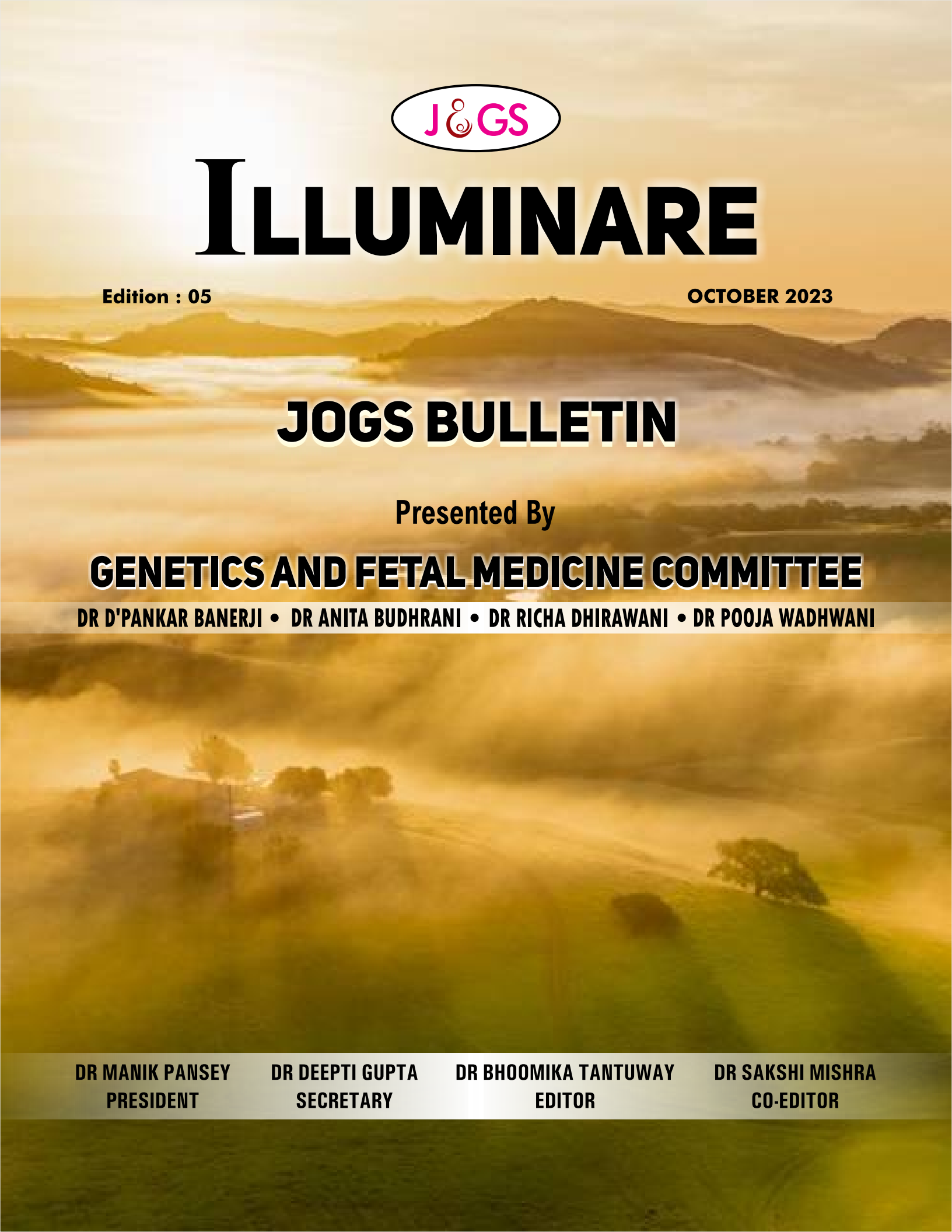
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CTG BASICS AND HOW TO APPROACH INTRAPARTUM TRACINGS

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Despite ongoing advances in maternal and fetal healthcare, over 2 million babies are stillborn worldwide every year. Depending on global region, between 6% and 49% of these stillbirths occur during labour compared to the antepartum period. Many of these stillbirths could be prevented with monitoring during pregnancy and timely obstetric intervention for complications during childbirth. The most common cause of stillbirth globally is intrapartum asphyxia. Since its introduction, Cardiotocography (CTG) or Electronic fetal monitoring is the most widely and frequently used technique for assessing fetal well being antenatally and in labour.

The main objective is to reduce perinatal asphyxia and prevent adverse fetal outcomes like Intrapartum fetal death, Neonatal HIE and Cerebral Palsy. However, only 10% Cerbral Palsy has its origin in labour.

Obtaining a CTG: CTG signal is obtained by Doppler ultrasound where pulsed ultrasound signals are emitted by the transducer place on the maternal abdomen and reflected off the fetal heart back to the transducer. In cases of multiple pregnancy, fetal heart rates should be plotted separately.

Simultaneously Uterine contractions are monitored by using an abdominal pressure transducer which records the frequency and duration of the uterine contractions. The recording is done usually for a minimum of 30 minutes with the paper speed set to 1 cm/m with the mother in the left lateral position, especially in the third trimester.

CTG can be performed in the Antenatal period, where it is called the Non Stress test or in Labour . It can also be done intermittently or continously depending on the indications.

Indications:

Antenatal risk factors:

1. Medical disorders in pregnancy
2. Previous CS births
3. Prolonged rupture of membranes
4. Non cephalic presentations
5. Multiple pregnancy
6. Fetal Growth Restriction
7. Post datism
8. Reduced fetal movements

Intrapartum risk factors:

1. Hyperstimulation of the uterus
2. Maternal pyrexia in labour
3. Presence of meconium
4. Suspected chorioamnionitis
5. Severe hypertension that develops in labour
6. Delay in first or second stage of labour
7. Augmented labour

Parameters and Interpreting CTGs:

When reviewing CTG trace, assess and document

Contractions- 1-2 contractions in 10 minutes

Baseline fetal heart rate- 110-160 b/m

Variability- 5-25 beats/ minute

Presence or absence of accelerations – Transient increase in fetal heart of 15 beats/ minute or more, lasting for 15 seconds and

Presence or absence of decelerations- Transient episodes when the fetal heart rate slows to below the baseline by more than 15 beats/minute with each episode lasting for 15 secs or more.

They are further categorized into early, variable and late in relation to the uterine contraction.

It is important to differentiate between the maternal and fetal heart beats initially and intermittently, especially during the second stage.

Interpretation:

Reassuring: When the baseline heart rate is 110-160 b/m with a variability of >/= 5 with no decelerations and presence of accelerations, the CTG is reassuring.

Non- reassuring: Baseline heart rate-100-109 or 161-180 with variability <5 for>40 but < 90 min withTypical variabledeceleration or Single prolonged decel< 3 min

Abnormal: Baseline heart rate-<100 or >180 with variability <5 for > 90 min withAtypical variable deceleration or late deceleration or Single prolonged decel>3 min

Based on this the CTG can be further classified into

1. **Normal:** All four features reassuring
2. **Suspicious:** One non- assuring feature and reminder reassuring
3. **Pathological:** Two or more non assuring or on or more abnormal

Antepartum CTG: The normal fetal heart rate varies with vagal and sympathetic tone adjustments and therefore varies with gestational age due to maturation of the autonomic nervous system. Hence Antenatal CTG performed before 30 weeks should be interpreted with caution as there is

a. Physiologically normal baseline
b. Reduced variability
c. Reduced amplitude of accelerations
d. Sporadic decelerations

Intrapartum tracings:

Special considerations in labour

1. CTG categorization should be part of the full assessment and should be used dynamically, together with antenatal and intrapartum risk factors.
2. Interpretation of CTG traces in the second stage of labour is more challenging than in the second stage of labour.
3. Onset of hypoxia is more rapid in the active second stage of labour
4. Ensure the fetal heart rate is differentiated from the maternal heart rate at least once every 5 minutes in the second stage of labour as accelerations are more likely to be maternal and decelerations could be pathological.

Approach to Intrapartum tracings:

1. An Admission CTG for 30 minutes can be done to exclude fetal risk and verify contractions.
2. The entire clinical picture should be taken into account when making decisions on further management of labour.
3. The woman and the couple should be informed about the need for intrapartum monitoring and her preferences should be taken into account as well, if possible.
4. If admission CTG normal and pregnancy low risk, CTG surveillance can range from once every 30 minutes to a maximum of every 2 hours with intermittent auscultation.
5. If CTG categorized as suspicious and no other risk factors, consider conservative measures like change of maternal position and avoiding supine position and identifying the cause.

   If there is hypotension, especially due to an epidural anasthesia, do not start Intravenous fluids. Instead, change her to left lateral position.

   If the cause is hypertonus, reduce the oxytocics and use of tocolytic drug can be considered.

   Maternal facial oxygen should not be used as part of a conservative measure, because it can cause more harm

then benefit.

Amnioinfusion should not be offered for intrauterine fetal resuscitation.

6. If CTG categorized as suspicious and there are additional risk factors such as slow progress or meconium, conservative measures may be considered with urgent review by a senior obstetrician and delivery may need to be expedited depending on the stage of labour.
7. If CTG is pathological, or remains pathological after conservative measures, the entire situation should be reviewed again and the CTG should be urgently reviewed by a senior obstetrician. Acute events like cord prolapse, suspected abruption that need immediate intervention should be ruled out and there should be a low threshold to deliver the fetus.
8. In cases of acute bradycardia or single prolonged deceleration for more than 3 minutes, urgent review should be sought and acute events ruled out. Preparations for urgent birth should be made with neonatal support, especially if bradycardia persists for more than 9 minutes.

Additional tools to complement CTG:

Fetal scalp stimulation: Digital scalp stimulation can be done when the CTG trace is suspicious with additional risk factors. If the fetus responds by accelerations, then this is a reassuring feature and the absence of accelerations can indicate fetal compromise.

Fetal blood sampling: Though fetal blood sampling was used earlier but due to limited evidence, there are no clear recommendations regarding its use.

Documentation and storing CTGs:

CTG is a very important evidence of fetal status before and during labour and appropriate documentation and record keeping of the CTG is very essential.

1. The date and time clocks on the CTG should be set correctly
2. The trace should have the patient's name, date of birth (preferably) and hospital number
3. Individual events in labour can be noted in the trace when continuous CTG is done.
4. Every CTG should have documentation of the trace reviewed by having the signature of the reviewer on it as a good practice point.
5. CTG traces can be stored for 25 years, but due to the risk of the ink fading, its preferable to store them electronically.
6. Tracer systems should be available for all CTG traces if stored separately.

Computerised CTG: In spite of all the recommendations, there is considerable variation in the interpretation of the conventional CTG and this affects the reliability of the test.

Computerised fetal heart rate analysis systems or computerised CTG (cCTG) have been developed to allow the automated evaluation of the CTG with the aim of bringing objectivity and reliability to CTG interpretation. It is used antenatally and should not be used in the presence of uterine activity and hence not used in labour. cCTG is an expert assistant and not a diagnostic or prognostic tool. It analyses certain features on the CTG and applies 12 criteria known as Dawes Redman criteria to evaluate the CTG.

A low short term variation (stv) is most commonly associated with fetal growth restriction and chronic hypoxia in the fetus.

Conclusion:

Intrapartum fetal hypoxia is unpredictable and CTG provides a window to act. Usually, IA is adequate for low-risk women.

And EFM is indicated in HRP or labour complications. However, it should be realised that EFM is imprecise tool; and correct interpretation requires adequate training. Secondly, CTG may not show abnormalities for non hypoxic risk factors or complications. Therefore, clinical decisions should be based on a full clinical assessment and CTG should not be used in isolation for decision-making. It only provides information about fetal condition at the time of recording, and it is not a tool.

FETAL GROWTH RESTRICTION (FGR)

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Introduction:

Fetal growth assessment is a critical component of prenatal care that helps monitor the development and well-being of the fetus. Accurate assessment protocols are essential for identifying potential growth abnormalities and making informed decisions regarding pregnancy management. This chapter aims to provide practical information on fetal growth assessment.

Importance of Fetal Growth Assessment: Accurate fetal growth assessment is vital for detecting fetal growth restriction (FGR), macrosomia, and other growth abnormalities that may impact the health of the fetus and the pregnancy outcome. Timely identification of growth disturbances allows healthcare providers to intervene appropriately and provide optimal care for both the mother and the fetus.

Fetal growth is a dynamic process that requires observing the size of the fetus over time. When a fetus's size falls below a predetermined threshold for its gestational age, it is considered small for gestational age (SGA).

Small-for-gestational age for a fetus in utero is an estimated fetal weight that measures < 10th percentile on ultrasound. This diagnosis does not necessarily imply pathologic growth abnormalities, and may simply describe a fetus at the lower end of the normal range[1].

Intrauterine growth restriction refers to a fetus with an estimated fetal weight <10th percentile on ultrasound that, because of a pathologic process, has not attained its biologically determined growth potential[1].

It's important to note that the distinction between SGA and fetal growth restriction (FGR) lies in the associated risks. While an SGA fetus may be small but not necessarily at an increased risk of adverse perinatal outcomes, a fetus with size above the 10th percentile may have FGR and be at a higher risk of adverse perinatal and long-term outcomes 2-6

Globally 13.9 stillbirths per 1000 births have been reported in 2019.7 India accounted for 17.3% of these with SBR of 12.4). 8

According to a study of 872 still births 73.5% are identified as potentially preventable. The most common factors associated with potentially preventable stillbirths are small for gestational age (SGA) (52.8%), maternal hypertension (50.2%), antepartum hemorrhage (31.4%), and death occurring after hospital admission (15.7%). 9

Fetuses with a birth weight below the 10th percentile face an increased risk of stillbirth and perinatal mortality. The highest risk is observed in those with a birth weight below the 3rd percentile.10 Therefore, if a fetus's abdominal circumference (AC) or estimated fetal weight (EFW) falls below the 3rd percentile on the growth charts, it can be considered an isolated criterion to define fetal growth restriction (FGR) at any gestational stage.11

India's Newborn Action Plan has articulated MOHFW vision and goal of "Ending preventable stillbirths to achieve "Single Digit SBR" by 2030, with all the states to individually achieve this target by 2030 with 4.4% average annual reduction rate (ARR) of Still Birth Rate.4

Brief Description of Methods and Indices for Fetal Assessment

In general, FGR is associatedwith Doppler signs suggesting hemodynamic redistributionas a reflection of fetal adaptation to undernutrition/ hypoxia, histological and biochemical signs of placental disease.14

- **Umbilical Artery Doppler:** UA Doppler is a valuable measure for diagnosing and predicting outcomes in fetal growth restriction (FGR). The progression of UA Doppler patterns to absent or reverse end-diastolic flow indicates increased risks. It is associated with adverse perinatal outcomes, but it cannot solely diagnose late-onset FGR.15
- **Middle Cerebral Artery Doppler:** MCA Doppler informs about brain vasodilation, indicating hypoxia. It is useful for identifying adverse outcomes in late-onset FGR, independently of UA Doppler, which may be normal in these cases.15
- **Cerebroplacental Ratio:** CPR combines UA and MCA Doppler to improve diagnostic sensitivity. An abnormal CPR predicts neurobehavioral problems.16 The anterior cerebral artery CPR shows a strong association with adverse outcomes.
- **Ductus Venosus Doppler:** DV Doppler is a strong predictor of short-term fetal death risk in early-onset FGR. Absent or reversed velocities during atrial contraction are associated with perinatal mortality independently of the gestational age at delivery, with a risk ranging from 40 to 100% in early-onset FGR.17 In about 50% of cases, abnormal DV precedes the loss of short-term variability (STV) in computerized cardiotocography (cCTG), and in about 90% of cases it is abnormal 48–72h before the biophysical profile (BPP).
- **Aortic Isthmus Doppler:** AoI Doppler is associated with increased fetal mortality and neurological morbidity in early-onset FGR. It reflects the balance between brain and systemic vascular systems. Reverse AoI may indicate severe placental insufficiency and the need for elective delivery beyond 34 weeks.
- **Cardiotocography:** Traditional fetal heart rate monitoring (CTG) has limitations in predicting adverse outcomes in FGR. Continuous CTG (cCTG) is more sensitive in detecting advanced fetal deterioration and can be used as an acute marker for predicting fetal death.18
- **Growth charts:** Descriptive reference ranges and prescriptive standards of growth serve different purposes. Descriptive reference ranges (e.g. Hadlock) describe the distribution of measurements in a population over time, but only a few of them have high methodological quality. On the other hand, prescriptive standards (e.g. Intergrowth 21) provide expected growth under optimal conditions for healthy women from healthy populations. Comparing observations to healthy-population standards is the usual method in medicine, hence it is important to use prescriptive fetal biometry charts derived from studies with minimal bias and representative populations, ensuring comprehensive pregnancy outcomes and low prevalence of complications. However, the most important part of using the growth chart is to visualise the trajectory of growth irrespective of the type of growth chart used.19

GROWTH ASSESSMENT PROTOCOL

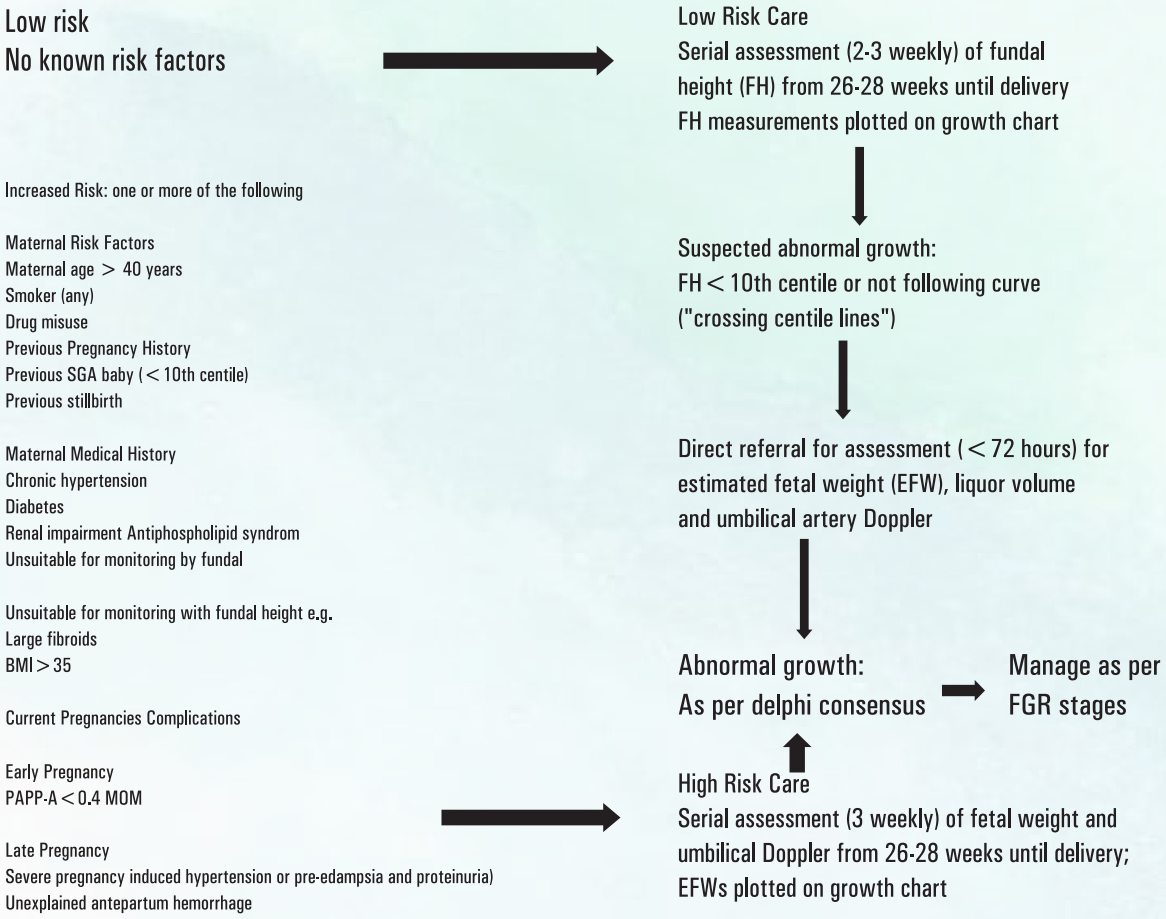
There are number of risk factors that are directly and/or indirectly associated with stillbirth. Hence, the first step would be to stratify them into low and high risk groups. There is a need to identify, investigate and appropriately manage at-risk pregnancies with the aim to avoid unnecessary intervention.20 The emphasis on surveillance would then be according to risk assessment, by standardised fundal height (SFH) and estimated fetal weight (EFW) measurements, serially plotted on growth charts to improve the distinction between constitutional and pathological smallness. Plotting SFH becomes more important in a low resource setting.

STRATIFICATION – LOW/HIGH RISK

- Early pregnancy risk assessment is vital for appropriate care pathway triage.
- Certain conditions such as pre-existing diabetes, diabetes during pregnancy, Hypertension, renal disorders, thrombophilias, pre-eclampaia or multifetal pregnancies are at an inherent increased risk for FGR.
- Previous small-for-gestational age (SGA) indicates a higher likelihood of fetal growth restriction (FGR) and

represents an increased risk.

- Previous stillbirth is considered an increased risk unless placental insufficiency has been excluded by histopathological examination.
- Aspirin is recommended for women with a history or significant risk factors for placental dysfunction.
- Obstetric review is essential for pregnancies at increased risk of early or late onset FGR, and next steps should be determined based on the severity of risk and unit policy.20



FETAL GROWTH SURVEILLANCE

- In the low-risk pathway, regular clinical assessments and fundal height measurements should be conducted every 2-3 weeks from 26-28 weeks until delivery, following unit policy. Measurements should be plotted on the growth chart. If suboptimal growth is suspected, direct referral for a growth scan and Doppler is recommended. Repeat scan for estimated fetal weight (EFW) after 3 weeks is advised for assessing growth trajectory.20
- For cases where fundal height measurement is unreliable due to high BMI (35+) or certain conditions like large fibroids, growth monitoring through serial ultrasound scans is recommended. If resources permit, scans should be done every 3 weeks starting from 28 weeks, as the EFW at this gestational age provides an important baseline for assessing fetal growth in the third trimester.20

- The normal growth rate varies with gestational age and the customised growth potential of each fetus. Growth velocity is highest in the middle of the third trimester. Growth charts provides specific growth rates for different gestational ages and expected birthweights.20
- Slow growth based on serial fundal height measurements is defined as a trajectory that falls below the 10th centile line on the customised chart. This indicates the need for referral for ultrasound biometry.20
- Slow or restricted growth based on serial EFWs is defined as a growth rate between scan measurements that is slower than the 3rd centile line on the growth chart at the same gestational age. A growth rate below this line predicts adverse perinatal outcomes.21 Serial EFW measurements should be at least 2 weeks apart for precision and to minimize the impact of scan error.20

Classification

In a first step, once a small fetus (i.e. EFW <10th centile) has been identified, UtA PI, UA PI, MCA PI and the CPR should be measured in order to classify FGR versus SGA. When either CPR, UtA PI or EFW <p3 is abnormal, the risk of adverse perinatal outcome is increased. Thus, the definition of FGR should include these three parameters.5

Early-Onset Fetal Growth Restriction

Early-onset FGR represents 20–30% of all FGRs.22 Early-onset FGR is highly associated with severe placental insufficiency and with chronic fetal hypoxia. This explains that UA Doppler is abnormal in a high proportion of cases. It often follows a cascade of changes which are reflected in a pattern of Doppler changes that allows to monitor the progression of fetal deterioration and tailor elective delivery. Management is challenging and aims at achieving the best balance between the risks of leaving the fetus in utero versus the complications of prematurity.

Late-Onset Fetal Growth Restriction

Late-onset FGR represents 70–80% of FGR.22 The degree of placental disease is mild, thus UA Doppler is normal in virtually all cases. There is a high association with abnormal CPR values. In addition, advanced brain vasodilation suggesting chronic hypoxia, as reflected by an MCA PI <p5, may occur in 25% of late FGR.15 Advanced signs of fetal deterioration with changes in the DV are virtually never observed. Thus, the cascade of sequential fetal deterioration described above does not occur in late FGR. Late FGR lacks a 'natural history' and may undergo rapid deterioration leading to severe injury or death without observable late-stage signs as in early FGR.

Summary of the main differences between early- and late-onset forms of FGR

Early-onset FGR (1 – 2%)	Late-onset FGR (3 – 5%)
Problem: management	Problem: diagnosis
Placental disease: severe	Placental disease: mild
Hypoxia ++: systemic cardiovascular adaptation	Hypoxia +/–: central cardiovascular adaptation
Immature fetus = higher tolerance to hypoxia = natural history	Mature fetus = lower tolerance to hypoxia = no (or very short) natural history

Definitions for early-and late-onset fetal growth restriction (FGR) in absence of anomalies, based on international Delphi consensus

Early FGR: GA<32 weeks, in absence of congenital anomalies	Late FGR: GA≥32 weeks, in absence of congenital anomalies
AC/EFW<3rd centile or UA-AEDF	AC/EFW<3r centile
Or	Or at least two out of three of the following
1. AC/EFW<10th centile combined with	1. AC/EFW<10th centile
2. UtA-PI>95th centile and/or	2. AC/EFW crossing centiles> quartiles on growth centiles*
3. UA-PI>95th centile	3. CPR<5th centile or UA-PI>95th centile

Stage-based classification and management of FGR

Small-for-Gestational Age:

EFW<10percentile, all dopplers are normal:- Fortnightly doppler and growth assessment is required. Labor induction should be recommended at 40 weeks.5

Stage I: Fetal Growth Restriction (Severe Smallness or Mild Placental Insufficiency).

Either UtA, UA or MCADoppler, or the CPR are abnormal. In the absence of otherabnormalities. Weekly monitoring seems reasonable. Labor induction beyond 37 weeks is acceptable, but the risk of intrapartum fetal distress isincreased.5

Stage II: Fetal Growth Restriction (Severe Placental Insufficiency).

This stage is defined by UA absent-end diastolic velocity (AEDV) or reverse AoI. Monitoring twice a week is recommended. Delivery should be recommended after 34 weeks.5

Stage III: Fetal Growth Restriction (Advanced Fetal Deterioration, Low-Suspicion Signs of Fetal Acidosis).

The stage is defined by reverse absent end diastolic velocity (REDV) or DV PI >95th centile. Monitoring every 24–48h is recommended. Delivery should be recommended by cesarean section after 30 weeks.5

Stage IV: Fetal Growth Restriction (High Suspicion of Fetal Acidosis and High Risk of Fetal Death).

This stage is defined by spontaneous FHR decelerations, reduced STV (<3 ms) in the cCTG, or reverse atrial flow in the DV Doppler. Monitoring every 12–24h until deliveryis recommended. Deliver after 26 weeks by cesarean section at a tertiary carecenter under steroid treatment for lung maturation.5

Note at early gestational ages, and at whatever stage, coexistence of severe PE may distort the natural history and strict fetal monitoring is warranted since fetal deterioration may occur unexpectedly at any time.5

Stage	Pathophysiological correlate	Criteria (any of)	Monitoring*	GA/mode of delivery
I	Severe smallness or mild placental insufficiency	EFW < 3rd centile	Weekly	37 weeks
		CRP < p5		LI
		UA PI > p95		
		MCA PI < p5		
		UtA PI > 95		
II	Severe placental insufficiency	UA AEDV	Biweekly	34 weeks
		Reverse AoI		CS
III	Low-suspicion fetal acidosis	UA REDV	1-2 days	30 weeks
		DV-PI > p95		CS
IV	High-suspicion fetal acidosis	DV reverse a flow	12 h	26 weeks**
		cCTG < 3ms		CS
		FHR decelerations		

MCQ's

1. What is the purpose of fetal growth assessment in prenatal care?

a. To monitor maternal health during pregnancy

b. To identify potential growth abnormalities in the fetus

c. To determine the gender of the fetus

d. To predict the exact birth weight of the fetus

2. What is the main difference between small-for-gestational age (SGA) and fetal growth restriction (FGR)?

a. SGA refers to a fetus with an estimated weight below the 10th percentile, while FGR refers to a fetus below the 3rd percentile.

b. SGA always indicates pathologic growth abnormalities, while FGR may describe a fetus at the lower end of the normal range.

c. SGA is associated with an increased risk of adverse perinatal outcomes, while FGR may not have the same level of risk.

d. SGA is diagnosed based on ultrasound measurements, while FGR is diagnosed based on maternal health indicators.

3. Which Doppler measurement can be used to assess brain vasodilation and indicate hypoxia in late-onset fetal growth restriction?

a. Umbilical Artery Doppler (UA Doppler)

b. Middle Cerebral Artery Doppler (MCA Doppler)

c. Ductus Venosus Doppler (DV Doppler)

d. Aortic Isthmus Doppler (AoI Doppler)

4. What is the recommended management for Stage II fetal growth restriction?

a. Induction of labor beyond 37 weeks

b. Monitoring twice a week and delivery after 34 weeks

c. Weekly monitoring and delivery at 40 weeks

d. Monitoring every 24-48 hours and delivery after 30 weeks by cesarean section

5. What is the significance of stage-based classification in fetal growth restriction?

a. It helps determine the gestational age of the fetus more accurately.

b. It provides a framework for assessing the risk of stillbirth.

c. It helps differentiate between early-onset and late-onset FGR.

d. It guides the choice of Doppler measurements for diagnosis.

Answers:

1. b. To identify potential growth abnormalities in the fetus
2. c. SGA is associated with an increased risk of adverse perinatal outcomes, while FGR may not have the same level of risk.
3. b. Middle Cerebral Artery Doppler (MCA Doppler)
4. b. Monitoring twice a week and delivery after 34 weeks
5. c. It helps differentiate between early-onset and late-onset FGR.

NON-IMMUNE HYDROPS FETALIS (NIHF)

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Definition:

- Hydrops fetalis is a condition in the fetus characterized by an accumulation of fluid, or edema, in at least two fetal compartments.
- NIHF (Non-ImmuneHydropsFetalis) : Non-immune hydrops fetalis refers to hydrops in the absence of maternal circulating red-cell antibodies.

Introduction:

- It generally presents as subcutaneous edema, accompanied by effusions in two or more serous cavities including pericardial or pleural effusions, and ascites. Polyhydramnios or placental thickening (> 6 cm) are often associated.
- When the fluid accumulation is limited to one cavity, for example isolated ascitis or pleural effusion, the situation should be described in terms of the involved site, since this may be helpful in narrowing the differential diagnosis.

Mechanism of NIHF:

- The three primary mechanisms associated with hydrops are intrauterine anemia, intrauterine heart failure, and hypoproteinemia.
- Chromosomal anomalies-blockade of lymphatics lead to edema- (aneuploidy, deletion, duplication, genetic mutation) and skeletal dysplasiamay also be associated with hydrops through a variety of mechanisms.

Causes:

- cardiovascular (21.7%)
- chromosomal (13.4%)
- lymphatic dysplasia (5.7%)
- infections: Parvovirus B19 , CMV (6.7%)
- urinary tract malformations (2.3%)
- extra-thoracic tumours (0.7%)
- gastrointestinal (0.5%)
- Idiopathic (17.8%)
- hematologic (10.4%)
- syndromic (4.4%)
- inborn errors of metabolism (1.1%)
- thoracic (6.0%)
- AV malformation
- twin-to-twin transfusion or placental (5.6%)
- miscellaneous (3.7%)

Investigations: In many instances, the underlying cause may be determined by :

- Maternal antibody and infection screening (TORCH)
- Fetal ultrasoundscanning, including echocardiography
- Doppler studies
- Amniocentesis for karyotyping and array. (fetal blood sampling)

Often the abnormality remains unexplained even after expert postmortem examination.

Fetal therapy in Hydrops Fetalis:

- Fetal anemia: intrauterine blood transfusions.
- Pleural effusions or large pulmonary cyst: insertion of thoracoamniotic shunts.
- Fetal tachyarrhythmias: transplacental or direct fetal administration of antiarrhythmic drugs.
- Teratomas, chorioangiomas, pulmonary sequestration: ultrasound-guided laser coagulation of feeding vessel.
- Recipient fetus in twin-to-twin transfusion syndrome: endoscopic laser coagulation of the communicating placental vessels.

Delivery:

- Timing and method of delivery depend on the cause of hydrops.

Recurrence:

- Fetal defects, infection: no increased risk of recurrence (Except CMV)
- Part of trisomies: 1%.
- Red blood cell isoimmunisation(Rh isoimmunisation): high.
- Metabolic disorders: 25%.

GENETICS : PRENATAL TO PREIMPLANTAION GENTIC TESTING

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A major goal of prenatal care is to help ensure birth of a healthy infant while minimizing risk to the mother. This achievement includes the early identification and management of pregnancies at risk for early reproductive lossand later fetal morbidity and mortality. This journey begins, when a couple plans to have a child. Care should be initiated as early as preconception in select cases and before the end of first trimester, since some screening and diagnostic tests can be performed by then.

Reproductive Genetics is a term, where planning begins before conception. Preconception genetic screening, Preimplantation genetics screening of the embryo reduces the load of genetically abnormal or diseased baby to the gravida.

Prenatal genetic screening and testing begins when the women is pregnant. There is always an effort to identify the fetus with chromosomal abnormality and genetic disorders, if the parents are carrier of some autosomal recessive disorders.

After being pregnant, if there is condition that the fetus is having some genetic problem and will have difficult life postnatally, then taking a decision for termination is a physically and psychologically a challenging task. So earliest is the better.

Reproductive genetics is now inseparably linked to reproductive medicine procedure and it becoming an integral part therof. It has been applied in the diagnosis of factors of infertility and genetic disorder carrier status. This is specifically used for screening assessments before admitting a couple to an in vitro fertilization program for targeted diagnosis of hereditary diseses or defects, or as part of preimplantation genetic procedures.

Ethnicity-based carrier screening for single-gene disorders is an integral part of preconception and prenatal care. The role of ethnicity based carrier screening has expanded over time with advancing technology. Patient and provider should understand the benefits and limitation of their screening option and engage in appropriate pretest and post test counselling. The future management of single-gene disorder is changing, and a time may be approaching when ethnicity based carrier screening will be replaced with expanded carrier screening.

Offering ethnicity based carrier screening is an important component of pre-conception and pre-natal care. Obtaining a family history of the person (and the person's partner, if possible) and establishing ethnicity is essential to determine their inherited risk. Traditionally, ethnic and family background has guided decision making regarding focussed population screening. More recently, with the admixture of marriages, pan-ethnic screening for hundreds of disorders has gained in popularity.

Timing of carrier screening is always an important consideration. The preferred approach is to perform carrier screening before pregnancy.

Interpretation of the carrier screening results and posttest genetic counselling are equally significant to the process. If both parents are determined to be carrier for the same autosomal recessive disorder, there is a 25% risk that their offspring will be affected with the disease. If they have the same mutation, the affected offspring would be a homozygote. If they have different mutations in the same gene, the affected offspring with both mutation would be considered a compound heterozygote. During pregnancy, prenatal diagnosis may be pursued after genetic counselling with amniocentesis or chorionic villus sampling. If the disease is confirmed on prenatal diagnostic testing, parent should be offered additional genetic counselling and discussion of the reproductive option. The parent may also decline prenatal diagnostic testing and undergo expectant management until after birth. Carrier should be encouraged to share the information with their relative. Negative carrier screening results are reassuring but still require genetic counselling. Although the risk of affected offspring is low with negative carrier

screening results, the risk is not zero. A residual risk remains that depends on the test performance in the population of the individual being screened. In addition, parent should understand that prenatal carrier screening does not replace newborn screening, and newborn screening should still be conducted at birth.

Preconception carrier screening allows for reproductive options such as in-vitro fertilization with preimplantation genetic diagnosis in families in which both parents are known carriers (ie,carrier couple). This technique allows the parents to minimize the risk of an affected offspring but may have other risks. Most commonly the diseases/disorders recommended by ACOG and ACMG are hemoblobinopathies like beta thalassemia, alfa thalassemia, sickle cell disorders, Ashkenazi Jewish panel disoders, cystic fibrosis, spinal muscular atrophy, fragile X syndrome etc.

Preimplantation genetic testing encompasses preimplantation genetic screening (PGS) and preimplantation genetic diagnosis (PGD). In todays scenario they are termed as Preimplantation Genetic test for aneuploidy (PGT-A) for selecting an euploid embryo in in- vitro fertilization treatment that may have a higher chances of implantation and resulting in a live birth.

Preimplantation Genetic test for monogenic disease (PGT-M) enables the identification of embryos with disease causing mutations and transfer of unaffected embryo. PGT-M is available for (combinations of) monogeneic disordersfor which the disease causing loci have been unequivocally identified. These loci are nuclear (X-linked, autosomal dominant or recessively inherited) or mitochondrial (maternally inherited) and involve (likely) pathogenic germline genetic variants. More frequently occurring disorders for which PGT-M is currently applied are cystic fibrosis for the autosomal recessive disorders and myotonic dystrophy type 1, neurofibromatosis, Huntington disease and hereditary breast and ovarian cancer (BRCA1/BRCA2mutations) for the autosomal dominant disorders. For the X-linked disorders, PGT is mainly carried out for Duchenne muscular dystrophy, hemophilia, and fragile X syndrome.

PGT-SR are with diagnosis of Structural re-arrangements in chromosomes. Structural chromosomal rearrangements (SRs) are among the major abnormalities accounting for genetic disorders an infertility. They are at increased risk of infertility, miscarriage, stillbirth, and liveborn with mental and/or physical disabilities due to increased risk of producing chromosomally unbalanced gametes. Male carriers often have altered semen parameters and fertility problems such that the incidence of translocation is 6.5 to 9.4 fold higher in infertile males than in newborn population.

Two major noninvasive screening tests include matrnal serum level of specific biochemical markers and cell-free DNA in the maternal circulation. Specific biochemical markers associated with Down Syndrome (trisomy21) and trisomy18, with or without ultrasound markers, may detect other fetal conditions beyond the primary targets. Non invasive prenatal screening using cell free DNA in the maternal circulation to screen for trisomy 21, 18,13 and sex chromosome aneuploidies is another acceptable approach. Most importantly patient should understand the difference between screening and diagnostic tests.

Invasive procedure for prenatal diagnosis of fetal disease is available throughout gestation from conception. Prenatal diagnosis requires direct assessment of fetal tissue. Since 1960s,this has been possible by the aspiration of amniotic fluid. Beginning in the 1980s, chorionic villi have been obtained either transabdominally or trancervically. Other tissues such as skin, muscle, liver and fetal blood have been obtained occasionally for those diagnoses that cannot be accomplished by sampling amniotic fluid or villi.

At the end it is very important to understand the importance of each method stated above as "These procedures have come to be known as a continuum of approaches, rather than distinct entities that are independent of one another"

Source of information :

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THE THREE VESSEL TRACHEA VIEW

Dr. Abhishek Goswami

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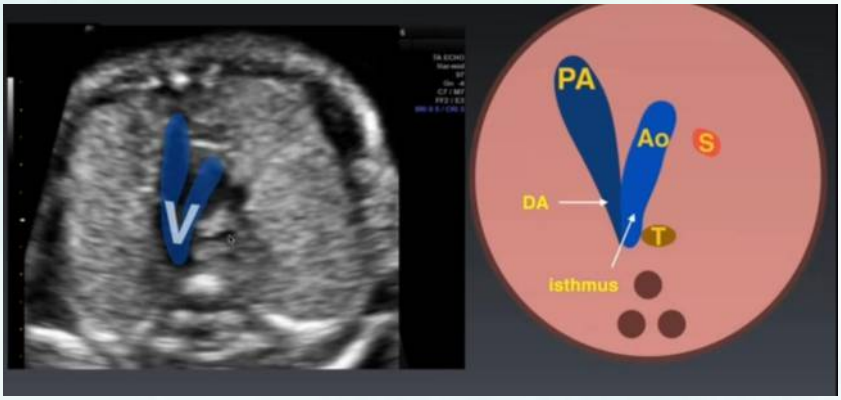
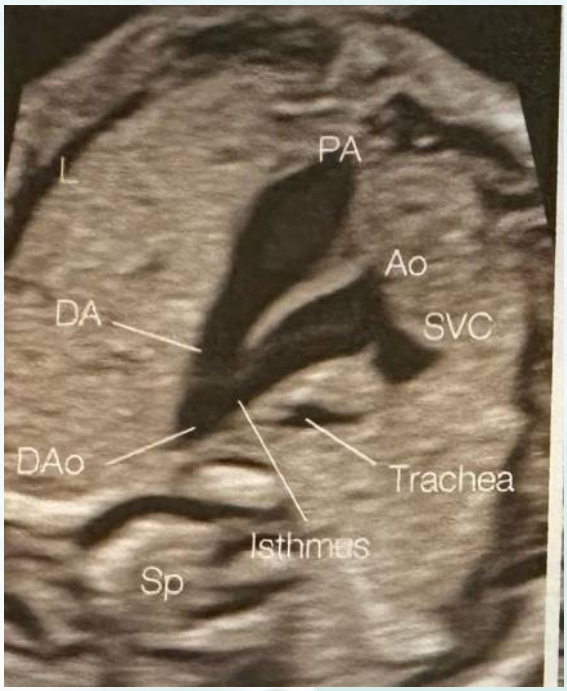
The sonographic assessment of vessels in upper fetal thorax in axial and oblique planes was first described in mid 1980s and early 90s but it's significant came to be known by late 90s .These axial and oblique planes. Basically assess ascending aorta the aortic arch, the main pulmonary artery the Ductal arch And the superior vena cava and also other neighbouring vessel and structures.

Most of the doctors doing obstetric, ultrasound are well versed with four chamber view of heart. But four chamber view of heart can only rule out less than 50% of cardiac anomalies. When we also include 3 vessel, trachea view in our cardiac assessment, we can rule out almost up to about 85% of cardiac abnormalities. Obtaining 3 vessel trachea view might be a little challenging in the initial stages but with practice and correct position of fetal heart one can always get a good three vessel trachea view. There are many cardiac anomalies which have a normal four chambered heart view, but if we don't know how to look for outflow tracts , we are almost certain of missing a lot of anomalies. A good assessment of this view can give you very significant hints about a lot of cardiac abnormalities. Structures seen in three vessel, trachea view.





1. The pulmonary artery.
2. The ductal arch.
3. Aortic arch and isthumus.
4. SVC.what to look for in three vessel trachea view:1. Number of vessels.2. Proportion of vessels.3. Orientation of vessels.4. Relationships to trachea.
5. Colour Doppler.
6. Thymus gland.

Systematic and detailed analysis of this view goes a long way in assessing the fetal heart.

FETAL ECHOCARDIOGRAPHY : IN ROUTINE OBSTETRICS PRACTICE

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Identification of ductal dependent Congenital heart disease (CHD) and Cyanotic CHD before birth by fetal echocardiography has advantageous clinical implications. Ductal-dependent CHD are dependent upon a patent ductus arteriosus (PDA) to supply pulmonary or systemic blood flow or to allow adequate mixing between parallel circulations.

Usually CHD accounts for 0.6 to 1.2 live births however it increases to 6.5 times in those with chromosomal abnormalities and 4 times in those with neural tube defects. Before underestimating this figure, it is important to realise that 30% of neonatal deaths and 10% of deaths in infancy accounts for Congenital lesions. However, still fetal echocardiography is suitable in conditions where risk exceeds 2-3%, and the bridge is generally provided by basic USG scans and extended basic scans.Fetal echocardiography is usually done at around 22 weeks however in high risk mothers and abnormal ductus venous doppler and in presence of TR, 1st trimester scans followed by repeated scans are required.Fetuses with family history of CHD, increased nuchal translucency at 10-12 weeks, diabetic mother, mother with SLE or sjogren syndrome and teratogenic exposure are at high risk of CHD. In fetal echocardiography, apical four chamber view, Outflow tract view, Three vessel view and three vessel tracheal view along with some additional views such as ductal arch view are usually sufficient to diagnose conditions such as Hypoplastic left heart syndrome, Coarctation of aorta, Severe Aortic stenosis, Pulmonary atresia and conotrancal anomalies. However, it is important to remember that screening ultrasound is the most effective tool to identify fetuses that would benefit from fetal echo and with basic scan more than 60% of CHDs can be detected by 4 chamber alone which can be further aided by extended basic scan including outflow tract views thus increasing CHD detection rate upto 90%.

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