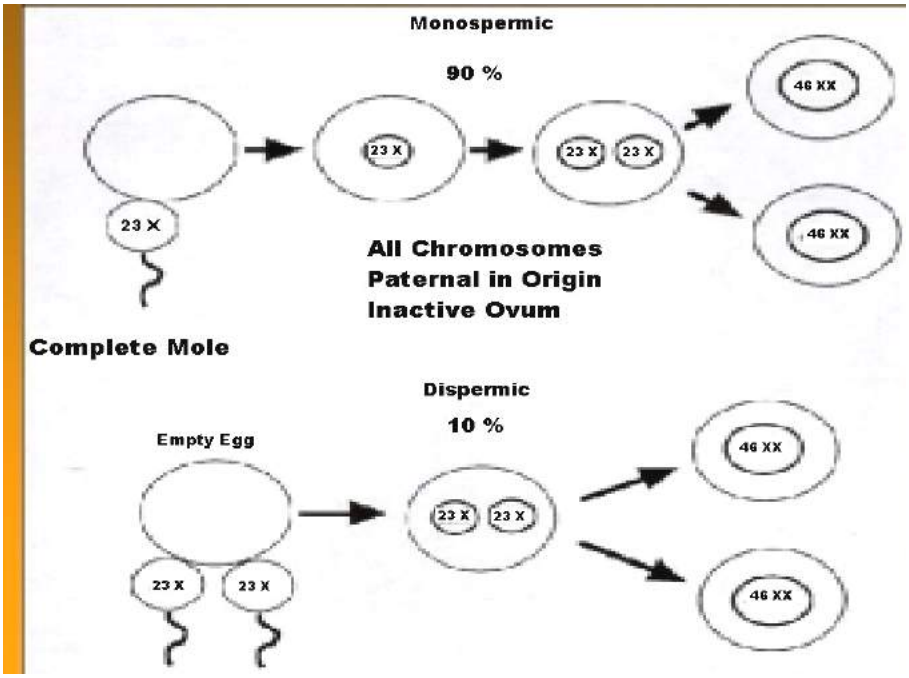
3. Risk factors are: -
   a. Duration of disease greater than 4 months.
   b. Pre-therapy βhCG level greater than 40,000 IU/ml (prior to any chemotherapy).
   c. Brain or liver metastasis
   d. GTD after term gestation
   e. Prior failed chemotherapy

COMPLETE MOLE

It is an abnormal pregnancy, where all placental villi change to molar vesicles and fill the uterine cavity but there is no embryo, fetus, or umbilical cord. Amnion however, is, found in some cases.[2] No capillary vessel is noted in the molar cyst which is covered by proliferated trophoblasts.

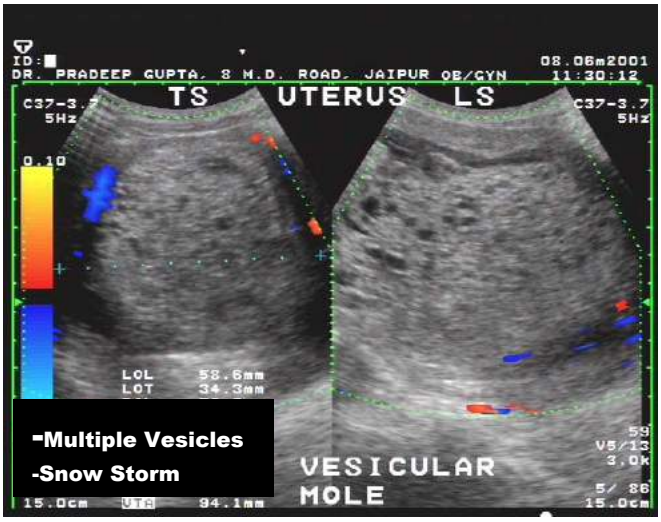
Genetic Composition

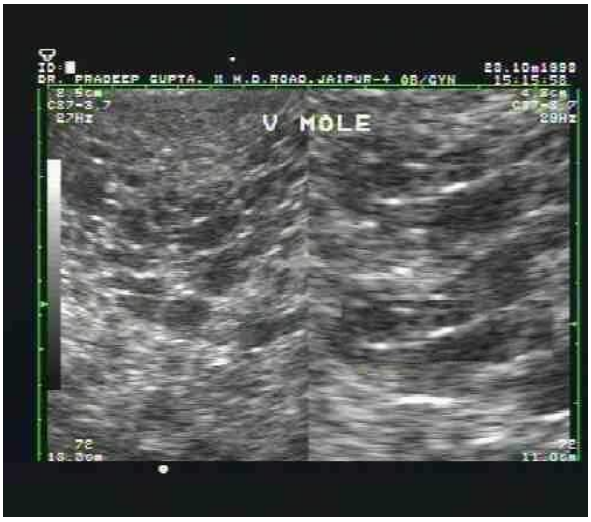
Chromosomes are usually diploid 46, XX where the XX are both of male origin, that is called androgenesis.[3] It is due to fertilization of an empty ovum by 1 or 2 sperms, maternal chromosomes are absent.[3] The chromosome is rarely 46, XY, where both X and Y are male origin.[4]



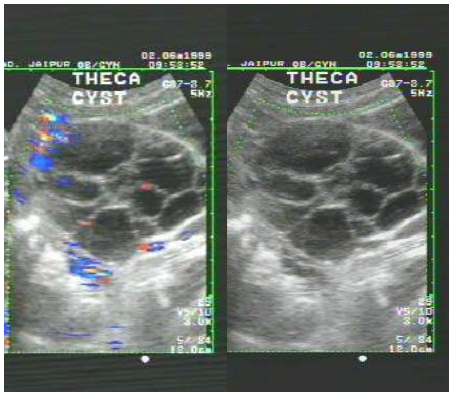
USG Findings

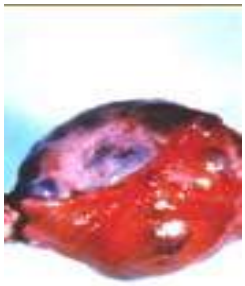
The uterus is enlarged with absent fetal echoes. Echogenic material with scattered small cysts fills the uterine cavity (Hydropic Degeneration). There is an extensive A-V communication so it appears highly vascular with high velocity and low impedance blood flow. The diastolic flow is large and RI is lower in uterine, arcuate, radial and spiral arteries as compared to that in normal pregnancy.[5] Also in the intervillous space, RI is low in molar pregnancy.[6]





Total mole is diagnosed when urinary or serum hCG is higher than 100,000 mIU/ml that (which) is the higher normal range of early pregnancy. The elevated hCG levels cause ovarian stimulation resulting in formation of bilateral theca lutein cysts (In 20-50% pts). Complications of cysts e.g. torsion, rupture, bleeding and ascitis may sometimes be seen.[7] On Doppler flow impedance is as high as 0.6 in the wall artery of theca lutein cyst.[8]



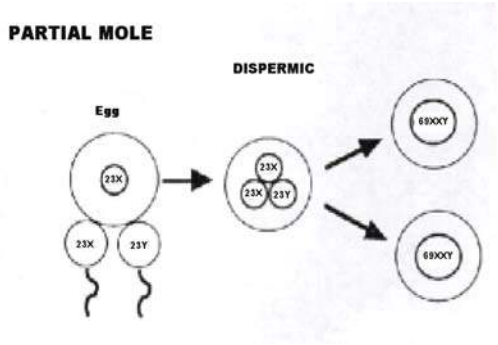


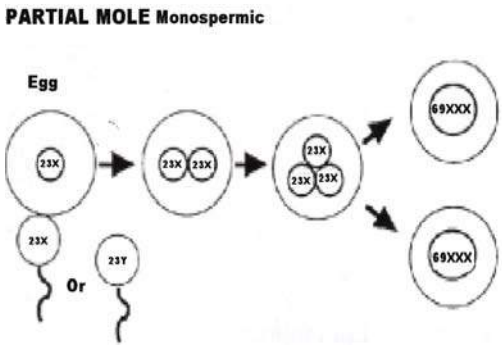
PARTIAL MOLE

In this condition partial placental villi undergo molar changes and alongwith embryo, fetus or fetal parts are also seen. Fetal anomalies are common.

Genetic Composition

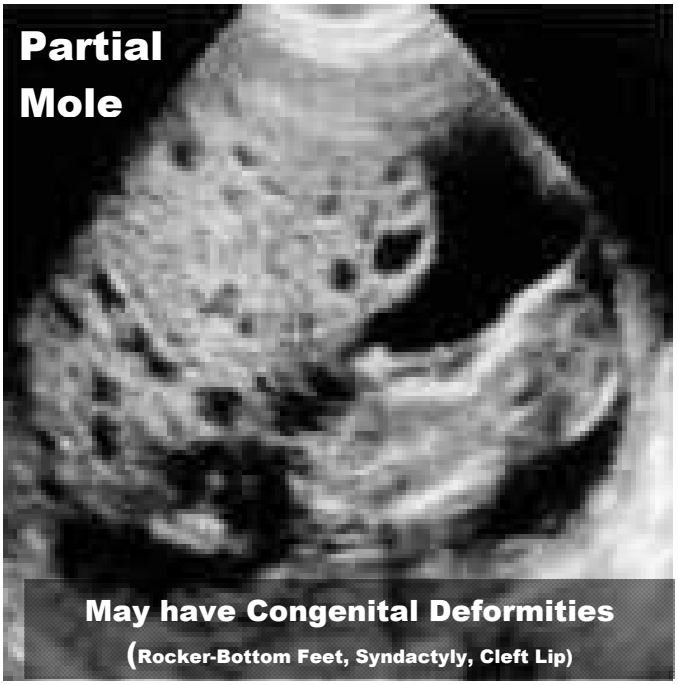
Partial mole is due to fertilization of an apparently normal ovum by two sperms, full component of maternal chromosomes are also present. Chromosomes are usually triploids, 69, XXX, 69, XXY, or 69, XYY.[9]





USG Findings

There is presence of the fetus, or the partial image of the fetus and partial changes of the placental villi into molar cysts. Anomalies are frequent in the fetus.



MANAGEMENT OF V-MOLE

Involves suction evacuation followed by post-molar surveillance. Beta-hCG urine/serum should be normal in 6 wks. If it does not come down or plateaus, it results in persistent trophoblastic disease.

PERSISTENT TROPHOBLASTIC DISEASE

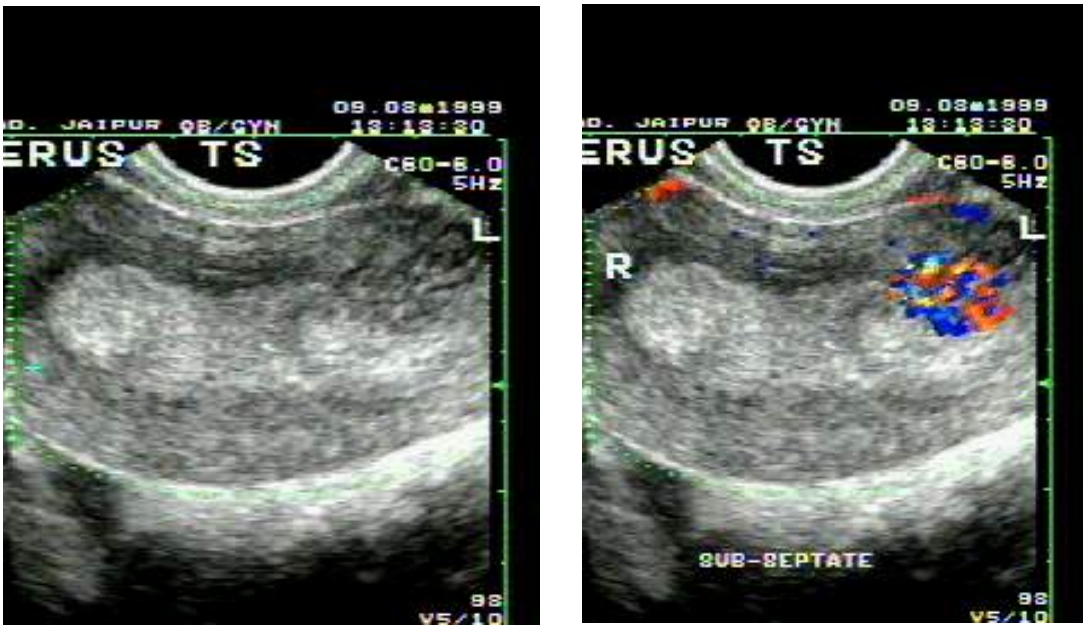
PTD includes three categories, i.e. postmolar persistence of hCG, clinical invasive or metastatic mole, and clinical choriocarcinoma.

USG helps in differentiating

- Fresh Pregnancy
- Residual Molar Tissue
- Invasive Mole
- Choriocarcinoma
- PSTT

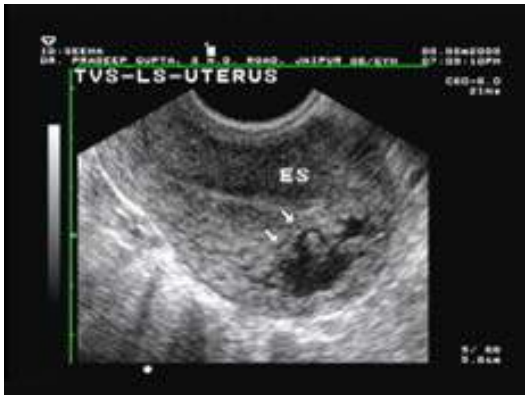
INVASIVE MOLE

It is the invasion of molar cyst into the myometrium with destruction and haemorrhage. It is mainly found after evacuation of total or partial mole, with short interval after the mole, which is less than half year, although the mole invades the myometrium during its pregnancy. It presents as persistent bleeding, persistently elevated serum beta-hCG levels and on ultrasound, central uterine echoes are seen as in complete vesicular mole, occasionally with demonstrable myometrial invasion. Rich blood flow is found on color Doppler and flow impedance is low.

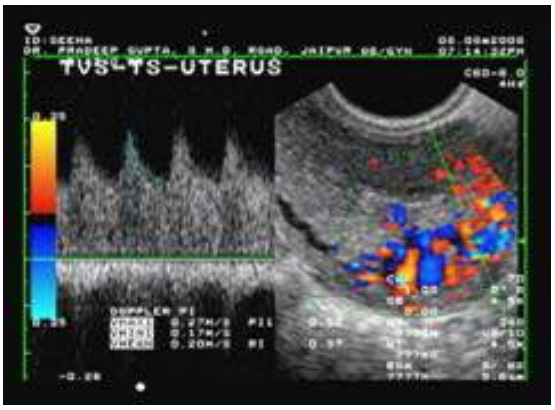


Here evacuation for vesicular mole was already done but the patient had persistent bleeding. Nothing was seen on follow up by TAS on two occasions. On TVS we saw a

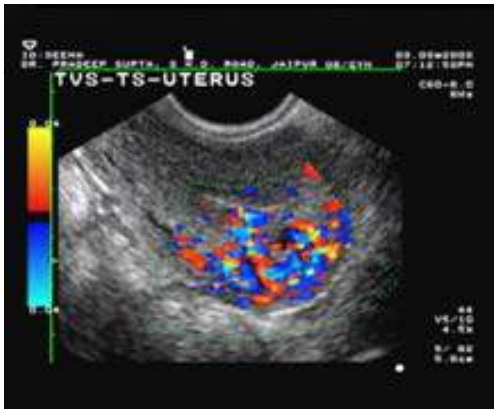
subseptate uterus and a hypoechoic irregularity extending from the endometrium into the myometrium was seen on the left side which was confirmed by Doppler.



After D&E twice for incomplete abortion cause of persistent bleeding - Diagnosed as above mymetrial echo poor lesion seen in posterior wall of uterus



Same patient on CDS shows high vascular myometrial lesion, endometrium is normal. βhCG after one month of evacuation was 22,000 IU/ml



Intra lesion vesicles show low resistance RI 0.39

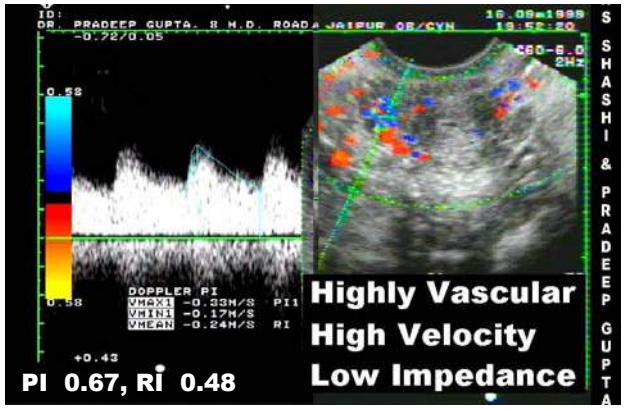
CHORIOCARCINOMA

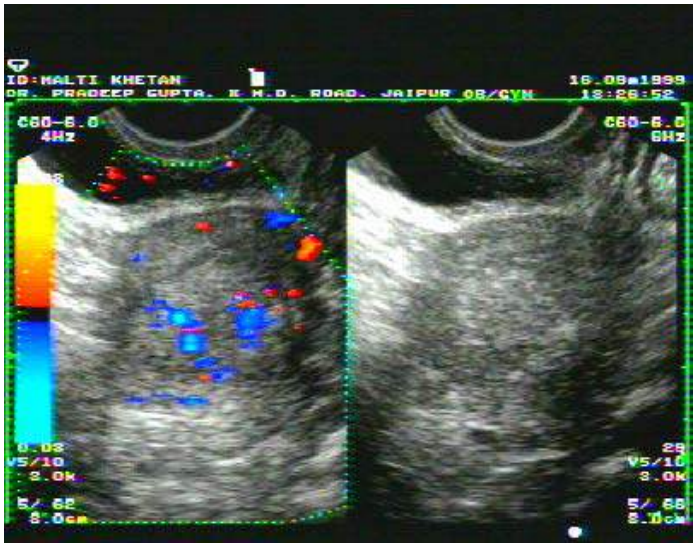
It occurs in 1:20,000 to 1:40,000 pregnancies, 50% after term pregnancy, 25% after molar pregnancy and 25% after other gestations e.g. ectopic.[10] Clinical symptoms are similar to the invasive mole and it is in early stage before the metastasis but invasive mole is characterised by its post molar detection, shorter interval after the mole and relatively lower titres of βhCG.

Choriocarcinoma is constructed of syncytio and cyto-trophoblasts, and shows no villus pattern at all.

USG in Choriocarcinoma

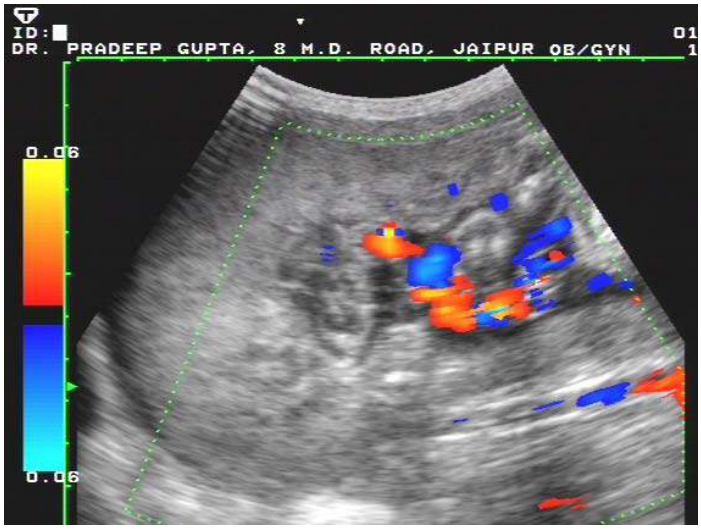
USG has limited role in detection of primary lesion but is useful for looking up for metastasis. However the primary lesion may show a mass enlarging the uterus. Sometimes only a discrete, central, infiltrative lesion is seen with heterogeneous appearance due to necrosis and hemorrhage. Differential diagnosis of choriocarcinoma from invasive mole is important, because the outcome is ominous in the former and less risky in the latter, in spite of the similarity of their clinical symptoms. Ultrasonic detection of cystic pattern in the focus is decisive evidence to invasive mole, i.e. ultrasound is an alternative to X-ray pelvic angiography. Ultrasonic B-mode image shows no cyst pattern in choriocarcinoma. Color Doppler flow mapping shows rich blood flow, and flow impedance is usually low in both diseases, but it is lower in choriocarcinoma than invasive mole. The RI of uterine artery is significantly lower in the choriocarcinoma than in hydatidiform mole.[11]

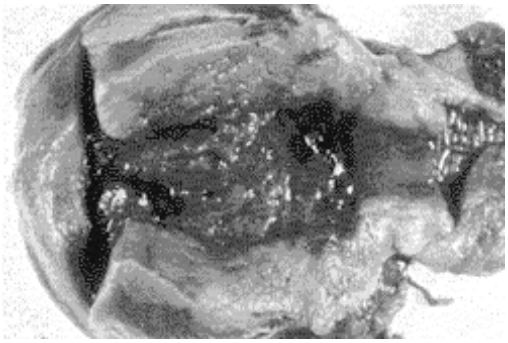




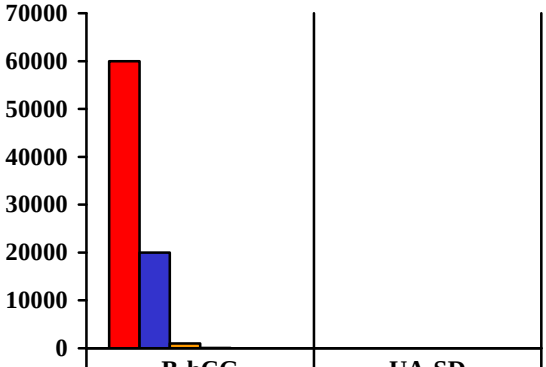
This 40 yr old Lady came to us with Menorrhagia 3 mts after an MTP. TAS did not reveal anything abnormal but on TVS showed slightly thick irregular endometrium. Careful scanning showed some echogenicity in the myometrium as well. Switching on the Colour showed vascular nodules in the Myometrium.

POST DELIVERY CHORIOCARCINOMA





This lady had a normal delivery 5 mts back. She then started having gradually increasing bleeding PV. She was sent with a clinical suspicion of Fibroid, TVS revealed a bulky uterus and Doppler showed a highly vascular sinusoidal mass in the uterine cavity. As a shot in the dark a BhCG was requested which was significantly high suggesting Choriocarcinoma. Hysterectomy was done and this is what was seen on cut section.



	B-hCG	UA-SD
Immediate	60000	2.5
1st Week	20000	2.7
2nd Week	1000	3.8
3rd Week	100	4.7
4th Week	25-50	5.0 AEDV
5th Week	< 25	AEDV

Kurjak et al 1992, Rajan et al 1995

Uterine Art. SDR Co-relates very well and is Inversely proportional to Falling B-hCG.
Helpful- Post-Molar Surveillance

CONCLUSION

USG is very helpful in gestational trophoblastic disease for early diagnosis, management, surveillance and assess chemotherapy response.

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PLACENTA AND THE UMBLICAL CORD

BY

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Evaluation of the location, size, shape and echo texture of the placenta as well as the retro placental area should be a part of every antenatal ultrasound examination. A thorough understanding of the normal maternal anatomy as well as the pathologic conditions is necessary to interpret the sonographic appearance which is very essential to understand the normal and abnormal sonographic findings. Some normal variations should also be understood in order to avoid over interpretation of sonographic findings.

NORMAL ANATOMY AND POSITION

Maternal surface

Termed basal plate , Lie congruous with the deciduas basalis.
It is Irregular in appearance.

Fetal Surface

Termed chorionic plate.
It is Smooth in appearance.
Covered by amniotic membrane.

Placental Circulation

Intervillous spaces located within placental lobules.
Oxygenated maternal blood enters the intervillous spaces via spiral arteries.
From intervillous spaces, blood flows around and over surface of villi. This process permits exchange of oxygen and nutrients with fetal blood flowing in villous capillaries .

Placental Size

Placental thickness usually does not exceed 5cm A-P dimension.

Placental Position

Placenta position in uterus can be categorized as:

1. posterior
2. anterior
3. RT lateral or LT lateral
4. fundal
5. combination

Placental Migration

It is the impression of placental ascension from the cervical os during the last trimester of pregnancy, due to differential growth of lower uterine segment.

Placental Number

Singleton

-

Dizygotic twin – 2 placentas (may be fused)

-

Monozygotic twin

1. Dichoriotic / Diamniotic – 2 placentas (may be fused)

2. Monochoriotic / Diamniotic – 1 placenta

3. Monochoriotic / Monoamniotic – 1 placenta

SONOGRAPHIC APPEARANCE OF PLACENTA

1. In 8 weeks, the early placenta is visible sonographically as a generalized thickening around the gestational sac.

2. 10-12 weeks, diffuse granular texture of the placenta is clearly apparent sonographically.

3. Chorionic plate is usually seen as an echogenic line in the fetal surface of placenta. Basal plate cannot be identified sonographically unless it becomes calcified near term.

4. The incidence of placental calcification increases exponentially with increasing gestational age, beginning at about 29 weeks. More than 50% of the placentas show some degrees of calcification after 33 weeks.

Calcification

The sonographic texture of the placenta remains unchanged throughout pregnancy, except for the deposition of calcium which is a physiological phenomenon that occurs throughout pregnancy. Calcium is microscopic during the first two trimesters but may be macroscopic after 33 weeks. Placental calcification has no proven clinical or pathologic significance. Post mature placentas do not show increased calcification. Since there is no proof that placental calcification has any significance, grading of degree of calcification seen at sonography is not worthwhile. Still grading is being mentioned for theoretical purposes:

PLACENTAL GRADING

NORMAL PLACENTAL GRADES

Grade 0

- Smooth chorionic plate, homogenous echotexture of substance of placenta. Most common in 1st trimester and early 2nd trimester (8-20weeks).

Grade I

- Small intraplacental calcification randomly dispersed within the substance of a placenta. May appear as early as 14 weeks, and is most common until 34 weeks.

Grade II

- Calcification of basal plate. Does not usually appear until after 30 weeks.

Grade III

- Calcified indentation of placenta extending from the basal plate to the chorionic plate (placental cotyledons). Usually not seen until 35 weeks. Found in 30% of term placentas.

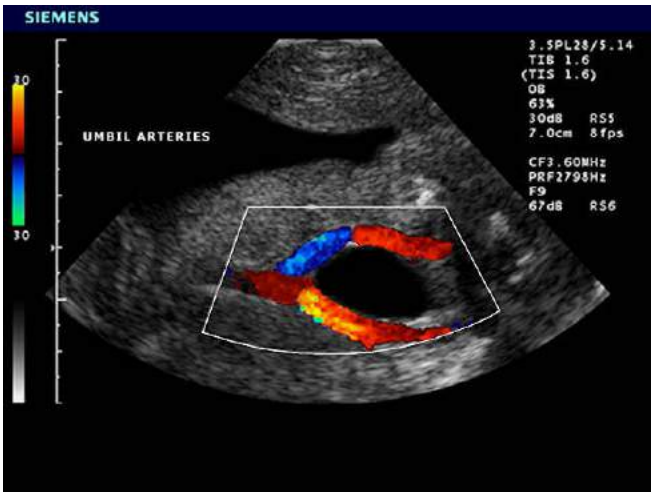
Umbilical cord

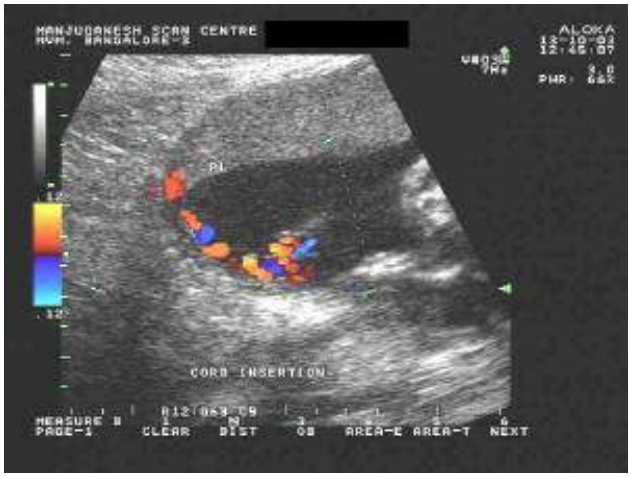
The normal umbilical cord has three blood vessels two arteries and one vein, which are surrounded by Wharton's jelly.
On sonography, it is diagnosed by demonstrating a complete parallel course of umbilical artery and vein in longitudinal section, and by demonstrating only two circular lucencies in the transverse section.

Documentation of the number of vessels in the umbilical cord is very important to be included in every obstetrical ultrasound. While the incidence of single umbilical artery is 1% or less, approximately 10% to 20% of these cases will have one or more additional malformations , including trisomy 18 , trisomy 13 , urinary tract abnormalities , CNS abnormalities , cardiac anomalies , omphalocele , sirenomelia and the VATER association. Umbilical cord usually measures 1 to 2 cm in diameter. Enlargement may be caused by hematoma or edema or it may be a normal variant .
Marginal insertion (battledore placenta) occurs in less than 6 % of the placentas and is likely to be of no clinical significance. In upto 1.6 % of deliveries the cord inserts on the membranes at a variable distance from the placenta (velamentous insertion) . In this situation , the unprotected intramemberanous umbilical vessels may bleed in the antepartum period and are susceptible to damage during labour and delivery . Vessels traversing the internal os of cervix (vasa previa) may also rupture during delivery. Colour flow Doppler is useful in antenatal detection of vasa previa.

Umbilical Cord Masses

- False knots
- True knots
- Haematoma
- Allantoic duct cyst
- Neoplasm
- Umbilical hernia
- Omphalocele and gastroschisis





Size of placenta

Placenta is a fetal organ and hence its size is proportionate to the size of the fetus.

Very large placentas are associated with various conditions , including blood group incompatibilities , maternal diabetes , severe maternal anaemia , fetal hydrops , and homozygous alpha thalassemia . Large placentas with hydatidiform change are found in cases of triploidy. Small placentas are seen in disorders characterized by underperfusion such as maternal hypertension, toxemia & severe diabetes as well as in some cases of multiple congenital anomalies & chromosomal abnormalities.

Variations in shape

Succenturiate lobes:

They occur in 3% to 8% of the placentas. Fetal blood vessels coursing through the membranes connect these lobes to the main placenta. Succenturiate lobes are visible at sonography. It is because of the complications that can result, including retention of the accessory lobe following delivery, implantation of the accessory lobe over the cervical os, & bleeding from the vessels connecting the succenturiate lobe to the main placental mass it is necessary to diagnose this in antenatal period.

Extrachorial placenta:

They are best visualized prior to 20 weeks. There are two types of extrachorial placentae: circummarginate & circumvallate; either of these forms may be partial or complete. Circummarginate placentas have no clinical significance.

Others:]

Annular (ring shaped) placentas & placenta membranacea have been associated with antepartum & postpartum hemorrhage. Placenta membranacea is a rare condition in which the entire face of the amniotic sac is covered with villi due to failure of regression during early pregnancy.

Intraplacental lesions

These include:

Subchorionic fibrin deposition

These are plaques of fibrin which are found in 20% of uncomplicated pregnancies.

Perivillous fibrin deposition

Occur to some degree in all placentas . not much clinical significance.

Intervillous thrombosis

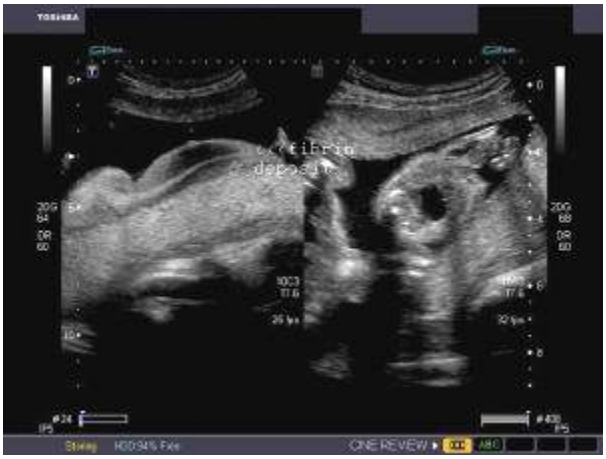
Not clinically significant.

Maternal lakes

These are anechoic lesions in placenta which are actually blood filled spaces. Many are an early stage in evolution of intervillous thrombosis or perivillous fibrin deposition. So there is not much clinical significance attached to these too.

Septal cysts

These appear on ultrasound as anechoic or hypoechoic intraplacental lesions. These are not considered to be pathologic.



Pathologic intraplacental lesions

Infarcts

On USG these appear as triangular lesions with their base towards maternal surface of placenta. On histopathology these contain villi that have undergone coagulation necrosis due to disruption of maternal blood supply. Small areas of infarction do not have much clinical significance . Infarctions which involve more than 10 % of the placenta are considered to be extensive and are associated with IUGR , fetal hypoxia , and fetal demise . Infarcts cannot be visualized on USG unless they are complicated with hemorrhage.

Non trophoblastic tumors

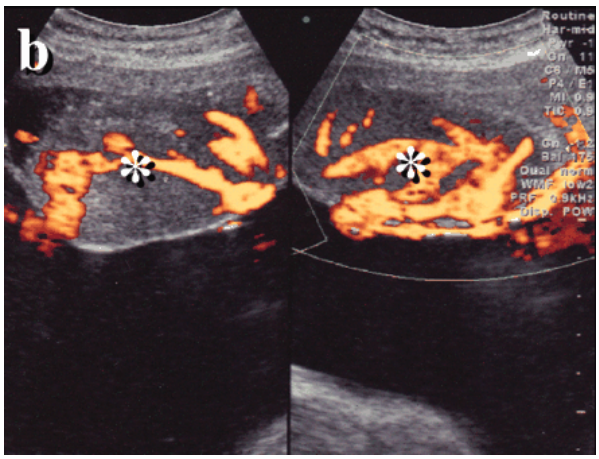
Primary placental tumors

Teratoma
Chorioangioma

These are usually solitary and not affecting much until they are sufficiently large to cause fetal compromise.

Metastatic changes

Maternal tumors which have metastatized to placenta include - CA breast , CA lung , melanoma.



Molar change

H mole on USG looks like solid collection of echoes with anechoic numerous spaces. There can be associated massive bilateral ovarian enlargement.In true mole no fetus is seen alongwith . If fetus is present that means it is a twin pregnancy in which one placenta has become H mole while the second has developed normally.

Retroplacental area

There is a thin hypoechoic line which is normally seen between the retroplacental myometrium and the decidua. It can be absent in certain pathologic conditions :

Placenta Acreta/accrete

Placenta is adherent to the underlying myometrium.

Placenta increta

Placenta invades the myometrium.

Placenta percreta

Placenta compeletly penetrates the myometrium.

Antepartum hemorrhage

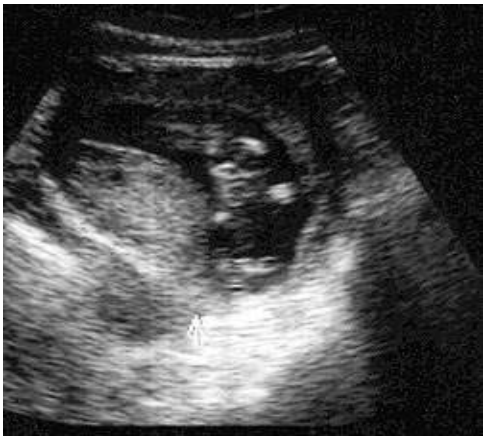
Placenta previa

Placenta extending into the lower segment of the uterus with or without covering the internal os is placenta previa. Placenta which is present less than 4 cm away from internal os is previa .

Two situations which can give false impression of placenta previa are –

An over distended bladder which extends much higher than usual and placenta starts appearing to be previa . The confusion resolves when patient is asked empty to bladder.

Second is during uterine contractions . Contractions in first and second trimester usually mimic placenta previa. So such a patient should be rescanned after 30 mins and again after an hour if necessary.



Abruption placentae

Clinical picture of such a patient is very diagnostic and sometimes there is no time to do an ultrasound . If patient is stable and USG in done then an illdefined echogenic collection can be seen which is either isoechoic or hypoechoic to the placenta.

Chronic retroplacental or sub membranous hematoma

May be seen as early an 9 wks of pregnancy , these usually resolve on follow up . Small lesions do not pose much risk to the fetus but cause for threatened abortion should be looked for.

Doppler in IUGR

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Chairman,Imaging Science Committee.,Fogsi.

Definition:

IUGR – intrauterine growth restriction, is suggested if gestational weeks determined by USG repeatedly show delay in the third trimester from the weeks determined by CRL or correct LMP.

So, very simply speaking, it is a process, which limits the intrinsic fetal growth potential in utero. Though it must not be forgotten that the normal fetal growth is influenced by genetic predisposition, parental influence, ethnic differences, environment (altitude) and fetal gender also.

The incidence of IUGR varies from 1 % to 12 %.

It is essential to diagnose IUGR because , these fetus have lots of immediate and late consequences like higher perinatal morbidity and mortality, perinatal asphyxia, hypothermia, persistent fetal circulation, hypoglycemia, polycythemia and some neurological deficits and learning and behavioural problems.

To decide whether the fetus is IUGR or not, it is essential to first **establish the correct gestational age** and to assess the fetal growth by biometric measurements. The gestational age can be established by either LMP, which has a higher error rate or by first trimester or possibly earliest USG – CRL measurement, which has lowest deviations from standard measurements. So earlier the fetus/ embryo is measured , more accurate is its age assessment. Even if the dating is done in the second trimester it is reliable, because acceleration of normal fetal growth does not begin till the third trimester. If the fetus is assessed for the first time in the third trimester, the transcerebellar diameter and the clavicular length can be taken to exactly assess the gestational age, because these are the ones which show the gestational age most accurately and are not affected by IUGR.

Commonest Causes of IUGR:

1. **Maternal**
    a. Pregnancy induced hypertension
    b. Chronic hypertension
    c. Severe Diabetes mellitus
    d. Collagen vascular disease
    e. Cardiac or renal disease

f. Smoking and poor nutrition
g. Previous history of IUGR baby.

2. **Uterine –placental**
    a. Uteroplacental dysfunction
    b. Placental infarct
    c. Chronic abruption
    d. Multiple gestation – twin to twin transfusion
    e. Confined placental mosaicism
3. **Fetal**
    a. Chromosomal abnormalities
    b. Confined placental mosaicism
    c. Major structural anomalies
    d. Skeletal dysplasias
    e. Multiple anomaly syndromes
    f. Infections
    g. Teratogens

Of all these if the aneuploidies are excluded, the commonest causes of IUGR are pregnancy induced hypertension and uteroplacental vascular dysfunction.

Classifications

- Campbell : first tried to classify IUGR based on BPD only and divided as **"Early low profile"** IUGR or **"late flattening"** IUGR
- Levi et al added a further dimension to this by taking HC/AC into consideration and classified IUGR as **Harmonious or Proportionate and disharmonious or disproportionate.**
- Anglo Saxon specialists then named them as **Symmetrical / asymmetrical** and added a third entity as **Symmetrical IUGR with femur sparing**. Naturally femur length was also taken into classification here.
- Finally in 1976**, Integrated classification** came in which took all the basic aspects of IUGR in consideration .

So integrated classification takes into account

1. Time of onset
2. Etiology
3. Anthropometrical data (HC,AC, FL, Wt.)
4. General morphology
5. Trophism (eutrophic, hypotrophic, dystrophic).

Integrated Classification:

– By Kurjak 1976

Type I : Harmonious

In harmonious or symmetric IUGR, adverse factors affect since conception or embryonic stage (hyperplastic stage). The fetal weight, length and HC are uniformly affected. The fetuses are hypoplastic but eutrophic fetuses and may be called microsomies. The chances of aneuploidy in this group is 25%, most commonly trisomy 18 and 13(Dicke and Crane) and chances of malformations are very high. 20-30% are of this type.

Type II: Disharmonious

This is most commonly seen with uteroplacental insufficiency and affects late stage of pregnancy (hypertrophic stage). It has little or no effect on HC and FL. It typically presents as large head and undernourished body. 70-80% of IUGR are of this type.

Type III : Semiharmonious

This is also due to extrinsic factors but they affect the growth early. It is more commonly due to nutritional deficiency. The fetus is hypotrophic / undernourished in appearance.

When first introduced, the symmetrical IUGR was suggested to reflect an underlying fetal cause like aneuploidies, where as asymmetrical IUGR was supposedly thought to be due to uteroplacental dysfunction. But this proved to be wrong. Triploidy fetuses show asymmetric IUGR and both symmetric and asymmetric IUGR babies show similar degree of acid-base impairment.

Because the Doppler study is useful for the diagnosis of IUGR type II, we will chiefly discuss that only.

Risk factors for IUGR

More than 50 % of pregnancies are free of any associated conditions that would alert to the possibility of IUGR. Though the high risk mothers are with

- Previous History of LBW / IUGR
- Hypertension
- Autoimmune diseases
- Smokers

Diagnosis of IUGR

1. Fundal height below 5th percentile – chronic distress is likely
2. Biometric findings
   a. depend on the scan in the first trimester - Crown rump length
   b. If only in 2nd trimester, depend on least variable parameters
   e.g. cerebellar diameter But the gestation age estimation and diagnosis of IUGR is also dependable by:
   Biparietal diameter
   Head circumference and cerebral index
   Abdominal diameter and circumference
   Femur length
   Fetal organ biometry
3. Doppler findings : most reliable method to predict and diagnose IUGR that is chiefly induced by uteroplacental insufficiency.
   Depend on the first scan at 22 – 24 weeks.
   As we are here to discuss only the role of Doppler in the diagnosis of IUGR, we will be discussing on the Type II IUGR, in the diagnosis of which Doppler plays a major role.

Prediction of PIH and IUGR:

The first prediction of IUGR based on Doppler studies of the uterine and umbilical arteries can be done at 22 – 25 weeks.

1. 22 –24 weeks Doppler – high resistance in uterine artery – 24 fold increased chances of preeclampsia and IUGR
2. Persistent early diastolic notch in the uterine artery waveform- increases predictive value of the Doppler study from 4.3% to 23%for IUGR and 68 fold increased risk of developing preecclampsia.
   Bower et al
3. Sensitivity of the Doppler study increases to 80% in patients who would need to be delivered before 32 weeks i. e. patients who are likely to develop a severe PIH and IUGR.

Physiology:

This type of IUGR is chiefly due to compromised uteroplacental circulation, so let's first briefly understand the same.

Normal umbilical artery FVW is the indicator of normal mother to fetus blood supply. Increase in umbilical artery flow velocities and decreased resistance are seen as the pregnancy advances normally and are due to :

– Continuous maturation of placental villi
– Widening of placental vessels
– Continuous rise in fetal cardiac output
– Continuous changes in vessel compliance

– Continuous rise in fetal blood pressure

As a result of lack of widening of some of the spiral arteries and the myometrial circulation, more pressure is required for the large quantities of blood to be flown through these vessels, leading to uterine artery resistance and notch and ultimately reduction in the umbilical artery flow.

IUGR can be divided into four periods of relatively well defined heamodynamic, biophysical and biochemical patterns and doppler plays an important role in diagnosis and decision making.

The biochemical stages are Hypoxemia, Hypoxia and acidosis.

These can be physiologically staged as a silent period of increased resistance. A period of reduction in umbilical artery blood flow and centralization of fetal circulation and decentralization of fetal circulation.

Silent period of increased resistance

Pathophysiology:

–Till decreased villous microcirculation upto 50%, no change is seen in the umbilical artery flow,if there is no defect in maternal supply

Doppler :

–No change till 50% for 3-6 weeks

–Normal FVW in umbilical art., aorta and MCA

–No extra study required if growth and umb. art. waveform is normal

Actually there is fetal hypoxemia, PO2 18 – 19mm Hg, pH 7.20 – 7.25.

Reduction in circulatory oxygen level then is sensed by peripheral chemoreceptors and leads to vagal stimulation of heart with preferential shift of cardiac circulation towards LV leading to increased umbilical artery resistance and low resistance MCA flow. As primary compensatory mechanism to hypoxemia, there is reopening of the ductus venosus to divert blood from liver and increase in the MCA flow in the distal segment for better peripheral cerebral circulation.Therefore,

- Mild increase in umbilical artery PI is the cause of hypoxemia.
- Abnormal MCA/ fetal vessel FVW is the indication of the adaptation to hypoxemia.
- Leading to : Decreased MCA PI in sector 2
  M1/M2 > 1
  Increased velocity and low resistance in DV
- Sometimes increased Aortic PI, but aortic and carotid chemoreceptors are mostly not affected

But Hypoxemia is a result and not a cause of umbilicoplacental circulation abnormality, so low PO2 with no pl. abn., no change in Umbilical artery FVW.

The second phase is the phase of Hypoxia , which can be divided into
Initial phase
Advanced phase
Terminal phase

1. Initial phase:

- Rise in umbilical artery PI but still positive flow throughout the cycle.
- Presence of enddiastolic flow in CCA at 32-34 wks due to fall in resistance leading to reduced CCA and MCA PI
- Umbilical artery PI / MCA PI : best indicator
- Biophysical profile is unaltered or doubtful. (Biophysical score < 7)
- Lasts for 9 – 60 days average of 2-3 weeks.
- Other progressive changes during this period are :

1. Reduced fetal respiratory and somatic movements
2. Reduced amniotic fluid index (5-8cms)
3. Raised aortic PI
4. Raised renal artery PI

2. Advanced phase :

- 80% reduced villous circulation
- Absent enddiastolic flow in umbilical artery
- Absent enddiastolic flow in aortaleading to higher risk of necrotizing enterocolitis and intracerebral haemorrhages, thus increasing the morbidity and mortality.
- Low MCA and CCA PI values
- Loss of fetal reactivity
- Fetus is already suffering from a degree of hypoxemia and acidosis.
- Hypoxemia in 70-80% of fetuses and acidosis in 40-60% of fetuses
- Absent aortic enddiastolic flow + Altered heart rate response has a sensitivity for IUGR of 85% with a specificity of 80%.

3.Terminal Phase :

This is a phase of acidosis.

- Absent enddiastolic flow in umbilical artery.
- Increasing resistance in MCA, due to cerebral oedema.
- Absent diastolic flow in aorta.

- IVC shows negative flow > 12% and Preload index (Pva/PVs) – 0 – 0.37 is normal
- Ductus venosus – reversed flow or decreased telediastolic velocity values
- Pulsating umbilical vein flow with apparent cyclical decrease in the flow

These findings with reversed flow in hepatic vein and deceleration in heart rate are indications of acidosis. In worst cases coronary artery blood flow may also be seen.

Obstetric management depending on Doppler findings:

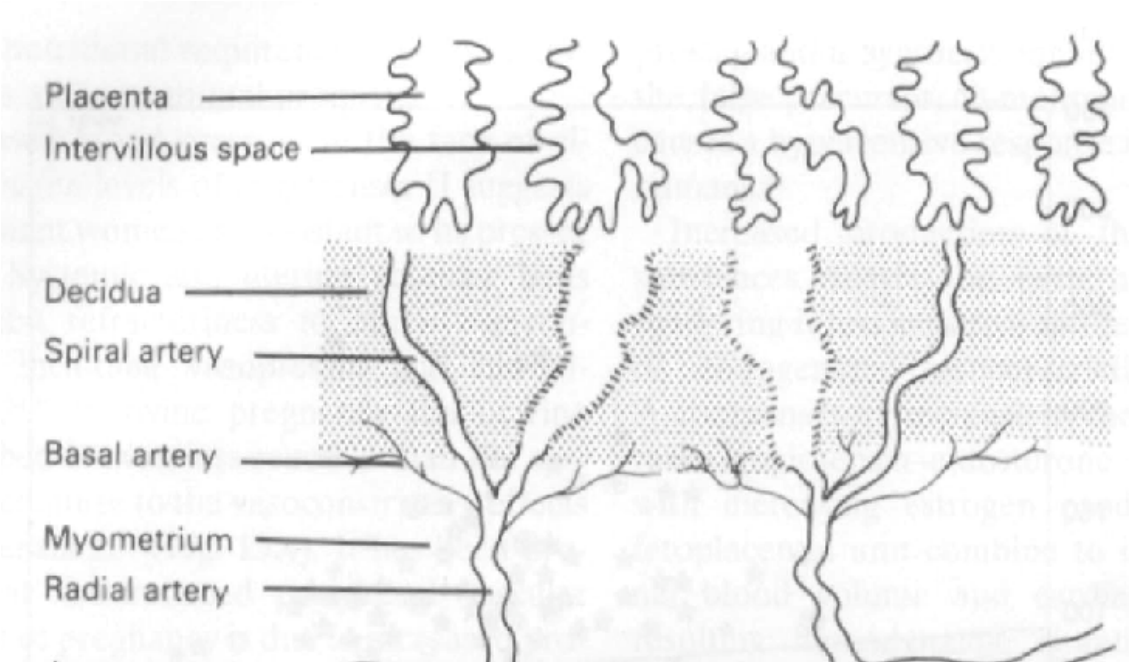
I . IUGR with normal heamodynamic profile : No immediate action and repeat Doppler and BPP every 2 weeks.

II. IUGR with heamodynamic redistribution 32 - 34weeks: early stage of centralization, Doppler and BPP monitoring . With late stage of centralization , termination of pregnancy.

III. If IUGR with heamodynamic redistribution is seen between 28 – 32 weeks, termination of pregnancy with advanced and terminal stages of centralization but expectant management with early stage of centralization with Doppler and BPP monitoring every 5 days.

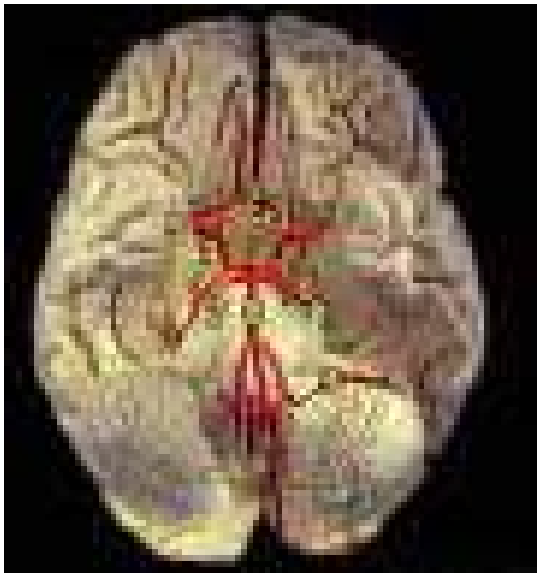
IV. Same is true for the gestational age of < 28weeks. All chances are taken to allow the pregnancy to reach 30 weeks.

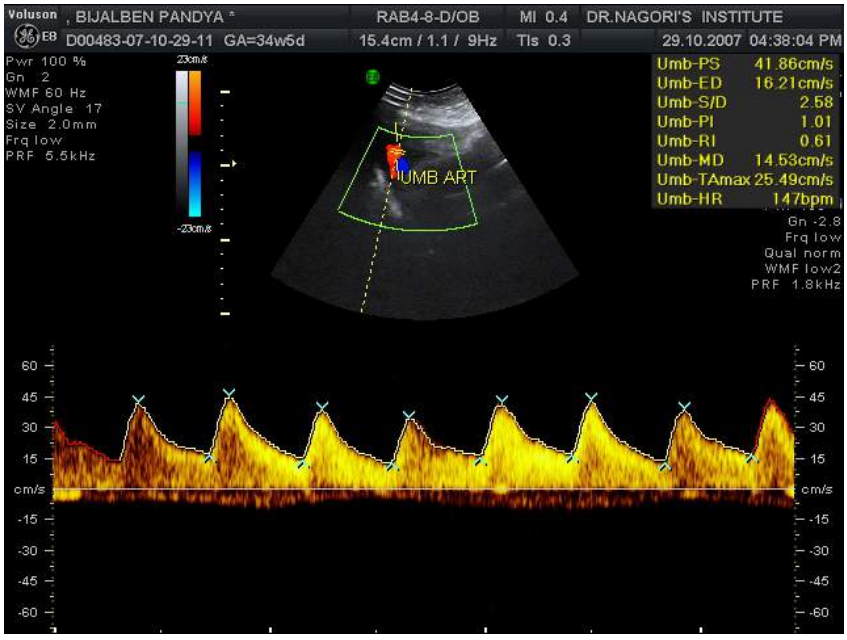
It must be remembered that as long as the fetal growth and the umbilical artery indices are normal, no further heamodynamic study is required and Doppler findings must be evaluated in conjunction with Gestational age, Fetal weight, Liqour, Biophysical profile, Fetal activity, CTG.



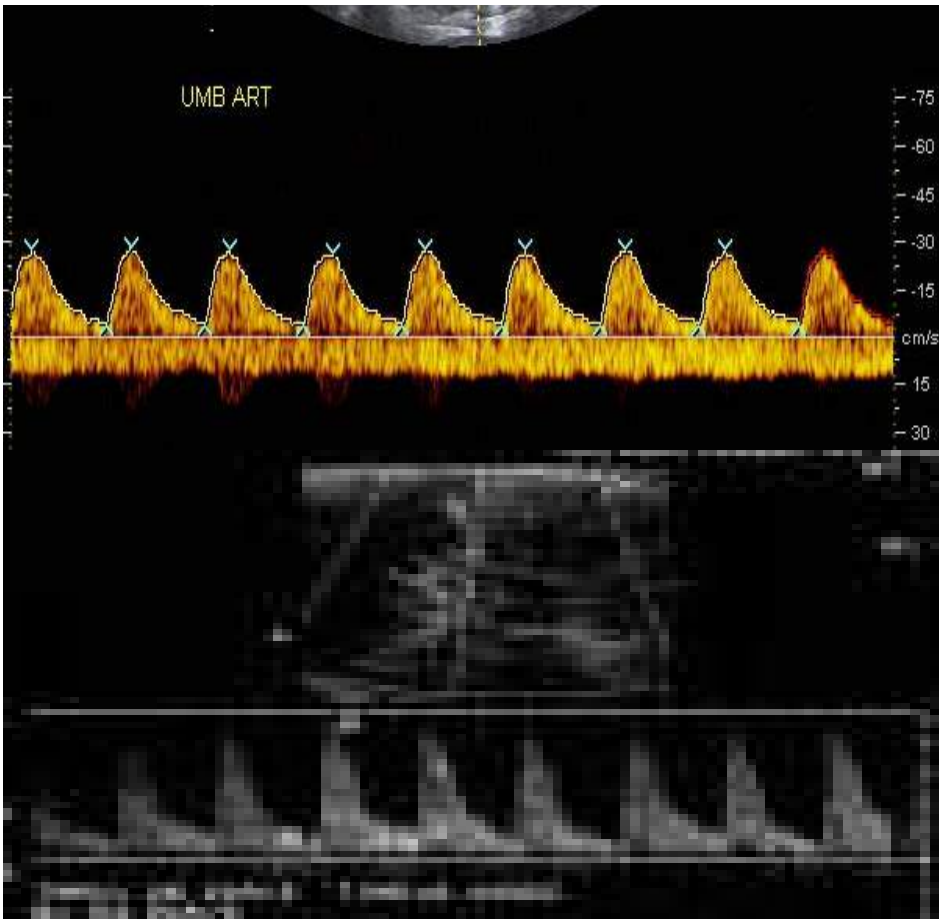
Primary compensatory mechanisms

In response to cardiac / cerebral hypoxia , Fall in PI of MCA in sector 2 with M1/M2 becoming >1(Normal <1).- to divert blood to the peripheral part of the brain.





Initial phase doppler



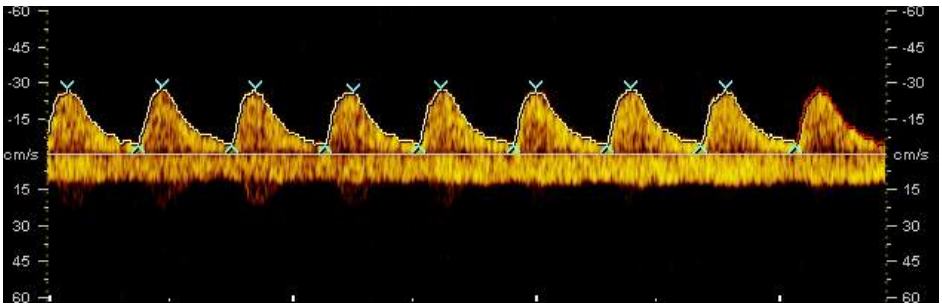
Umbilical Art

MCA

Advanced phase : Doppler

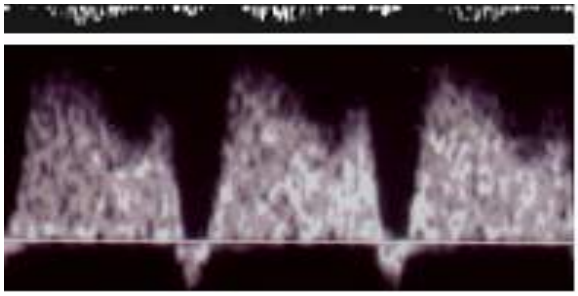
Umbilical Art

Aorta



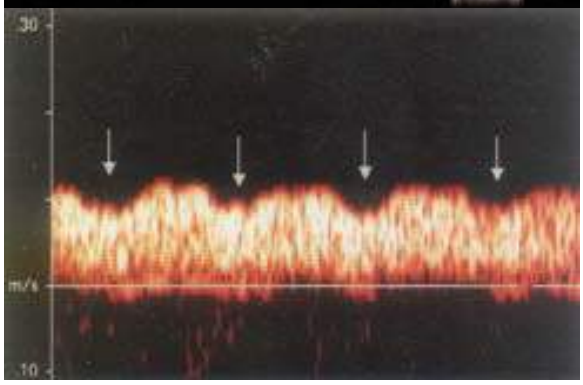
MCA

Venous Doppler findings



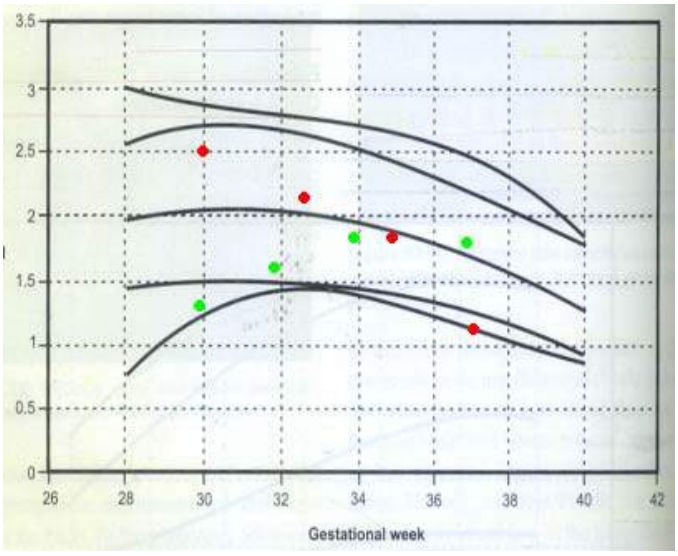
Ductus venosus

Ductus venosus

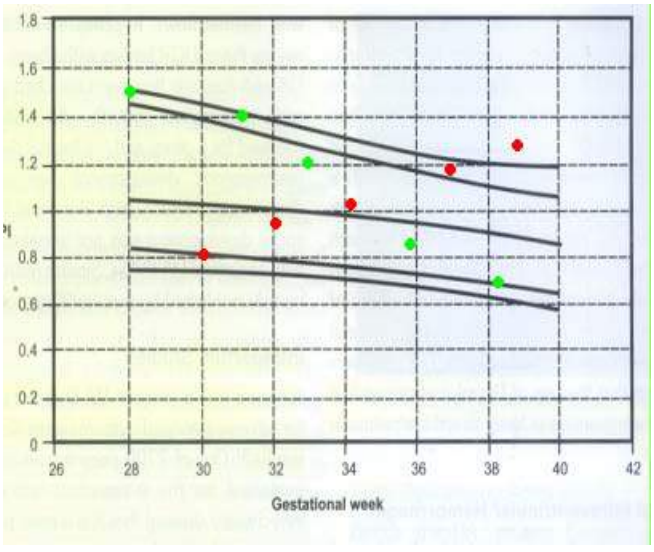


Umbilical vein

Follow Doppler trends



MCA PI



Umbilical art PI

Systematic examination of fetal Central nervous system

Dr. Sonal Panchal MD., Dr. C.B. Nagori MD.DGO.

Introduction:

Abnormalities of central nervous system are the commonest of all congenital anomalies. Neural tube defects are most frequent of these and the incidence is 1 -2 per 1000 births. Though intracranial anomalies with intact neural tube are often missed antenatally and so exact incidence of central nervous system is not known but is estimated to be approximately 1 in 10.

USG has been used for nearly 30 years now for diagnosis of fetal CNS abnormalities. With increasing efficacy of technology the anatomy of CNS can now be studied much more in detail than ever before and as early as late first trimester.

To study and diagnose CNS abnormalities precisely it is important to know the development of the brain as brain has the highest changing anatomy amongst all the body organs during embryonic life. The developmental maturation of the normal brain follows a predictable time table and this maturation can be followed by ultrasonography.[1]

Though in selected cases fetal MRI after 20 – 22 weeks has given promising results, its advantage over ultrasound remains debated.[2]

To make the examination standardized, of this quick changing anatomy, ISUOG (International Society of Ultrasound in Obstetrics and Gynecology) has established guidelines of CNS examination. The examination has been staged as:

I. Optimized approach to the evaluation of fetal CNS : Basic examination

II. Detailed evaluation of fetal CNS : fetal neurosonogram : indicated for pregnancies at high risk for CNS abnormalities and may need 3D USG.

Time of examination:

For basic studies in low risk population, around midgestation - 20 weeks – is a good time for the study of fetal CNS. Studies later in gestation hampers visualization of intracranial structures because of calvarial ossification. First or early second trimester scans at 14 – 16 weeks has an advantage that bones are thin and brain evaluation at all angles is possible. Though with high end machines it is now possible to assess the fetal neural development from as early as 8 – 9 weeks.

Equipment & approach

1. **Transabdominal approach:**
   transducer frequency of 3 – 5 MHz. This is the most commonly used approach usually for basic examination and is adequate for any fetal presentation. The sections most easily obtained by this approach are axial and sometimes coronal. Sagittal sections are very difficult by this approach. The quality of image is deteriorated by factors like maternal obesity, adverse fetal position inadequate liquor and advanced pregnancy leading to calcification of fetal calvarium.
2. **Transvaginal approach :**
   5 and 10 MHz transvaginal probe. Transvaginal probe being a high frequency probe gives much more details and is a more preferred approach for detailed fetal neurosonogram. When ever fetus is in cephalic presentation and fetal head is not too high to be approached, this route of examination is chosen. Gentle manipulation of the fetus may

be required for correct sections and planes. The imaging window is anterior fontanelle.

Method of transvaginal scan:

This scan is done through anterior fontanelle of the fetus. All sections are fanning from front to back or side to side respectively parallel to coronal and sagittal sutures. Transverse or axial sections can not be obtained by this approach.

The coronal sections obtained are divided as frontal 1,2, midcoronal 1,2,3 and occipital 1,2 and the side to side sections obtained are median, paramedian1,2. These sections are similar to the neonatal scans and so easier to understand for pediatricians.

3D may be used with both the approaches whenever indicated.

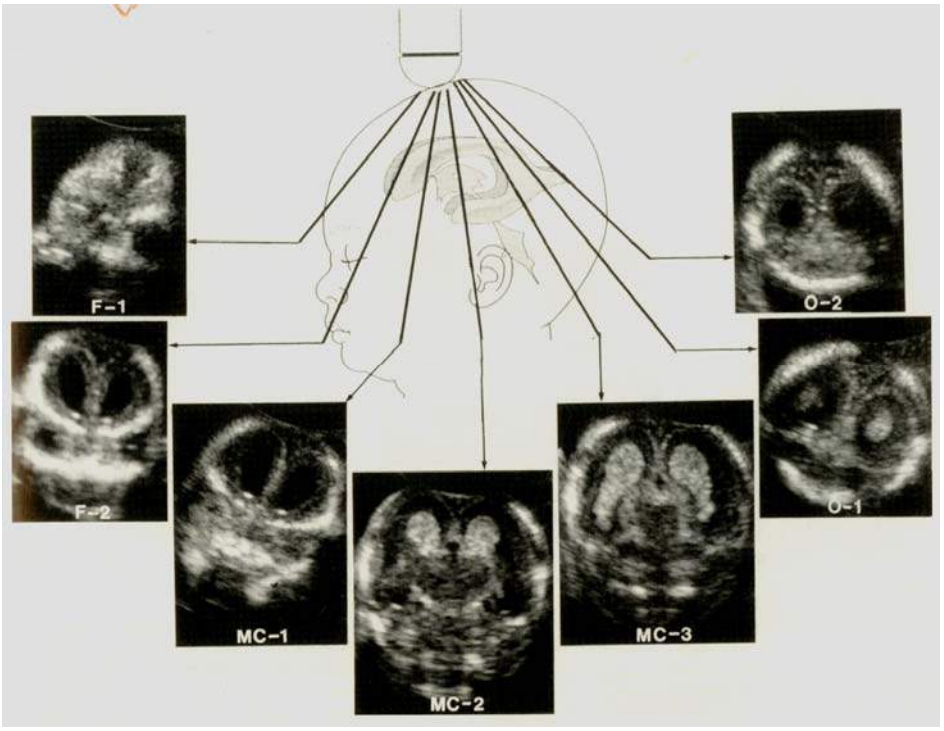


Figure from Prenatal diagnosis, Marks I Evans et al, page 203

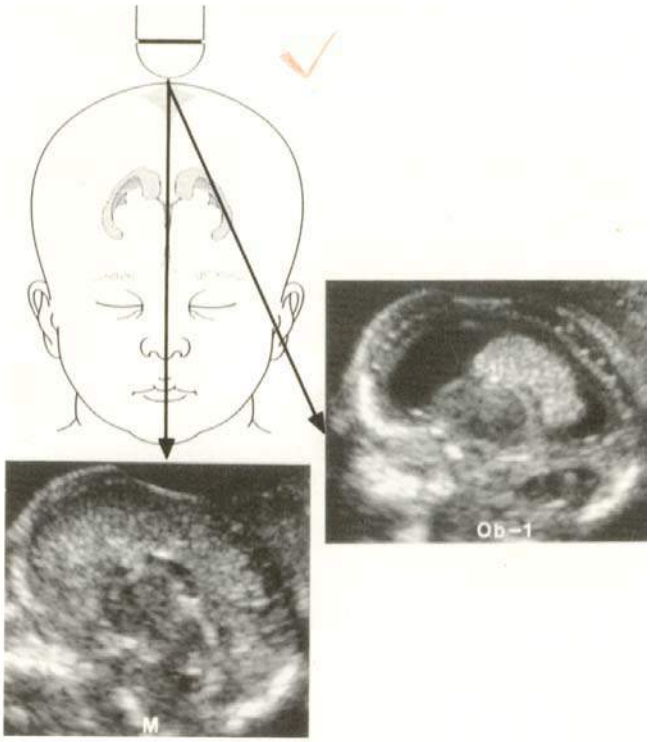


Figure from Prenatal diagnosis, Marks I Evans et al, page 204

Imaging Parameters:

- Gray scale
- Harmonics : enhances subtle anatomical details
- Colour and power Doppler for vascular studies : to pick velocities of 20 – 40 cms/sec
- Increased persistence to pick up small vessels

The basic fetal CNS evaluation is divided into

a. qualitative evaluation

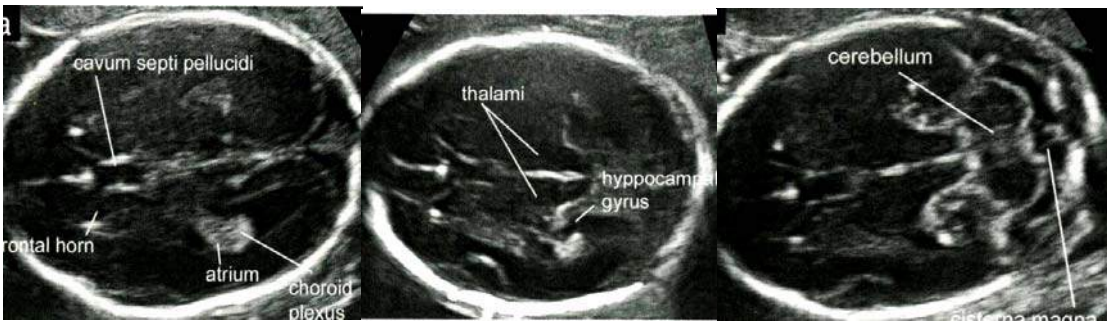
b. quantitative evaluation

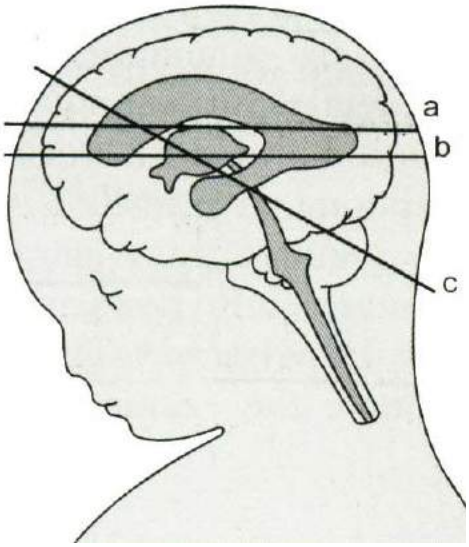
a. **Qualitative Evaluation of head** consists of three Axial planes

   i. Transventricular plane

   ii. Transthallamic plane

   iii. Transcerebellar plane

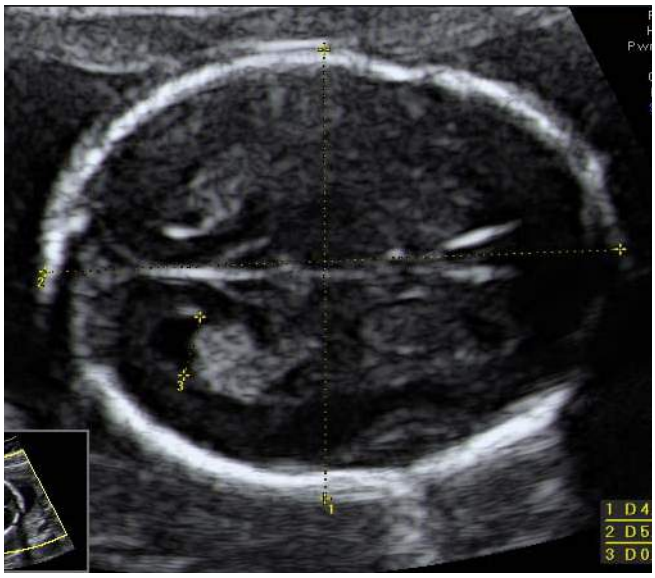




- **Qualitative evaluation of the spine** requires at least two of the three planes
    i. Longitudinal / sagittal
    ii. Transverse / axial
    iii. Coronal

Qualitative examination of the head
i.Transventricular plane

- Head shape
- Lateral ventricles
- Cavum septum pellucidum
- Thallami
- Cerebellum
- Cisterna magna



Transventricular plane

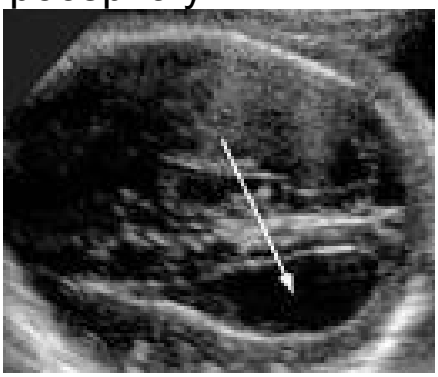
Lateral Ventricles:

Lateral ventricle consists of anterior horn (frontal horn), Posterior horn (Occipital horn) , atrium – contains glomus of choroid plexus and inferior (temporal horn). In first trimester, the ventricles are large and fill up almost the whole cranial cavity with choroids plexus in the centre. As the fetus grows the comparative size of the ventricles decreases and the inferior horn is almost obliterated by 18 weeks. In second trimester the lateral ventricles are seen only as anterior and posterior horns and atrium, which is occupied by choroid plexus. In transventricular section the maximum normal ventricular diameter is 7.6 ± 0.6 and remains fairly constant in 2nd and 3rd trimester. Medial and lateral walls of ventricle are parallel to midline in 2nd and 3rd trimester.[3]

The lateral ventricle to hemisphere ratio changes decreases from 70% at 18 – 20 weeks to 30% at 28 -29 weeks and remains steady there after. The ratio of anterior horn width to the hemisphere decreases from 60% at 14 weeks to 40% at 21st week.[4].

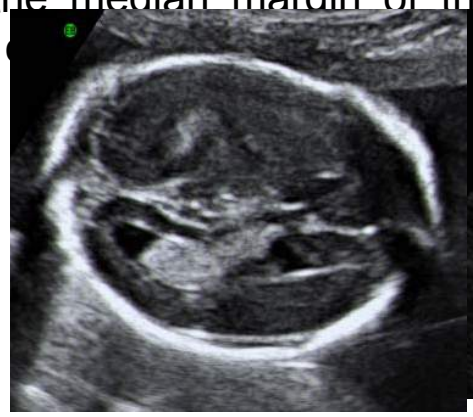
The ventricular width is measured from inner to inner margin at the atrial level. Anything more than 10mm is termed as

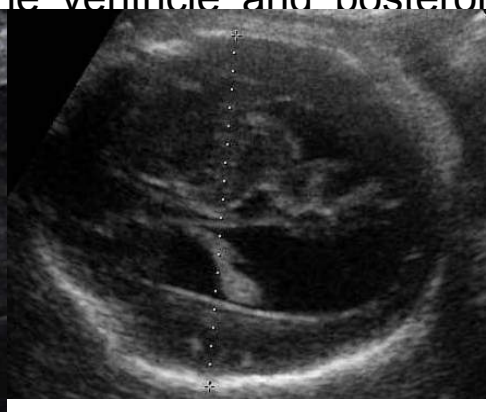
ventriculomegaly and ventricular size of more than 15 mm and intracranial pressure is increased, it is hydrocephalus. Dilatation of only posterior horn(tear shaped ventricles) which is seen in agenesis of corpus callosum, is known as colpocephaly.



Colpocephaly

Choroid plexus normally fills the atrium. Thinned out and hanging dangling choroids plexus is an indirect sign of ventricular dilatation. An angle can also be drawn by a line along the median margin of the ventricle and posteromedial margin **Error!**





Normal ventricle *ventriculomegaly*

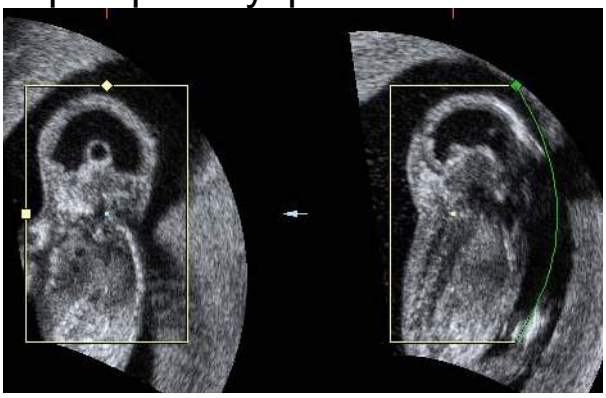
Most severe cerebral lesions are symmetrical and in basic examination symmetry of the brain is assumed, though most of the times the hemisphere on the near side is not visualized.

Cavum Septum Pellucidum:

Sonographically it appears as 2 sheets of tissue which extends from corpus callosum and separates the lateral ventricles from cavum. It becomes visible at 16 weeks and remains nearly

steady through out the 2nd trimester but, obliterates near term. It must always be visualized between 18 and 37 weeks when BPD is of between 44 – 88 mm. Septum cavum pellucidum is not seen in

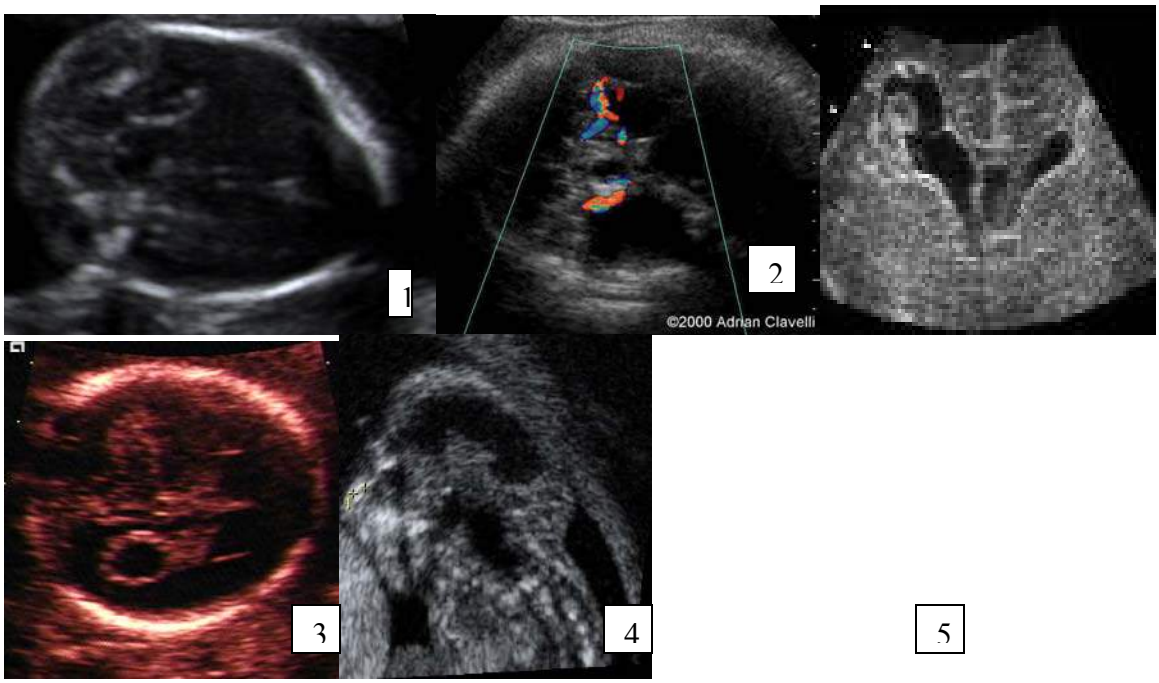
- holoprosencephaly,
- agenesis of CC,
- severe hydrocephaly
- septooptic dysplasia.



Holoprosencephaly

Pathologies diagnosed on this plane

Lemon skull
Arachnoid cyst
Porencephalic cyst
Choroid plexus cyst
Anencephaly

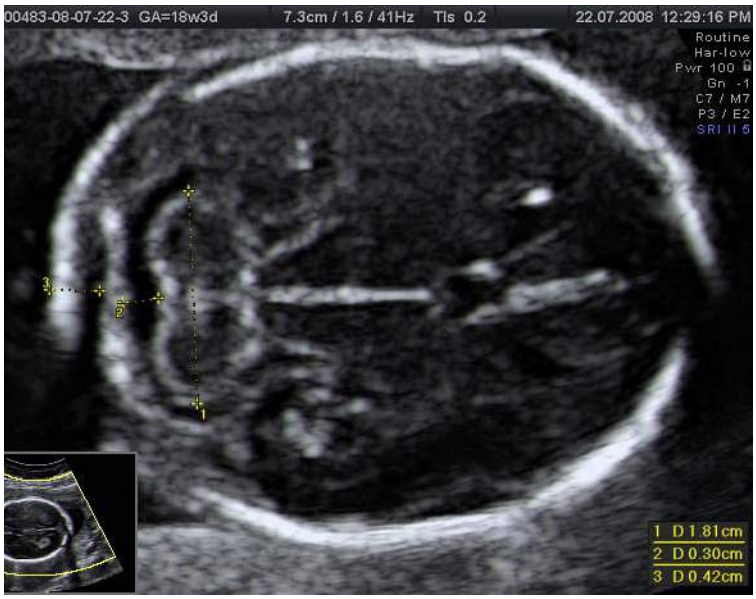


1. lemon shaped skull, 2.arachnoid cyst, 3. porencephallic cyst, 4. choroids plexus cyst, 5. anencephaly

2. Transcerebellar plane :

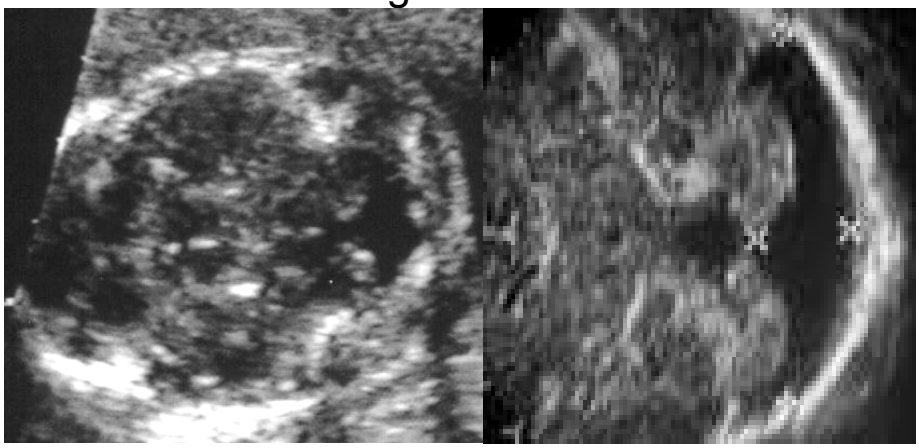
It cuts through cerebellum and shows

- Frontal horns of lateral ventricles
- Cavum septum pellucidum
- Thallami
- Cerebellum:
  - Two hemispheres
  - Vermis – after 20 weeks



Transcerebellar plane

Cerebellum which is seen as a B shaped structure in the posterior fossa in 2nd trimester and onward pregnancy, becomes visible as early as 11 – 12 weeks. In 2nd trimester, its diameter in mms corresponds to the number of weeks of gestation. In late 2nd and third trimester sulci and gyri can also be appreciated on the cerebellar surface. Between the two cerebellar hemispheres a thin solid structure is seen called vermis. Inferior vermis is seen at little lower level than superior vermis. It is consistently visible after 18 weeks, but is be absent in Dandywalker syndrome and variants and also as a solitary finding. When inferior vermis is absent the cisterna magna communicates with the 4th ventricle.



Absent inferior vermis – Dandy walker malformation

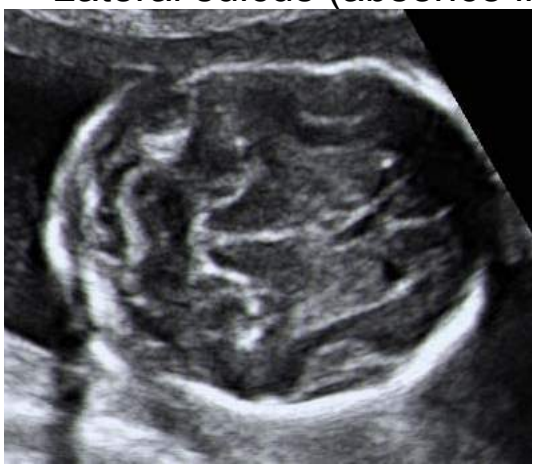
Cisterna magna :

This is the space between the cerebellum and the occipital bone and shows thin arachnoid septations as lines crossing the lucent space anteroposteriorly.

3.Transthallamic plane :

This plane is commonly known as biparietal plane and is easily reproducible in late pregnancy. It shows

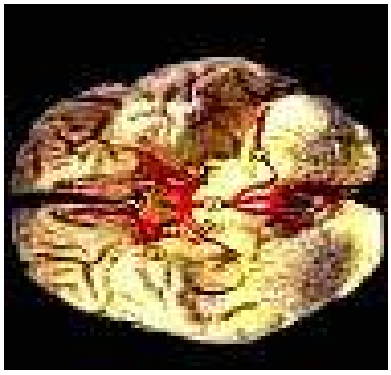
- Frontal horns of lateral ventricles
- CSP
- Thallami
- Hippocampal gyrus
- Lateral sulcus (absence indicates lissencephaly)



Transthallamic plane

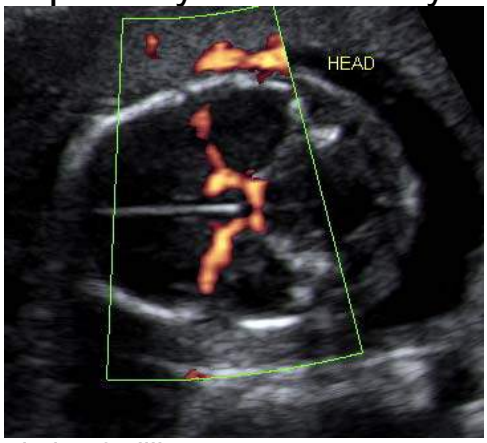
Study of the cerebral vasculature:

Cerebral circulation is first detectable at 8 weeks and can be seen in all the fetuses at 11 weeks. The circle of Willis which is formed by two posterior cerebral arteries, a posterior communicating artery, two middle cerebral arteries, two anterior cerebral arteries and anterior communicating artery, is the landmark of fetal circulation.



This circle can be examined on axial plane and MCA is the most commonly interrogated vessel in fetal circulation. From transthallamic view, slight angulation of the probe towards skull base gives a good view of circle of Willis. From circle of Willis, vessel traveling towards the probe in Sylvian fissure is middle cerebral artery. It has a high resistance flow in first trimester but with increasing gestational age the resistance decreases and peak systolic velocity in this vessel increases. MCA PI remains constant till 32 weeks and then falls.

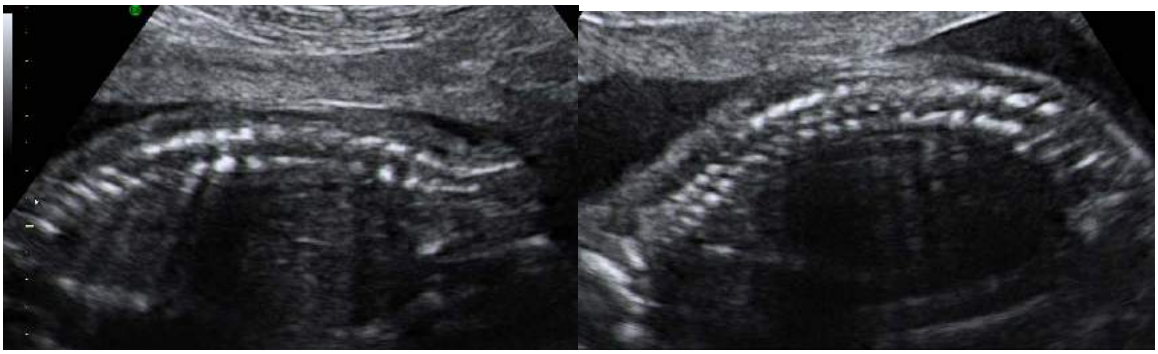
Low resistance (< 5^{th} centile) flow in this vessel especially in third trimester is an indication of compensating fetal hypoxia. But an absent diastolic flow in this vessel in third trimester is often an indication of noncompensating fetal hypoxia and cerebral oedema, though it needs to be correlated clinically as well as other ultrasonographic signs of fetal hypoxia. Increase in peak systolic velocity beyond 2SD indicates fetal anemia.



circle of willis

Qualitative evaluation of Fetal Spine

Full examination of spine with all projections is not a part of basic examination. This requires expertise and extra skill. Longitudinal section is must. After 14 weeks always three ossification centres are seen as three parallel lines. It is important to see intact overlying skin on longitudinal and transverse section.



longitudinal spine

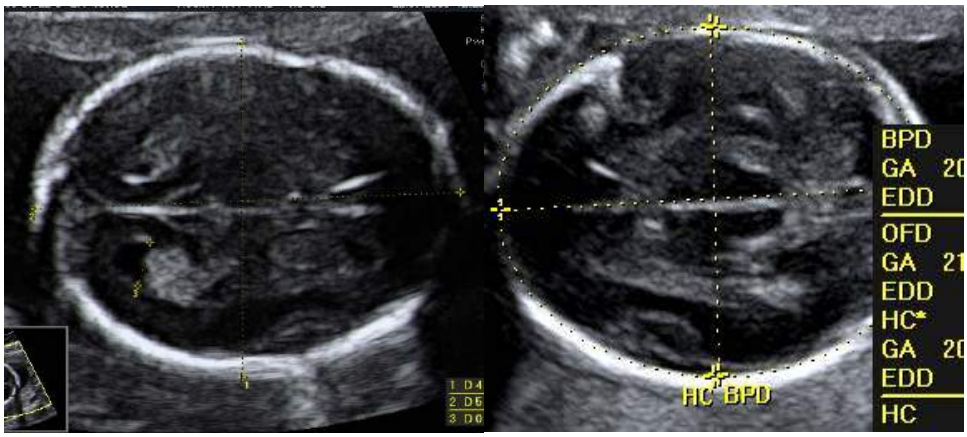
Quantitative Assessment of head:

Measurements include:

- Biparietal diameter
- Head circumference
- Internal diameter of the ventricular atrium
- Cerebellar transverse diameter
- Depth of cisterna magna

Biparietal diameter

It has been a trend for many years to measure the biparietal diameter in transthallamic plane and to measure it from outer to inner margin. The Fetal medicine foundation though prefers to measure it in transventricular plane and measure it from outer to outer wall. As far as assessment of growth is the matter it is essential that the same method and the same section are always used for this measurement.



BPD : Transventricular plane, Transthallamic plane

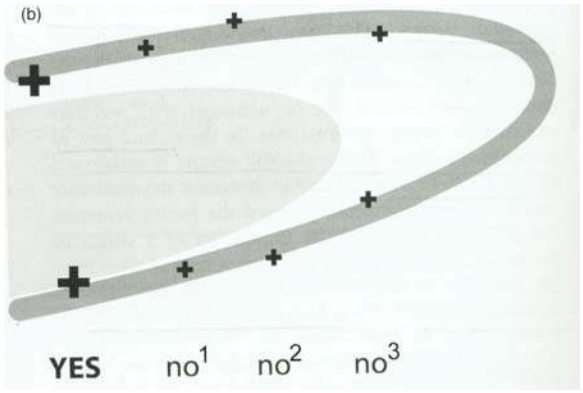
Head circumference

Is measured from outside the skull bone echoes or may be calculated as HC = 1.62 x (BPD + OFD). Shape of the skull is represented by the ratio BPD / OFD = 75 – 85%.

Moulding of fetal head in early gestation is frequent. Breech presentations may show mild dolichocephaly where this ratio is decreased and in brachycephaly this ration is increased.

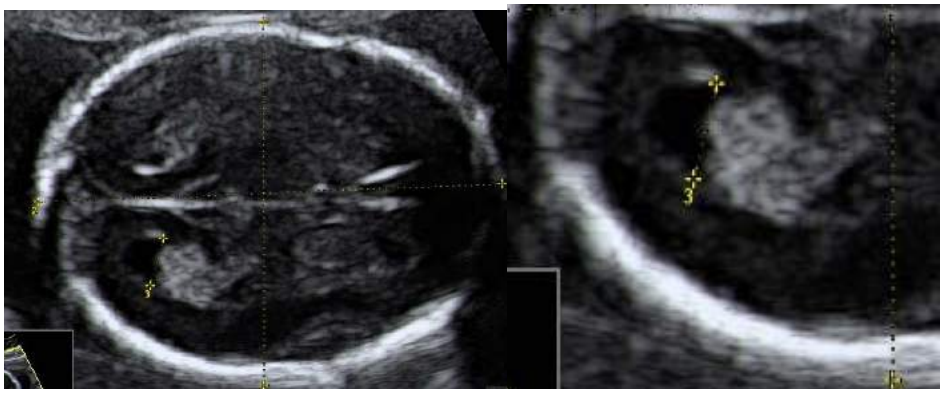
Ventricular atrium:

It is measured at the level of glomus of choroid plexus, perpendicular to the ventricular cavity. Calipers are placed inside the ventricular walls.



In second and third trimester it measures 6 – 8 mm, 8 – 10 mm is borderline ventriculomegaly and > 10 mm is ventriculomegaly. Ventricular diameter of upto 10 mm is treated

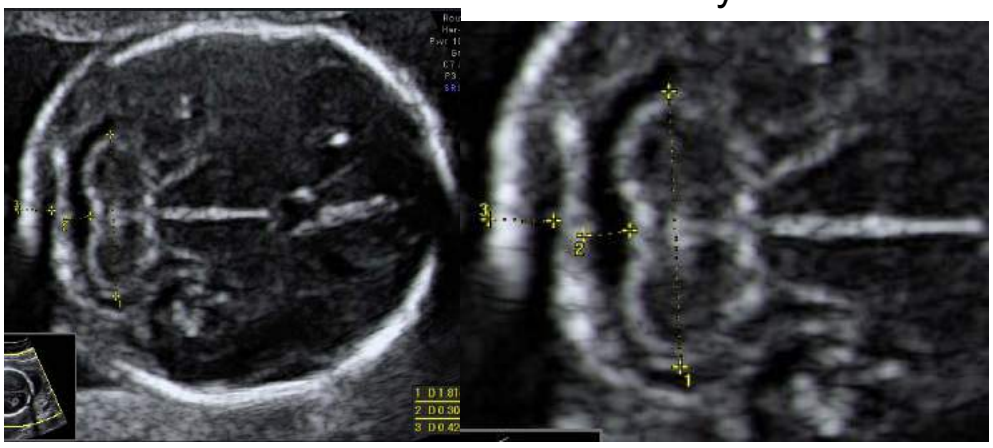
expectantly, if it is a solitary finding. Though it is considered as a soft marker for chromosomal abnormality. It is a marker of abnormal cerebral development. Ventricular diameter of > 15 mm with increased pressure is called hydrocephalus and may be associated thinning of skull vault and widening of sutures. This may require in utero or immediate postnatal treatment of the fetus.



measuring ventricular width

Cerebellum

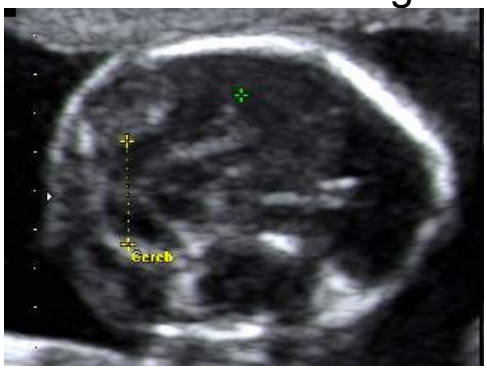
It is measured from outer to outer margin. Transverse cerebellar diameter increases by 1mm every week from 14 to 21 weeks. Diameter corresponds to the weeks of gestation in second trimester and is not affected by IUGR.



Cisterna Magna

It is measured from posterior margin of cerebellar vermis to the anterior margin of overlying occipital bone. It measures 2 – 10 mm but is slightly more in dolicocephaly. It is enlarged in

cerbellar pathologies, especially in vermian ones, like Dandy walker's malformations or variants but in neural canal defects when cerebellum is pulled caudally, it is obliterated. This is known as 'banana sign' of cerebellum.



Banana sign, lemon sign

For patients with increased risk of CNS abnormalities and suspicious findings on basic examination, a detailed fetal neuroscan is required which includes the use of multiplanar and 3D ultrasound. It has a much greater diagnostic potential. Orthogonal planes – axial, sagittal and coronal, are obtained by aligning the transducer with sutures and fontanelles of fetal head.

Axial, coronal and sagittal sections of the spine are also studied as a part of **detailed fetal neuroscan**.

Systematic evaluation of the head consists of detailed study of anatomical structures by

- Four coronal planes
- Three sagittal planes

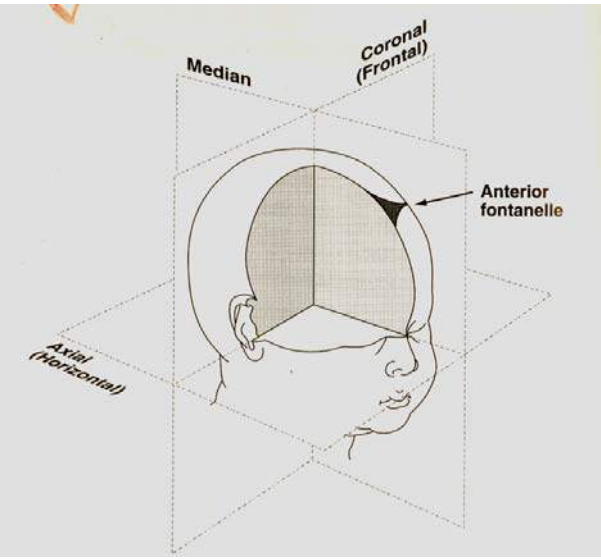
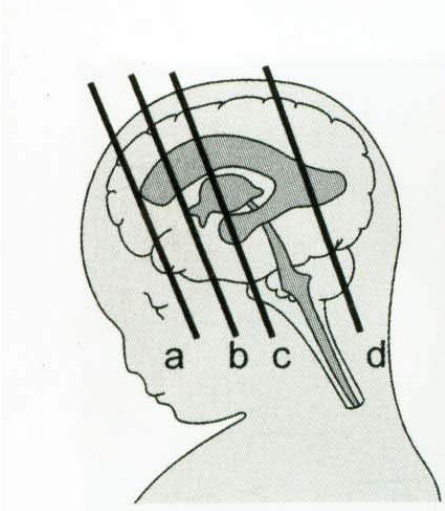


figure from prenatal diagnosis. Mark Evans; Imaging of fetal brain, Monteagudo A, Timor Tritsch I; page 200.

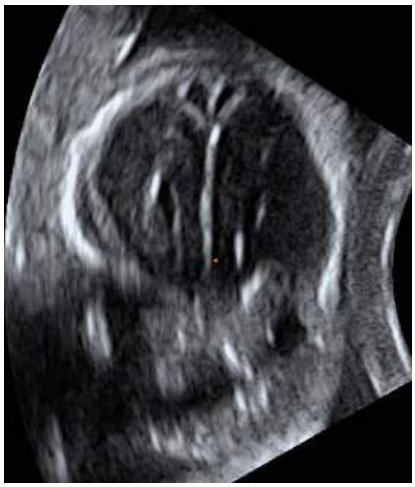
Coronal planes

1. Transfrontal plane (frontal 2 plane)
2. Transcaudate plane (midcoronal 1 plane)
3. Transthallamic plane (midcoronal 2 plane)
4. Transcerebellar plane (Occipital 1 & 2 plane)



1.Transfrontal plane

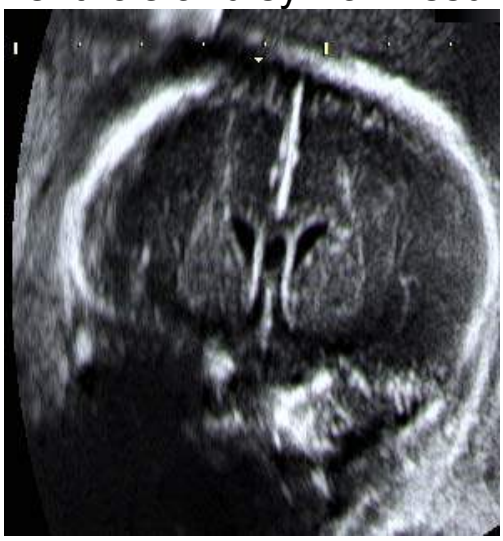
This plane cuts through anterior fontanelle & is rostral to the genu of corpus callosum. It shows midline hemispheric fissure, anterior horns of lateral ventricles, spenoidal bone and orbital plates.



Transfrontal plane

2. Transcaudate plane :

This section is at the level of caudate nuclei and cuts genu of corpus callosum. It shows corpus callosum in cut section, triangular cavum septum pellucidum, frontal horns of lateral ventricle and sylvian fissures laterally.

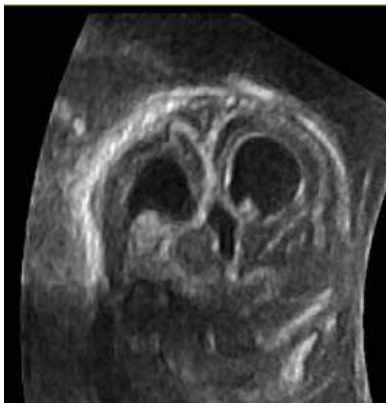


transcaudate plane

3. Transthallamic plane:

This section cuts across the thalami which are seen closely apposed to each other on the sides of midline. Third ventricle is seen in midline. Third ventricle more than 5mm is considered as dilated.This section also shows interventricular foramina and atria of lateral ventricles with choroid plexus, basal cistern

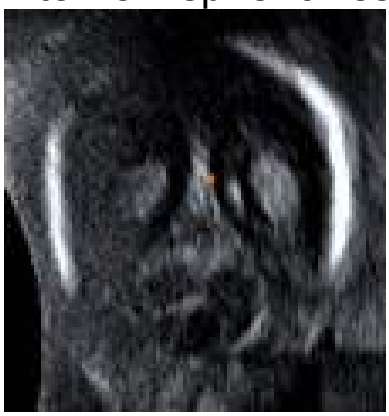
containing vessels of circle of Willis and optic chiasma close to the skull base.



Transthallamic plane

4. Transcerebellar plane:

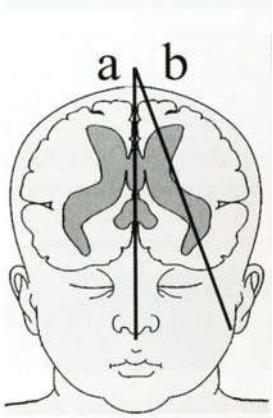
This section is taken across the cerebellum, through posterior fontanelle. It shows occipital horns of lateral ventricle, interhemispheric fissure, cerebellar hemispheres and vermis.



Transcerebellar plane

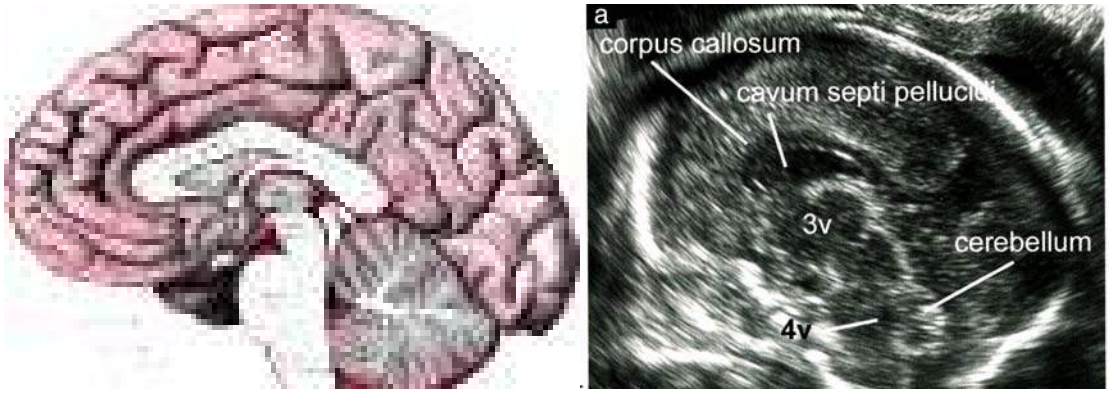
Sagittal planes

1. Mid sagittal
2. Right parasagittal
3. Left parasagittal



1. Midsagittal plane:

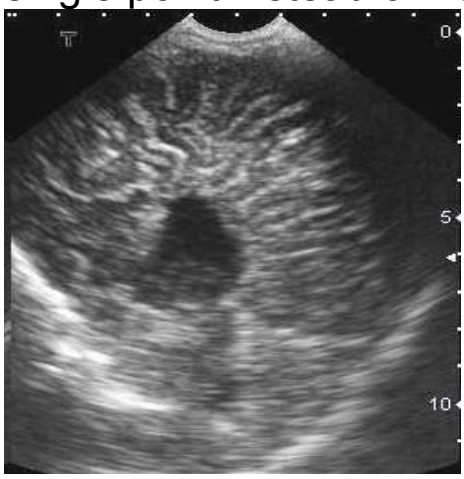
This section is in midline along the sagittal suture and shows corpus callosum, and cavum septum pellucidum under it. In some cases cavum vergi and cavum veli interpositi may also be seen. Posterior part of the section shows brainstem, pons, vermis and 4th ventricle, possibly aqueduct also. Fourth ventricle is seen in median plane as a sonolucent triangle at the level of cerebellum.



midsagittal plane

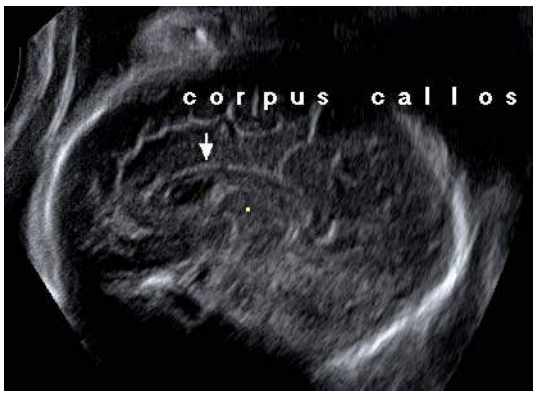
Corpus callosum starts forming at 12 weeks and is completed at 20 – 22 weeks. It can be seen only on sagittal section. Indirect sign of its presence is pericallosal artery. Indirect sign of absent corpus callosum is tear shaped ventriculomegaly/ colpocephaly on axial section and directly upward pointing frontal horns on coronal section. But sagittal section shows

absent corpus callosum with cortical sulci and gyri arising from single point instead of from coronal section.

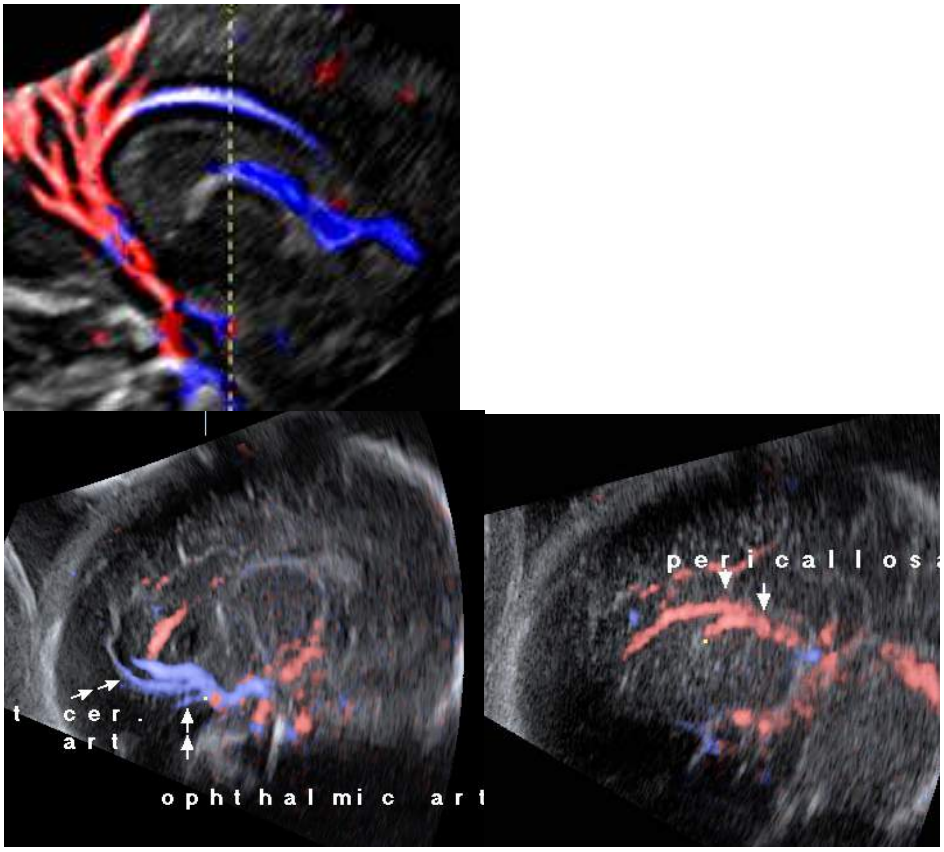


Agenesis of corpus callosum

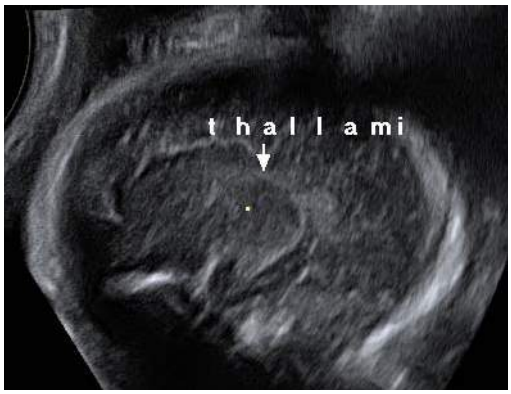
Cingulate gyrus and sulcus, the parieto-occipital fissure and calcarine fissure can be seen in this sections. Both lateral and cingulated gyri appear between 22 – 24 weeks. Though this is pretty late than its anatomical appearance which is at 18 weeks. Maximum sulci and gyri appear between 28 – 30 weeks.

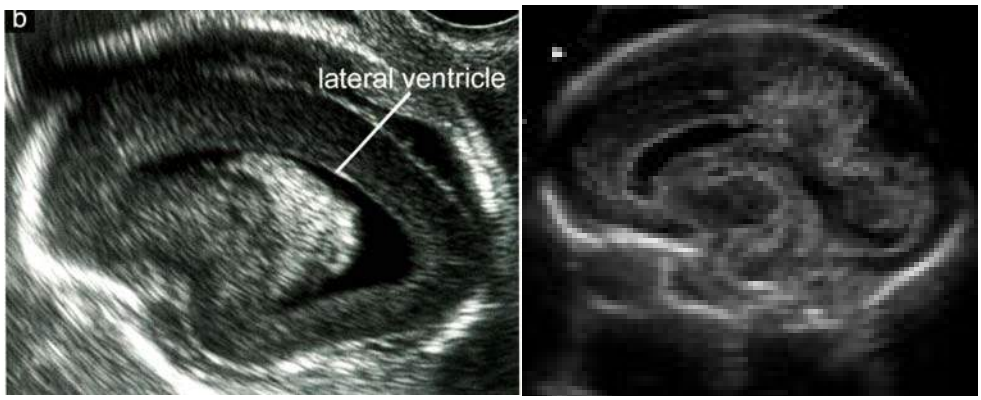


Doppler studies in mid sagittal section explains the branches of internal carotid artery. Its main branches are anterior and middle cerebral arteries and pericallosal artery. Superior sagittal sinus and vein of Galen can also be seen on this section.



Parasagittal or oblique plane shows entire lateral ventricle with choroid plexus, paraventricular tissue and cortex.
This plane is for diagnosis of intraventricular heamorrhage.

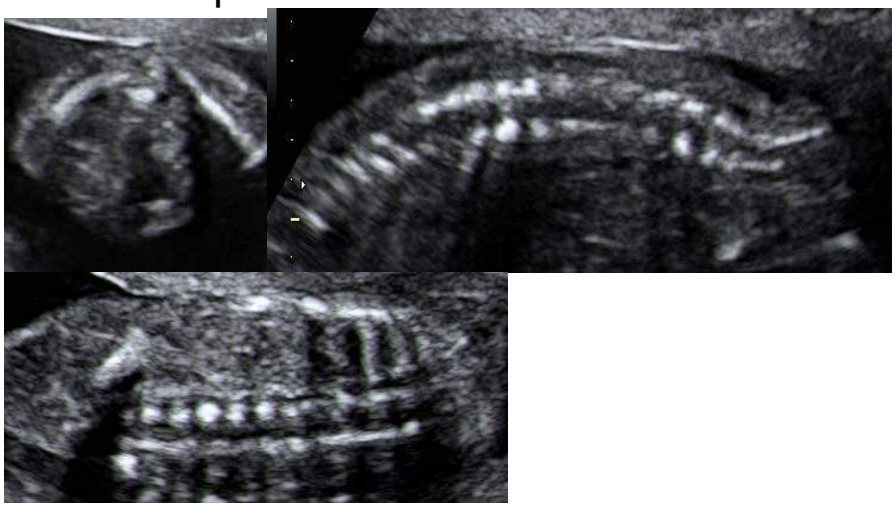




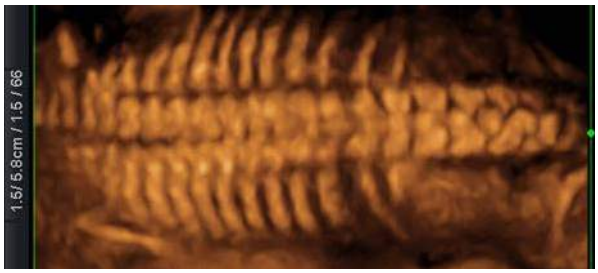
Amongst the coronal planes the midcoronal plane is most revealing. This shows middle cerebral artery, ventriculostriate arteries, cross section of pericallosal arteries and superior sagittal sinus.

Fetal Spine examination in high risk patients demands study of all the three planes.

- Transverse / axial plane
- Sagittal / longitudinal plane
- Coronal plane

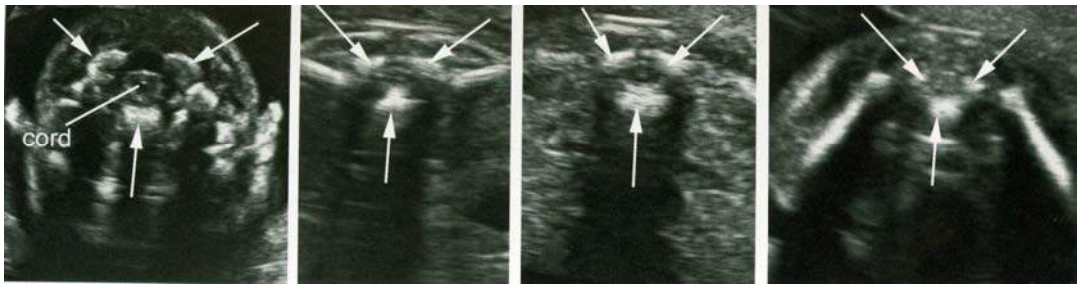


Usually only two of these three planes can be examined in any fetus depending on its position but 3D US may be used of required to obtain all the three planes.



3D US of spine

Transverse or axial plane of spine is obtained by sweeping the probe through the entire fetal length. When this is done the level of fetal spine must be kept in mind as vertebrae have different anatomy at different levels. Upper cervical vertebrae are quadrangular, thoracic and lumbar vertebrae are triangular and sacral vertebrae are flat.



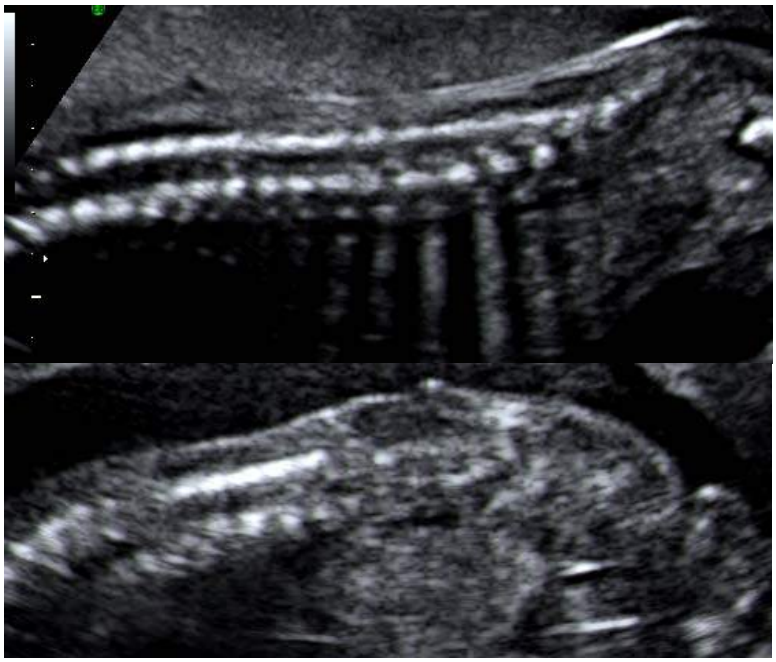
Spine in transverse sections

On sagittal plane:

The ossification centers of vertebral bodies and posterior arches form two parallel lines which converge at the sacrum.Spine if anterior – can also see spinal canal and spinal cord, through the unossified spinous processes.

Conus medullaris at L2-3 can be seen in 2^{nd} and 3^{rd} trimester.

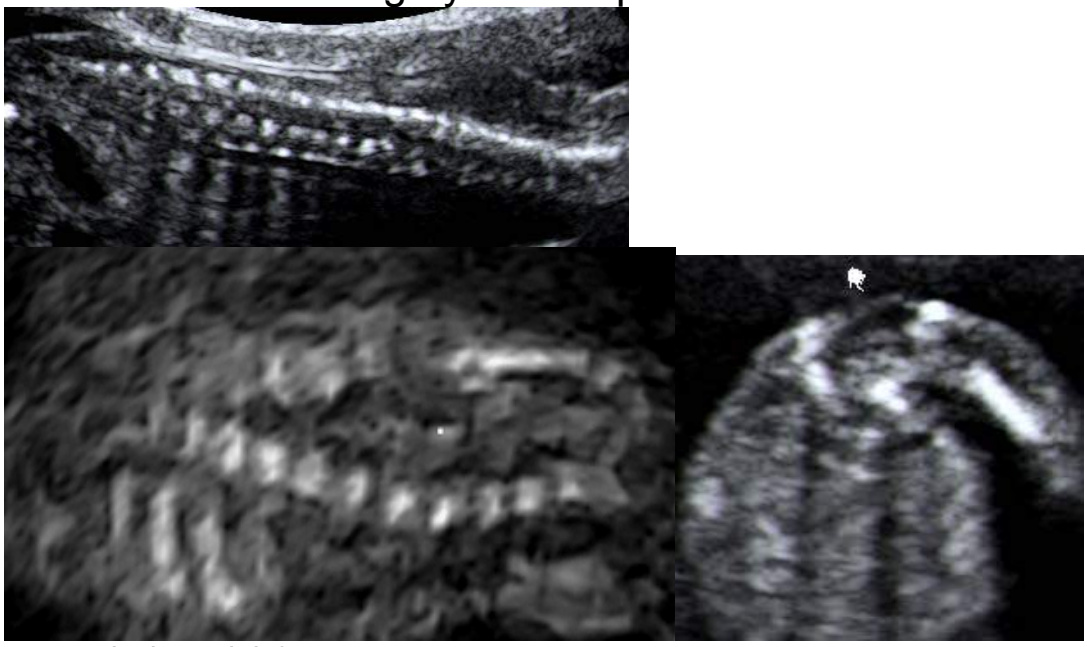
It is essential to establish the integrity of the overlying skin also.



Meningocoele

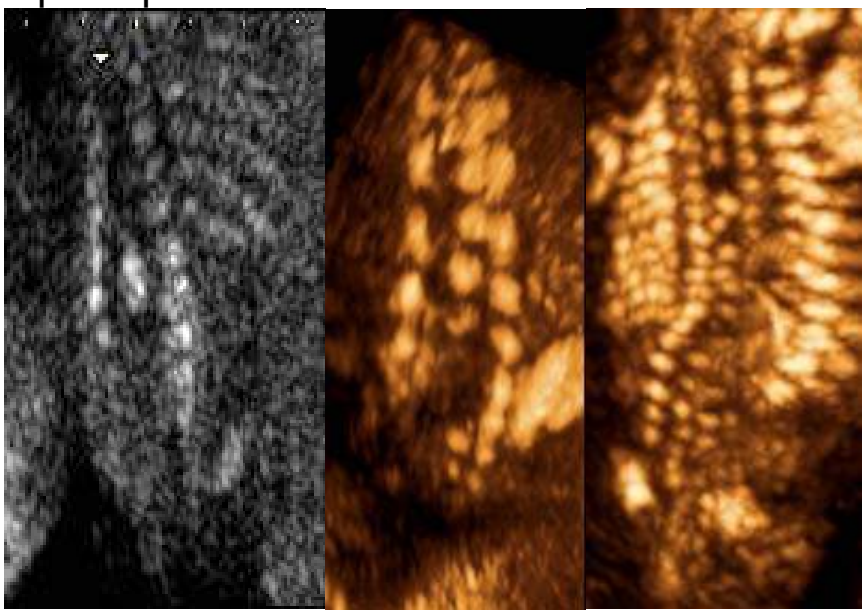
Coronal plane:

This plane will show one, two or three parallel lines, depending on the section at the level of vertebral bodies or transverse process or spinous process. Regular disposition of ossification centers indicate integrity of the spine.

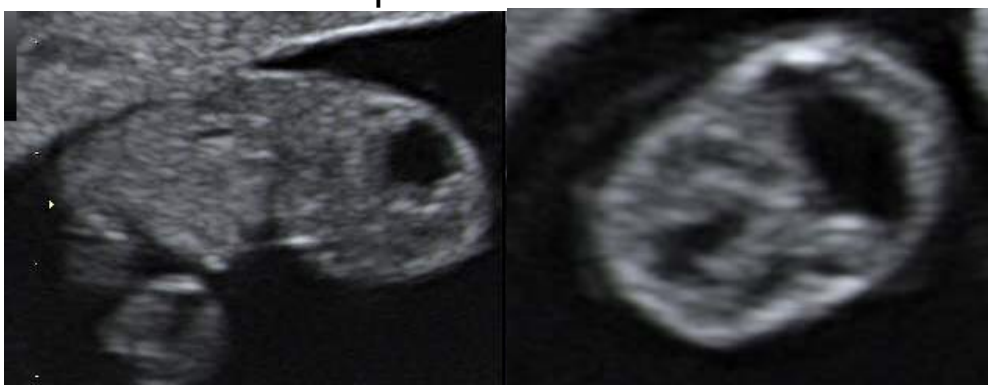


open spinal canal defect

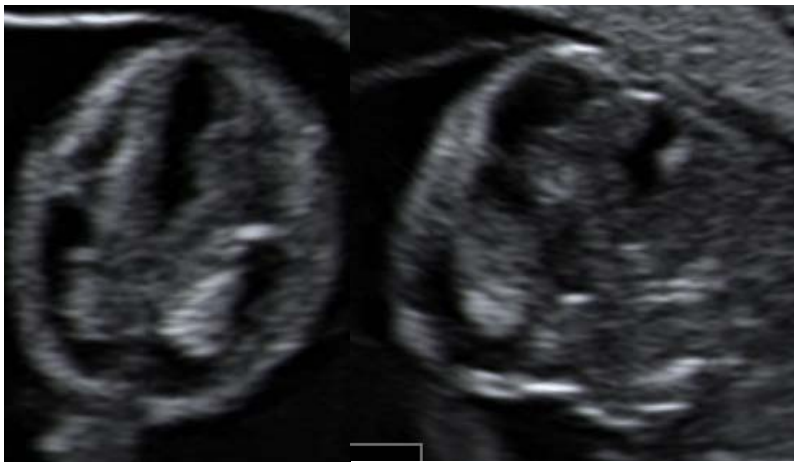
Spinal abnormalities commonly seen are
Hemivertebrae
Open spinal canal
Spine bifida
Platyspondyly
Diastometomyelia
Open spina bifida – often associated with cranial abnormalities



With advanced technologies now it is possible to make **early diagnosis of cranial lesions.**
8weeks:rhombencephalon



9 weeks: lateral ventricles



13 weeks
Decrease in Lateral ventricles & Choroid plexus.
Diencephalons and postr. Fossa structures appear.

Fetuses develop particular pattern at particular gestational age and studying that would allow to assess the neurological development of the fetus.

The inference is that at midgestation if :

- Normal transventricular and transcerebellar plane anatomy
- Normal BPD and HC
- Atria < 10mm
- Cisterna Magna 2 – 10mm

in a low risk pregnancy, no further examination is indicated.

Fetal neurososnography is an extremely informative imaging modality that is relatively inexpensive and definitely noninvasive. Better understanding of developmentand neuroanatomy makes detection of pathologies easier especially by use of coronal and sagittal planes.

Though the facts to be remembered are that

- Anomalies of neuronal proliferation like microcephaly, tumours and cortical malformations may remain undetected as brain continues to develop in the second half of gestation and in neonatal period.

- Moreover some cerebral lesions may also develop due to acquired prenatal and perinatal insults and in these cases antenatal development has been normal.
- And even in expert hands some types of anomalies may be difficult or impossible to diagnose in utero, as they might have developed later during pregnancy or even in early neonatal life.

References:

1. Monteagudo A, Timor Tritsch I. Development of fetal Gyri, Sulci and fissures: a transvaginal sonographic study. Ultrasound Obstet Gynecol 1997;9:422-428
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3. Cordoza et al. Exclusion of fetal ventriculomegalywith a single measurement: the width of the lateral ventricular atrium. Radiology 1988;169:711 -14
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HYDROPS FETALIS

Dr. Purnima Nadkarni

Definition:

Hydrops is defined by abnormal accumulation of serous fluid in skin (edema) and body cavities (pericardial, pleural, or ascitic effusions).

Prevalence:

Hydrops fetalis is found in about 1 per 2,000 births.

Etiology:

Hydrops is a non-specific finding in a wide variety of fetal and maternal disorders, including hematological, chromosomal, cardiovascular, renal, pulmonary, gastrointestinal, hepatic and metabolic abnormalities, congenital infection, neoplasms and malformations of the placenta or umbilical cord. Hydrops is classically divided into immune (due to maternal hemolytic antibodies) and non-immune (due to all other etiologies). With the widespread introduction of immunoprophylaxis and the successful treatment of Rhesus disease by fetal blood transfusions, non-immune causes have become responsible for at least 75% of the cases, and make a greater contribution to perinatal mortality. While in many instances the underlying cause may be determined by maternal antibody and infection screening, fetal ultrasound scanning, including echocardiography and Doppler studies, and fetal blood sampling, quite often the abnormality remains unexplained even after expert post-mortem examination. The most common demonstrable causes of the disorder, in the series conducted by Holzgrave w et al, were cardiac anomalies, followed by chromosomal disorders, congenital malformations, alpha-thalassemia, and the twin-twin transfusion syndrome.(1)

Pathophysiology:

Several hypotheses regarding the pathophysiologic events that lead to fetal hydrops have been suggested. The basic mechanism for the formation of fetal hydrops is an imbalance of interstitial fluid production and the lymphatic return. Fluid accumulation in the fetus can result from congestive heart failure, obstructed lymphatic flow, or decreased plasma osmotic pressure.
Hypoproteinemia and hypoalbuminemia are common in human hydrops, and reduced intravascular oncotic pressure has been speculated to be a primary cause for the disorder(2)

Diagnosis:

The advent of diagnostic ultrasound has now made it possible to evaluate early markers of developing fetal hydrops. Measurement of Nuchal thickness at 8-12 wks & seeing presence of nasal bone is an important ultrasound marker of fetal chromosomal anomaly in the 1st trimester. Fetal cystic hygromas are fluid-filled masses of the neck which arise from abnormal lymphatic development. They are generally anechoic, with scattered septations and the presence of a mildline septum arising from the nuchal ligament. If the lymphatic disorder causing the hygromas is widespread, it may produce fetal hydrops and intrauterine death.(3-4)

Fetal hydrops or fetal anasarca may be identified by the distortion of the normal fetal surface by skin edema. Ascites, pleural effusion, and pericardial effusions also may be identified. The etiologies of fetal hydrops are many and varied.(5-6)

The diagnosis of hydrops based on sonographic findings- subcutaneous oedema, pleural , pericardial effusion, ascites, polyhydramnios or placental thickening is simple.

However determining the underlying cause which may grossly influence fetal & neonatal outcome & thus decision making about intervention, elective delivery or termination of pregnancy can be difficult in several cases even today. According to reviews on non-immune hydrops fetalis from the past ten years, in 14 to 25 percent of the case no clear etiology could be established despite extensive investigation, thus designating them as idiopathic.(7-10) Once the diagnosis of hydrops fetalis is established through ultrasonographic examination including fetal echocardiography should be performed to detect possible congenital malformations, tumors, cardiac defects or dysarrhythmias. Parvovirus B19 and TORCH serology of the mother should be carried out, and if invasive diagnostic methods as cordocentesis or amniocentesis are undertaken, laboratory tests for detection of specific IgM antibodies or viral/parasital DNA from the fetal blood or amniotic fluid should be attempted. Kleihauer-Betke test is important for the exclusion of fetomaternal hemorrhage. Fetal karyotyping is indicated especially if hydrops occurred in the first half of the pregnancy. If parental consanguinity or a history of hydrops fetalis is present, the less frequent causes like lysosomal storage diseases or rare red blood cell disorders should be considered as well. The determination of carrier status of the parents regarding a specific disease is possible in most instances. Follow-up serial ultrasonographic assessments are important to determine the course of hydrops.

In utero diagnosis of hydrops fetalis: ultrasound methods.(11)

The advent of diagnostic ultrasound has now made it possible to evaluate early markers of developing fetal hydrops. Because of these developments, the recognition of these adverse findings can be treated and the disease process reversed. This has been done in cases of Rh disease by intervening intrauterine transfusion, by medically treating the fetal arrhythmias, and/or by treating the basic underlying maternal disorder. The ultrasound examination of the hydropic fetus must include a thorough evaluation for structural anomalies, which can best be accomplished at referral centers where Stage II ultrasound examinations can be carried out. Not all conditions associated with hydrops fetalis are lethal.

Ultrasound Diagnosis:

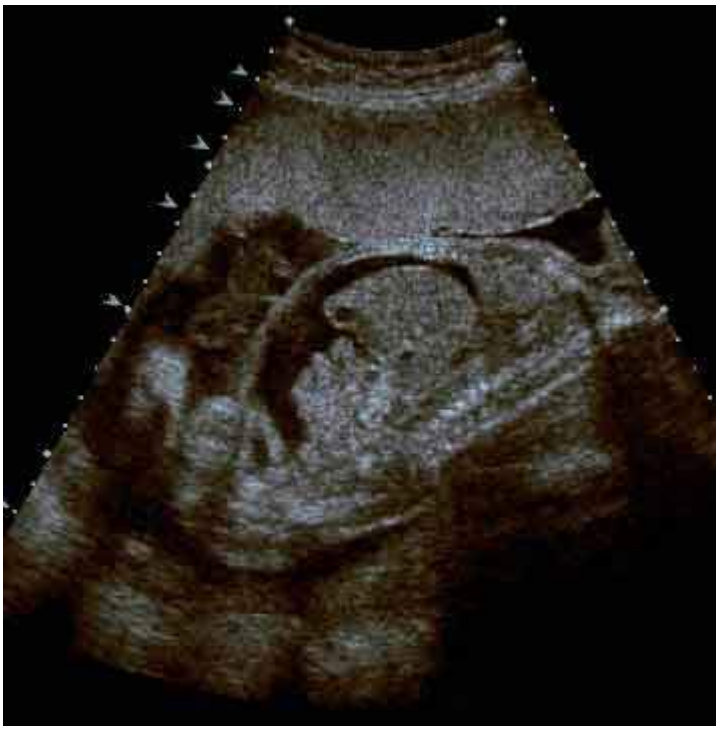

Figure 1 - longitudinal view, abnormal accumulation of serous fluid at the body cavities (pericardial, pleural, or ascietic effusions).

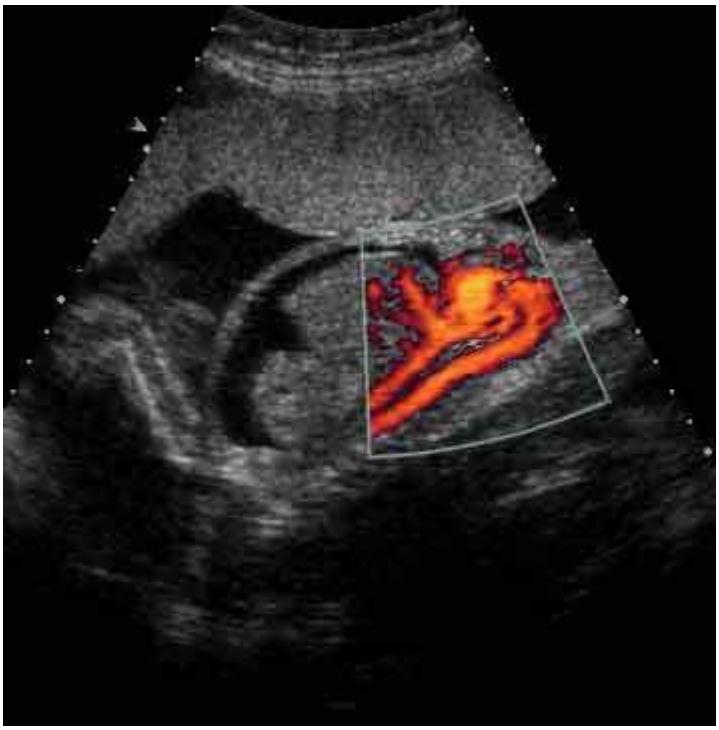

Figure 2 - longitudinal view, abnormal accumulation of serous fluid at the body cavities (pericardial, pleural, or ascietic effusions) & Color doppler energy flow.

ascitic

ascitic

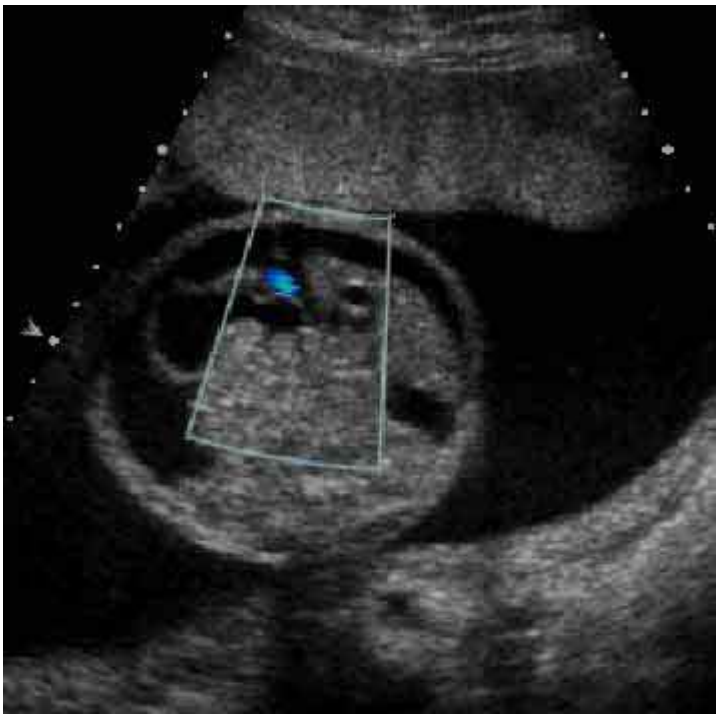

Figure 3 - transverse view, at the stomach and bowel

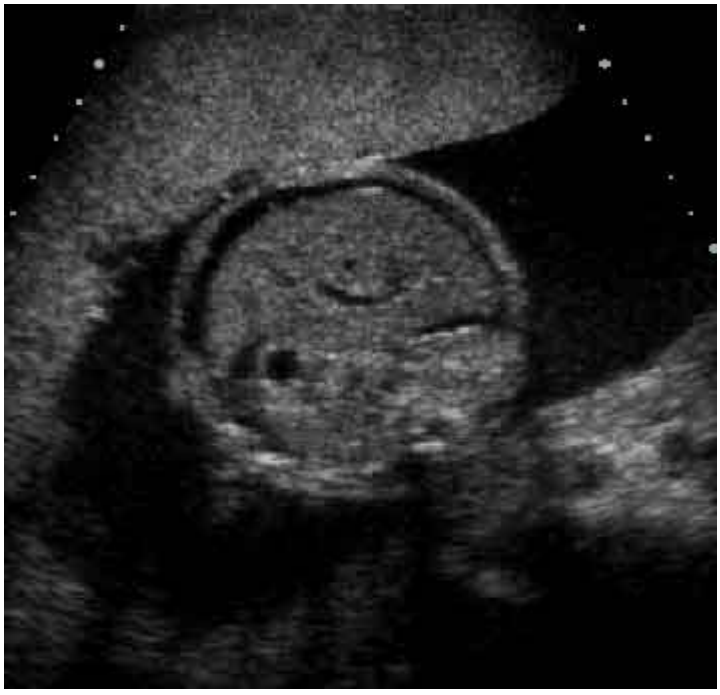

Figure 4 - transverse view, at the stomach level, with

level, with abnormal accumulation of serous fluid at the abdomen or ascietic effusion.	abnormal accumulation of serous fluid at the abdomen or ascietic effusion.
ascitic	**ascitic**

Therapy:

Therapeutic interventions may have two goals, definitive therapy of the underlying cause of hydrops or interventions to ameliorate perinatal outcome by reducing hydrops, avoiding possible complications like lung hypoplasia or improving fetal condition by elevating fetal hemoglobin level in severe fetal anemia. The pharmacological therapy of fetal tachy-arrhythmia with different antiarrhythmic agents or the transplacental antibiotic therapy in intrauterine Toxoplasma and Syphilis infections belong to the first group. The laser ablation of the placental AV fistula to stop twin-to-twin transfusion can also be considered as a curative theraphy. Open fetal surgery was performed for lesions such as congenital cystic adenomatoid malformation, pulmonary sequestration, and sacrococcygeal teratoma in fetuses with NIH.(12-13)

If fetal anemia of any origin is diagnosed as the cause of hydrops fetalis, intrauterine blood transfusions can be attempted; however, not all causes of anemia will be manageable even if the fetus survives until birth. Thoraco-amniotic shunting in severe cases of hydrothorax (14-15) and intrauterine drainage of ascitic fluid have been reported. Transplacental administration of digoxin can be attempted to reduce hydrops.

Prognosis:

The mortality of hydrops generally remains higher than 70 percent, the prognosis being the poorest if fetal hydrops in associated with cardiac structural malformations, thoracic tumors, pleural effusion or diaphragmatic hernia with a perinatal mortality rate of 80 to 100 percent in these cases.(16) Gestational age at the onset of hydrops appears to be inversely correlated with in utero or neonatal mortality.(17)

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Basic Fetal echocardiography

Dr. Jayprakash Shah

Introduction:

Since the first application of sonography in obstetrics by Ian donald, it has gradually become indispensable & routine application. Day by day higher end highly sophisticated machines are available which help us in looking for greater & minute details. It was the cardiac activity which was first evaluated, but for the first time systematic approach to the fetal heart assessment was suggested by Allan in 1980 . Now today basic fetal echocardiography has became the part of basic scan. All the tertiary referral centers are doing basic & advance echocardiography.

Congenital heart disease (CHD) have incidence of approximately 1%. It is a leading cause of infant mortality, with an estimated incidence of about 4–13 per 1000 live births1–3. Between 1950 and 1994, 42% of infant deaths reported to the World Health Organization were attributable to cardiac defects (4). Structural cardiac anomalies were also among the most frequently missed abnormalities by prenatal ultrasonography5,6. Prenatal detection of CHD may improve the pregnancy outcome of fetuses with specific types of cardiac lesions7–11. Half of CHD children are having major cardiac malformations requiring immediate attention while half of them may remain asymptomatic. Among CHD ASD, VSD & PDA are most common

Heart Disease & Aneuploidied:

Post natal CHD are having 3-5% aneuploidy While antenatal diagnosed cardiac malformation are having 30% aneuploidies & that is due to the fact that majority of cardiac malformed fetuses either have abortion or preterm delivery or still born.(Table 1)

Table 1

Prevalence of Chromosomal abnormality in Cardiac malformation Fetal karyotyping & CHD Obs Gyn 1993				
N = 274	Cardiac malformation = 109			
Trisomy 18	Trisomy 21	Trisomy 13	Others	Mandelian transmition
45%	24%	12%	19%	3%

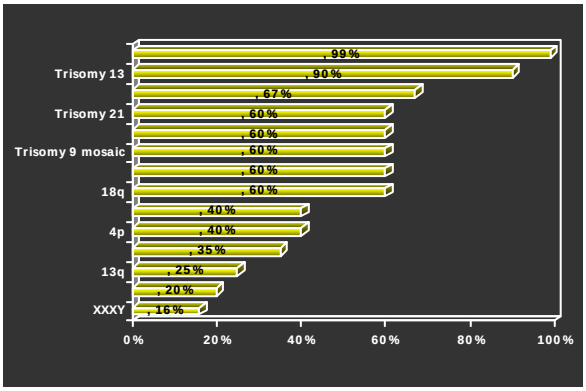
Table 2

Factors affecting Echocardiography 13-14
1. Examiner experience, 2. maternal obesity, 3. transducer frequency, 4. abdominal scars, 5. gestational age, 6. amniotic fluid volume, 7. fetal position

Association with Chromosomal Abnormality:

Cardiac malformation is common soft tissue marker among all confirmed aneuploidies to the tune of 50% on an average. If you look at trisomy 13 CHD is detected in 98% while among confirmed trisomy 21 in nearly 50% of the cases CHD is detected. (Graph 1)

Graph 1



Recurrence risk of CHD: (Table 3)

	Recurrence risk (%)		
Defect	One child affected	Father aftected	Mother affetcted
Aortic Stenosis	2	3	13-18
ASD	2.5	1.5	4-4.5
AV Canal	2	1	14
Coarctation	2	2	4
PDA	3	2.5	3.5-4
Pulmonary Stenosis	2	2	4-6.5
Fallot's Tetralogy	2.5	1.5	2.5
VSD	3	2	6-10
Over all	2%	2	10%

Indications for Fetal echocardiography:

It is suggested that in all the malformation scan which is suggested at 22-24 weeks (18 – 22 weeks by some people) basic fetal echo shall be carried out which include defining situs, four chamber view & outflow tracts.A detailed fetal echocardiogram should be carried out if recognized risk factors raise the likelihood of congenital heart disease beyond what would be expected for a low-risk screening population. Unfortunately, a high proportion of prenatally detectable cases of congenital heart disease occurs in patients without any risk factors or extracardiac anomalies (49). Specific details of this specialized procedure are not within the scope of this article. Common indications are listed in (table 4).

Table 4

Common indications for fetal echocardiography			
Maternal indications		**Fetal indications**	
Family history	First-degree relative of proband	Suspected fetal heart anomaly	
Pre-existing metabolic disease	Diabetes	Abnormal fetal karyotype	
	Phenylketonuria	Major extracardiac anomaly	
Maternal infections	Parvovirus B19	Abnormal nuchal translucency	≥3.5 mm before 14 weeks' Gestation
	Rubella	rhythm disturbances	Persistent tachycardia
	Coxsackie		Persistent irregular heart rhythm
Cardiac teratogen exposure	Retinoids		
	Phenytoin		
	Carbamazepine		
	Lithium carbonate		
	Valproic acid		
Maternal antibodies	Anti-Ro (SSA)		
	Anti-La (SSB)		

Major differences of fetal heart from maternal heart

Anatomic	Physiological
1. Foramen ovale 2. Ductus arteriosus 3. Ductus venosus	1. Fetal ventricles operate at it's peak capacity 2. Fetal heart rate is higher & output cannot be increased by increasing heart rate. 3. Pulmonary flow is negligible 8% of cardiac output Fetal heart work in parallal right heart supplies to lower part of body, left supplies to upper part of body

Technique of Fetal echocardiography:

The main objective of fetal echocardiography is the prenatal diagnosis of congenital heart disease. It is recommended that before starting any malformation scan including echocardiography; one must go through the detail study & understanding of embryology. Cardiac abnormalities encompass a broad spectrum of structural disorders, ranging from a simple communication between two cardiac chambers to an almost complete rearrangement of the connections between the different cardiac segments. This demands a systematic approach to the investigation of the fetal heart. Our approach is to recognize morphology and connections of the three segments of the fetal heart: Atria, Ventricles, and Great vessels.

Steps to Echocardiography:

1. **Establish side of fetus Establish situs**
2. **Check the six connection of heart**

Right side	**Left Side**
SVC & IVC to right atrium	**Pulmonary veins to Left atrium**
Right AV Valve (Tricudpid Valve)	**Left AV valve (Mitral Valve)**
Right Ventricular Out flow tract(RVOT)	**Left Ventricular Out flow tract (LVOT)**

3. **3 Vessel view**
    a. **At Ductal arch level**
    b. **At Aortic arch level**
4. **Aortic arch / Ductal arch view**
5. **Additional views as per requirement**
6. **Color Doppler in Echocardiography**
7. **Spectral Doppler in echocardiography**
8. **M Mode in echocardiography**

Establish side of fetus:

An ideal echocardiographic examination should begin with determination of the position of the head and the spine, establishing the right and left sides of the fetus. (Picrue 1)

Establishing the side of fetus:			
Fetus cephalic (head **Distal** from maternal head)		Fetus Breech (head **Proximal** from maternal head)	
Fetal Spine to left	Fetal Spine to right	Fetal Spine to left	Fetal Spine to right
Fetal Left Side **Distal** – away from probe	Fetal Right Side **Distal** – away from probe	Fetal Left Side **Proximal** – Near from probe	Fetal Right Side **Proximal** – Near from probe

Identification of the visceral situs:

AC view:

The visceral situs can be easily identified in the fetus by using ultrasound in a transverse cross section of the upper abdomen. In this view, the stomach and spleen are normally positioned on the left. The portal sinus, which topographically corresponds to the hilum of the liver, can be seen to the right. Anterior to the spine, the abdominal aorta and inferior vena cava are seen on both sides of the spine. The abdominal aorta is to the left and appears as a round structure, and the inferior vena cava is to the right and is flattened and more anterior (Picture 2 & 3). Recognition of the relative positions of the aorta and inferior vena cava is of special importance in identifying the atrial chambers, since the morphologic right atrium is almost invariably on the same side of the inferior vena cava.5

Four Chamber View:

Normally, the heart is in the left side of the chest with the apex pointing toward the left, the right ventricle and atrium being anterior to the left ventricle and atrium (levocardia). (picture 4 & 4a). Fetal cardiac axis is approximately at 45 ± 20 degree angle to the fetal transverse axis. (Picture 5). Two atrias are seen separated from each other by atrial septum. & in atrial septum, one can see in center, foramen ovale through which both atrias are connected. Flap of foramen ovale can be seen flipping in left atrium. Superior septum secondum & inferior septum primum can be identified very well. Pulmonary veins can be seen entering left atium, two at a time.The two atrial chambers are connected to the ventricular chambers. The atrioventricular junction is characterized by offst crusiate, the more apical insertion of the tricuspid valve than the mitral valve on inter ventricular septum. Other important anatomic details in differentiating the morphologic right and left ventricle is the trabecular pattern. Whereas the left ventricle has a smooth internal surface on ultrasound studies, the right ventricle has a much coarser appearance. Particularly evident is the moderator band of the trabecula septomarginalis, which appears as a thickening of the inter ventricular septum at the level of the apex

Four chamber view can be

- Apical (Apex of heart towards the probe)
- Lateral (Apex of heart towards the lateral aspect)
- Basal (Base of heart towards the probe)

Apical four chamber view allows optimal visualization of the atrioventricular junction and of the relative position of the atrioventricular valves, but the interventricular and interatrial septa are often inadequately imaged. Lateral view (Picture 6)gives us a good visualization of IVS, still in four chamber one is likely to miss membranous VSD for which LVOT is must.

Interventricular values can be nicely seen opening & closing with simultaneous contrations & relaxation of both atria & ventricles. Both atria are of equal size & both ventricles are also of equal size although it seems as if right ventricle is little smaller due to low placed tricuspid valve & moderator band at apex.

Little pericardial fluid can be seen measuring less than 2 mm.

Any abnormal vessel visible between the base of the heart & Descending aorta is suscpicious of Anomalous pulmonary venous return. Coronary sinus can be seen with specific location in modified four chamber view (Picure 7). When suspecting persistence of superior vena cava again four chamber will be useful.

As you see in the (Picture 7a) when reviewing four chamber view you are not seeing the area of great vessel connection. So if basic echocardiography is restricted to four chamber view only you

will miss all anomaly associated with great vessel & few other like small membranous VSD. So it is advisable to extend basic echo to include outflow tract & 3 vessel view.

Dr Devore has nicely demonstarated simple sweep from AC view to 3 vessel view which can be seen in Picture 8 & 9.

Left Ventricular out flow tract:

From four chamber view sweep little cephalad keeping the transverse position will lead to LVOT . Alternatively longitudinal coronal section passing anteriorly from spine also helps us to get LVOT & RVOT.

In LVOT one will see that anterior wall of aorta is in continuation of IVS & posterior wall of aorta is in continuation of leaflet of mitral valve. Aortic valve will be seen opening & closing. (Dot is visible when valve is closed) Small membranous VSD & overriding of aorta will be nicely seen in this view. (Picture 10 & 10a)

Right ventricular Outflow tract:

From LVOT sweep cephalad will show RVOT or alternatively coronal longitudinal section as described in LVOT section can also help. In RVOT one will be able to see main pulmonary artery arising from RV, Pulmonary valve opening & closing, Bifurcation of main pulmonary into right & left pulmonary artery. In the center of this view you will see ascending aorta. Main pulmonary is little more in diameter than ascending aorta. (Picture 11 & 11a) If you compare LVOT & RVOT you will see that both are crossing each other suggestive of normal anatomic relation.

3 Vessel view:

This view can be at 2 level. One (Picture 12 & 12 a) at lower level where we can see Main pulmonary artery as line on the extreme left connecting to descending aorta, in center Ascending aorta is seen as dot & on the extreme right there will be smaller dot of superior vena cava. So popularly this view is also described as line, dot dot view. Just above this view you will see ascending aorta connected to descending arota in center & on the left little larger Main pulmonary & on the right small dot of SVC is seen. In this 3 vessel view if color Doppler is put on, MPA & AA will show same color while SVC will show opposite color.

Aortic arch view & Ductal arch view:

This view are complimentary when on the outflow tracts & 3 vessel view great vessel abnormalities are suspected. Ductal Arch view can be seen easily in longitudinal sagittal section passing through spine. For Aortic arch we need to tilt the transducer to the left. Aortic arch (Picture 13) is acute curve like walking stick with neck vessels arising from it. Ductal arch is slow curve like hockey stick no vessel can be seen from the arch. This view becomes specifically important for diagnosis of aortic abnormality like stenosis or aneurysm.

Superios Vena cava, Inferior vena cava to Right atrium:

This view helps in visualizing the connection of inferior vena cava & superior vena cava to right atrium (Picture 14).

Advanced Fetal Echocardiography

When an abnormal finding is picked up or specific soft marker like increased NT, multiple fetal malformations, certain isolated malformations like duodenal atresia, omphalocele are picked up, advanced & detailed fetal echocardiography is needed. It requires help of high resolution machine with color Doppler, spectral Doppler & M Mode to reach to the specific diagnosis. In spite of all the efforts it is not possible to diagnose fetal cardiac malformations with 100% accuracy & specificity. It is reported that in approximately 85% of the advanced fetal echocardiography one can reach to conclusion. Added facility of STIC may help to improve the diagnostic capacity.

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