"ECHOCARDIOGRAPHIC FEATURES OF CONGENITAL CARDIAC ANOMALIES IN A FETUS -A DESCRIPTIVE STUDY"

By

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Under the guidance of

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Date- ***Dr. Jonna Uday Bhaskar***

Place:

ABBREVIATIONS

CCA – Congenital cardiac anomalies

CHD – Congenital heart diseases

CCD – Congenital cardiac defects

ASD - Atrial septal defect

VSD – Ventricular septal defect

DORV – Double Outlet Right Ventricle

TOF – Tetralogy Of Fallot

ICEF/ IEF – Intracardiac Echogenic Foci

TGA – Transposition of Great Arteries

HRH – Hypoplastic right heart

HLHS – Hypoplastic left heart syndrome

AIUM - American Institute of Ultrasound in Medicine

STIC - Spatio-temporal image correlation

SVC – Superior vena cava

IVC – Inferior vena cava

AAo – Ascending Aorta

DAo – Descending aorta

4CV - Four chambered view

OTV - Outflow tract views

3VTV -Three-vessel & tracheal view

RVOT – Right Ventricular Outflow Tract

LVOT - Left Ventricular Outflow Tract

AoA – Aortic arch

SHF - Secondary heart field

PHF - Primary heart field

USG - Ultrasonography

ABSTRACT

AIMS & OBJECTIVES OF THE STUDY:

To describe the echocardiographic features of congenital cardiac anomalies in a fetus.

SOURCE OF DATA:

All pregnant women (between 16 weeks to 28 weeks of gestation) who came for antenatal scans to our department of radio-diagnosis, B.L.D.E.U's Shri B.M. Patil Medical College Hospital and Research Centre were screened for congenital cardiac anomalies.

MATERIALS AND METHODS:

4693 fetuses between 16-28 weeks of gestational age were assessed for anomalies between August 1, 2013 to July 31, 2015. 33 fetuses found to have cardiac anomalies on routine obstetric scans and were subjected to detailed fetal echocardiographic scans at department of Radio-diagnosis BLDEA's Shri B.M Patil Medical College, Hospital and Research Centre, Bijapur. Consent was taken for each case.

RESULTS:

Out of 4693 subjects studied, 33 (0.7%) had congenital cardiac defects that were randomly selected for screening, along with some of the high-risk cases. The maternal age range was from 16-36 years and the period of gestation ranging from 16 to 28 weeks.

Ventricular septal defect was the most common defect (18.2%) found in our study followed by atrioventricular septal defect and other anomalies. In fetal echocardiography, using four chamber view, outflow tract views, three vessel view, short axis view of ventricles and aortic arch view we have described and identified the congenital cardiac anomalies.

There were extracardiac associations noted with some of the cardiac anomalies.

CONCLUSION:

Fetal echocardiography is an important diagnostic tool in fetuses at risk of heart diseases, which in the hands of trained professional / fetal echocardiologist has high accuracy. Moreover, the imaging modality (ultrasound) is readily available. An early diagnosis provides professionals as well as families precious time to prepare for aneuploidy assessment and also prenatal & postnatal management.

The following review will assert the value of fetal echocardiography, not only as a diagnostic tool but most importantly as a means to improve the outcomes of fetuses affected by cardiac diseases, by serial prognostication and in monitoring fetal therapy in certain cases and ruling out associated multisystem anomalies and aneuploidies.

KEY WORDS: Fetal echocardiography, congenital cardiac anomalies, cardiac views.

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INTRODUCTION

Congenital cardiac disease (CCD) is the commonest form of the congenital abnormalities. Congenital heart disease (CHD) as a gross structural abnormality of the heart or intrathoracic great vessels that is actually or potentially of functional significance.

Approximately, 8/1,000 live births are affected. Over half the deaths from congenital heart disease occur in the first year of life. Delay in diagnosis of a defect is an important contributor to morbidity & mortality in early infancy. Prenatal diagnosis of a defect will therefore optimize the chances of survival of an affected infant.

The cross sectional echocardiogram is becoming an increasingly useful diagnostic tool in the study of congenital heart disease prenatally. Experience of the echocardiogram appearances of cardiac malformation is expanding and becoming increasingly accurate & reliable. This study was undertaken to describe methods of the normal fetal cardiac anatomy, normal cardiac axis, normal cardiac views describing the individual structures of the heart and to describe fetal echocardiographic features of congenital cardiac anomalies using these standard views.

The fetal heart of randomly selected, pregnant women falling under 18 weeks to 28 weeks of gestational age was examined using real-time cross sectional ultrasound in our department at BLDE University's Shri B.M. Patil Medical College, and Hospital & Research Centre.

The study was done to show anatomical details, which could be visualized in the normal heart & the echocardiographic features in congenital cardiac anomalies. It was found that venous, intracardiac & arterial connections of the fetal heart in every case between 16 weeks and term could be positively identified. The gestational range

at which the fetal echocardiographic study was found to be easiest was 18 and 28 weeks.

The use of M-mode imaging provided additional information to the cross sectional study. Although M-mode echocardiography may not be necessary routinely in fetal echocardiographic examinations, it is essential in differentiating some arrhythmias. M-mode imaging is useful in acquiring measurements of chamber size and wall thickness, although not absolutely necessary. M-mode imaging is helpful in evaluating contractility in abnormalities that may affect wall motion, such as cardiomyopathies. Cardiac wall and valve motion could be studied. The structures measured were septal and posterior left ventricular wall thickness, aortic root and left atrial internal dimensions and left and right ventricular internal dimensions. The availability of normal measurement data was of value in the elucidation of structural cardiac abnormality.

Some of the pregnancies with 'high- risk' factors are noted during the study.

4693 gravid women who had their routine obstetric scans were screened for anomalies. Thirty-three, 33, cardiac abnormalities were detected during this study. Some showed associated anomalies.

Fetal echocardiography is a time-consuming procedure that requires many two-dimensional cross-sectional views of the heart and additional Doppler and color-flow investigations; skilled investigators with special experience in fetal cardiology are also needed. Thus, detailed fetal echocardiography is not part of routine prenatal screening programmes but is reserved for cases at high risk & suspected cases of congenital heart disease. Consequently, cardiac abnormalities are among the major malformations that are most frequently missed in prenatal ultrasound examinations,

which is a cause for concern because undetected congenital heart disease increases the risk of early neonatal mortality.

The diagnosis of congenital cardiac anomalies during prenatal age will be fundamental since it helps the counseling and enables parents to be informed and be prepared psychologically for the moment of birth, alternatively it offers them the free choice of termination of pregnancy.

Therefore, fetal echocardiography has been incorporated as a part of routine obstetric second trimester scans in our department.

AIMS AND OBJECTIVES

The aim & objective of this study is to describe echocardiographic features of congenital cardiac anomalies in a fetus.

REVIEW OF LITERATURE

HISTORICAL REVIEW:

In 1971, Mitchell et al. defined congenital heart disease (CHD) as a "gross structural abnormality of the heart or intrathoracic great vessels that is actually or potentially of functional significance". [1]

The first report of visualization of the fetal heart using ultrasonic imaging was that of Garrett and Robinson in 1970. [2] They identified the fetal cardiac activity and interventricular septum using a static B scanner and followed the growth of the cardiac activity during pregnancy. They found the cardiac activity to increase proportionately with the fetal thorax during gestation, the heart occupying about one half of the thoracic cavity. They suggested a ratio of 1.23 for the right to left ventricular chamber measurement in 96 patients.

In 1972 Winsberg studied the fetal heart in 150 pregnancies using a static scanner to locate M-mode beam.[3] He obtained good quality M-mode echocardiograms and measured the left ventricular internal dimension and left ventricular wall thickness during pregnancy in 13 patients. He was attempting to estimate fetal cardiac output from the echocardiogram, a technique that is now considered too unreliable in postnatal life when compared to direct measurements. [4,5]

Further reports on the identification of fetal cardiac anatomy during pregnancy came from Egeblad et al in 1975 [6] and Lee et al in 1977 [7]. The accuracy of moving cardiac structures was limited in Lee's study by use of a static B scanner. Egeblad made first attempt to identify cardiac anatomy using real time ultrasound, but the poor resolution of equipment available at that time did not allow positive identification of

such prominent features as the atrioventricular valves. Also the study was limited to twenty pregnancies between 36 weeks & term.

Baars and Merkus in 1977 used real time cross-sectional imaging to direct an M-mode beam and recorded partial M mode echocardiograms in 43 cases. [8]

Henrion and Aubry in 1979 [9] published the first case of a cardiac abnormality detected prenatally. They were unable to identify the interventricular septum and autopsy confirmed “single ventricle”.

Two papers describing normal fetal cardiac anatomy appeared almost simultaneously in 1980. Sahn et al [10] described four chamber, long axis, short axis and inferior and superior venacaval sections in a study of 71 patients between 20 and 41 weeks gestation. He also estimated total cardiac dimension, right and left ventricular chamber dimensions, pulmonary artery and aortic root size using real frozen frame images for measurement. He derived growth charts for these values against estimated fetal weight. Allan et al [11] described eight echocardiographic sections obtainable of the fetal heart. Two hundred patients between 14 weeks and term were studied echocardiographically and anatomical sectioning of preserved fetal specimens was undertaken to imitate the echocardiographic sections and thereby verify interpretation.

In 1980, Kleinman et al [12] described a case of pulmonary atresia detected at 34 weeks gestation and a univentricular heart at 28 weeks gestation. This was in a study of 180 high-risk pregnancies. Two arrhythmias, namely complete heart block and atrial flutter, were also seen within this study.

In 1982, Lindsey D. Allan et al [13] described the M-mode echocardiogram in the developing human fetus from around the 16th week of gestation until term and the familiarity with the appearance of the normal M-mode echocardiogram and the growth of cardiac structures in the normal fetus which helped in the elucidation of structural cardiac abnormalities in the fetal life and cardiac function in the normal fetus.

In 1982, Kleinman et al studied thirteen fetuses presenting with non- immune fetal hydrops. [14] All were studied by cross sectional and M-mode echocardiography. Ten of the thirteen fetuses were found to have cardiovascular anomalies which were thought to account for the observed intra- uterine cardiac failure. Three fetuses had arrhythmias, seven structural cardiac anomalies.

TECHNICAL ASPECT OF THE STUDY:

PHYSICS OF ULTRASOUND

Ultrasound is now utilized to visualize soft tissue organs of the human body. Some understanding of the physical properties of ultrasound is necessary to appreciate fully the advantages and limitations of this diagnostic procedure.

Sound is transmitted through all media by a series of compressions and rarefactions. The combination of one compression and one rarefaction represents one cycle. The distance represented by one cycle is known is the wavelength (λ). The frequency (f) is defined as the number of cycles per given time, usually in terms of cycles per second, commonly known as Hertz or Hz. The velocity (v) represents the speed with which sound waves travel through a particular medium and is equal to frequency time the wavelength. [15]

$$V = f \times a$$

The velocity at which sound travels through a medium depends on its density and elasticity. Sound travels faster through a dense medium, a solid such as iron bar or bone, than it does through a less dense medium, for example water.

By definition ultrasound is sound with a frequency above the audible range, greater than 20,000 cycles per second (20 kHz). Generally, frequencies in the range 1-20 MHz are used for medical diagnostic purposes. Ultrasound is particularly useful for diagnostic imaging because, in contrast to audible sound, it can easily be directed in a beam and is also reflected by objects of small size. The principle advantage of ultrasound is that it propagates poorly through a gaseous medium. As a result the ultrasound transducer must be in airless contact with the body during the examination of a patient and organs containing air cannot be visualized.

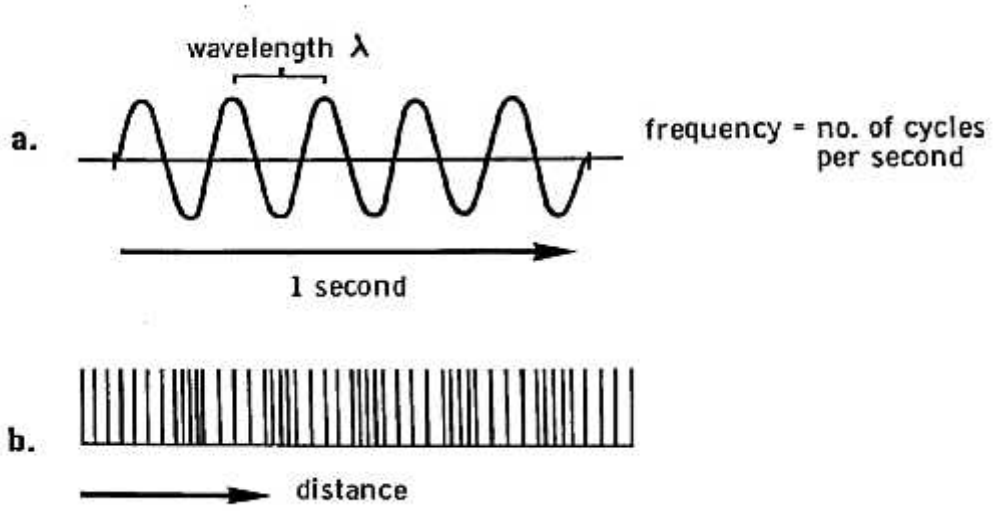


FIG 1: *Sound is transmitted through media in a series of compressions and rarefactions as represented in the above figure. The wavelength (λ) is the distance between each cycle and the frequency of the sound wave is the number of cycles that occur in each unit of time usually expressed in cycles per second. The velocity of sound transmission in a given medium therefore will be the frequency multiplied by the wavelength,* $v = f x$ [a].

An ultrasonic beam obeys the laws of reflection and refraction when passing between media of different acoustic impedance. Acoustic impedance, z, is by definition, the density of the medium, p, times the velocity (v) that ultrasound travels through that medium.

$$\mathbf{Z = p \times v}$$

At an interface between two media a portion of the incident wave is reflected and the remainder refracted. For smooth plane interfaces the angle of incidence equals the angle of reflection, and the angle of refraction is related to the ratio of velocity of ultrasound in the two media. The amount of ultrasound that is reflected depends on how the beam strikes the interface, the angle of incidence and also the difference in acoustic impedance of the media. The closer the angle of incidence is to 90^{o} the greater is the amount of sound reflected. The reflected fraction of the incident energy (R) is also determined by the impedance mismatch between the two media. This is approximately 1% for a fat-muscle interface, 40% for a muscle-bone interface and 99.9% for an air- soft tissue interface.

In biological tissues ultrasound also interacts with many small structures within the parenchyma. The energy is re-radiated in all directions, and is lost from the propagating beam.

In addition ultrasonic energy is absorbed by chemical and molecular mechanisms. The absorption and scattering of the ultrasound beam results in attenuation of the beam's energy as it propagates through the biological media. The extent of the attenuation depends on the frequency of ultrasonic beam and the scattering and the scattering and absorption properties of the tissue, which are usually expressed in db/cm/MHz. **(16)**

The need to balance improved resolution, obtained by using higher frequencies, against reduced penetration of the ultrasonic beam restricts the application of ultrasound to frequency range 1-20MHz, usually 3-5 MHz for general abdominal and cardiac imaging purposes.

IMAGING METHODS:

Special devices, called ultrasound transducers, are required to produce frequency range of 1-20 MHz in their simplest form there are small discs of a ceramic material, generally lead zirconium titanate, across the special property of being able to transmit and receive ultrasound waves. An electrical signal across the face of ceramic crystal will propagate an ultrasound signal; similarly, returning reflected ultrasound signals could be converted to an electric signal. The same transducer is used for transmission and reception of ultrasound. The transducer emits short pulses of ultrasound, rather than a continuous signal, so that during the pauses in transmission the weaker returning echoes can be detected. Typically a pulse of ultrasound lasting 1 microsecond, containing several cycles of ultrasound is transmitted every second. The transducer therefore transmits for 1 sec and ‘listens’ for 900 microseconds.

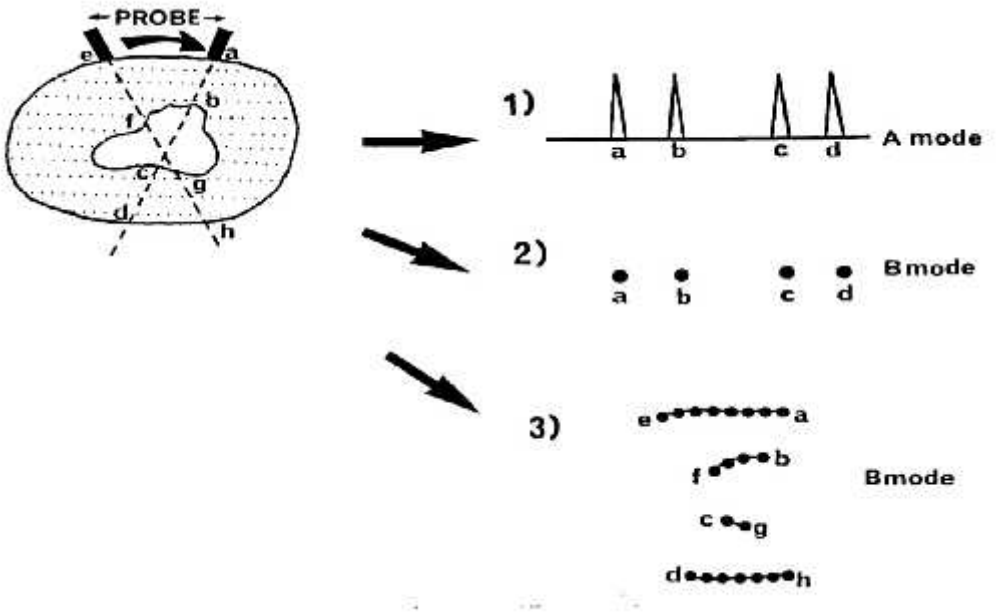


FIG 2: *The ultrasound signal will be reflected at each interface encountered. The amplitude, A-mode, of these echoes can be displayed on an oscilloscope.* ***(FIG 2.1)*** *The base line represents the time interval between pulse transmission and detection of the reflected echoes. When the velocity of ultrasound in soft tissues known the A-scan can be used as an anatomical 'range finder'. Dimensions of organs can be measured by defining the depth of each interface from the transducer. An alternative way of displaying echo amplitude is by the brightness of a spot on a cathode ray tube or television monitor, B – mode* ***(FIG 2.2)****. In a similar way to A-mode, the distance between the transducer and each interface is represented by the time taken for the reflected echoes to return.*

Amplitude is represented by the brightness of each dot on a grey scale display. If a B-mode is displayed on a screen and light sensitive paper is drawn across the display, the motion of structures within the body can be recorded with time. As, for example, the position of the heart valves and cardiac structures is constantly changing

with time, by this means the depth of each interface relative to the transducer can be traced. This demonstrates the pattern of movement with time and is M mode echocardiography. **(*Further way of using b- mode display is seen in* FIGURE 2. 3)**

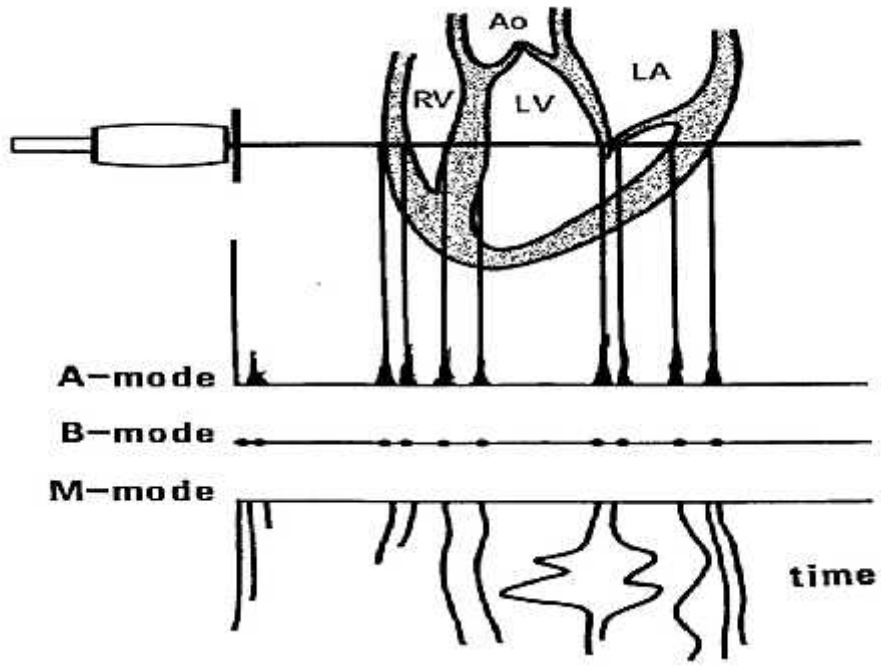


FIG 3: *The transducer is moved across the patient and an image of the shape of interfaces can be built up by a series of B mode dots. This type of B mode display is suitable for static objects, for example, the kidney. The shape of the organ can be delineated on a screen by repeated sweeping of the ultrasound beam across it. This is a cross sectional image. However, this method of producing a cross section is not suitable for cardiac imaging, because a hand manipulated probe could not delineate a section of the heart before movement had occurred. A method of displaying a cross sectional image in 'real time' therefore must be used.*

A real time cross sectional image can be produced in several ways; by using a mechanical sector scanner, a linear array system or a phased array system.

A 3-12 MHz transducer was found to be most suitable for this study.

ANATOMY

EMBRYOLOGY :

Congenital heart disease is the most common severe congenital abnormality found among the live births. Because development of the heart is an interaction of genes, environment & chance, approximately 70-85% of the CHDs have multifactorial causes.

All the major organ systems are formed between the 4th & 8th weeks of development, is called period of organogenesis. It is during this time embryo is most susceptible to factors that interfere with the development.

The cardiovascular system, including the heart, blood vessels, and blood cells, originates from the mesoderm germ layer.[17]

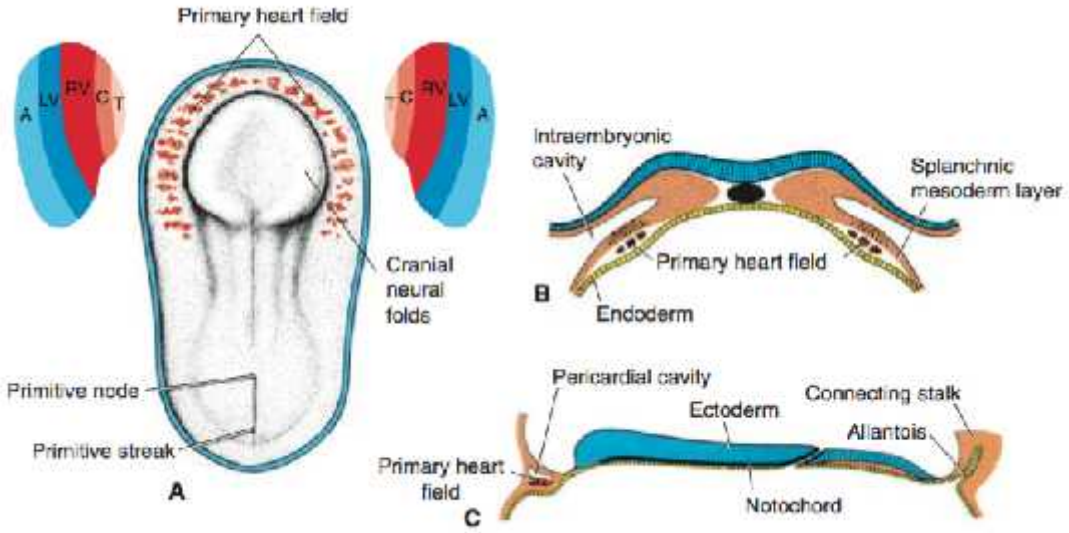


FIG 4: A. Dorsal view of a late presomite embryo (approximately 18 days) after removal of amnion. Progenitor heart cells have migrated and formed the horseshoe shaped primary heart field (PHF) located in the splanchnic layer of lateral plate mesoderm. As they migrated, PHF cells were specified to form the left and right sides of the heart and to form the left and right sides of the heart and to form the atria, left ventricle and part of right ventricle. The remainder of the right ventricle and the

outflow tract consisting of conus cordis and Truncus arteriosus are formed by the secondary heart field (SHF).

B. Transverse section through a similar staged embryo to show the position of the PHF cells in the splanchnic mesoderm layer.

C. Cephalocaudal section through a similar-staged embryo showing the position of the pericardial cavity and PHF.

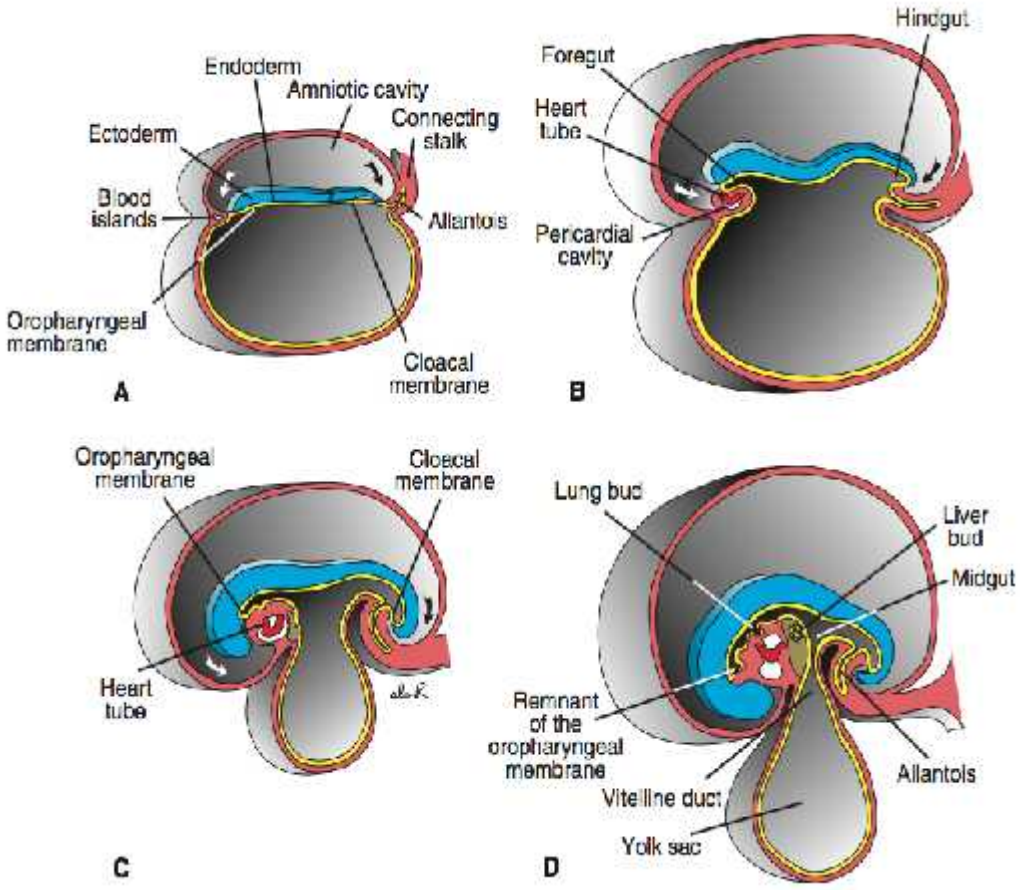


FIG 5: Effects of the rapid growth of the brain on positioning of the heart. Initially, the cardiogenic area and the pericardial cavity are in the front of the oropharyngeal membrane. A. 18 days. B. 20 days. C. 21 days. D. 22 days.

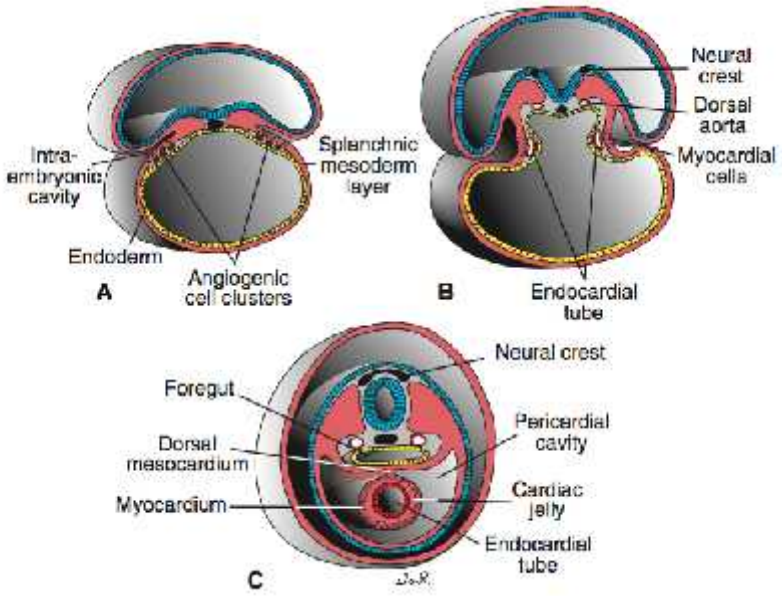


FIG 6: Transverse section through embryo at different stages of development showing formation of a single heart tube from paired primordia. A. Early presomite embryo (17days). B. Late presomite embryo (18 days). C. Eight – somite stage (22days), fusion occurs only in the caudal region of the horseshoe shaped tube. The outflow tract and most of the ventricular region form by expansion and growth of the crescent portion of the horseshoe.

Stage I:

The heart initially consists of a paired tubular structure that by twenty second day of development, where the embryo is 2.5 to 3mm in size, forms a single, slightly bent heart tube.

Heart tube consists of:

1. An inner, endocardial tube
2. And outer myoepicardial tube.

At this stage, the heart tube connects with the developing arch system & with the vitelline & umbilical veins.

Stage II:

It begins with the formation of the atrioventriculobulbar loop.

The cephalic portion of the heart tube bends ventrally & to the right, whereas the caudal portion begins to bend in a dorso-cranial direction & to the left, thus forming a loop. As the heart loop continues to bend, a common atrium is formed & enters the pericardial cavity, carrying along the right & left segments of the sinus venosus. From here the atrioventricular canal forms, which connects the common atrium to the early embryonic ventricles. It is at this time (approximately 28 days) that the contractions are thought to begin in the ventriculobulbar portion of the heart, and the heartbeat is initiated. [17]

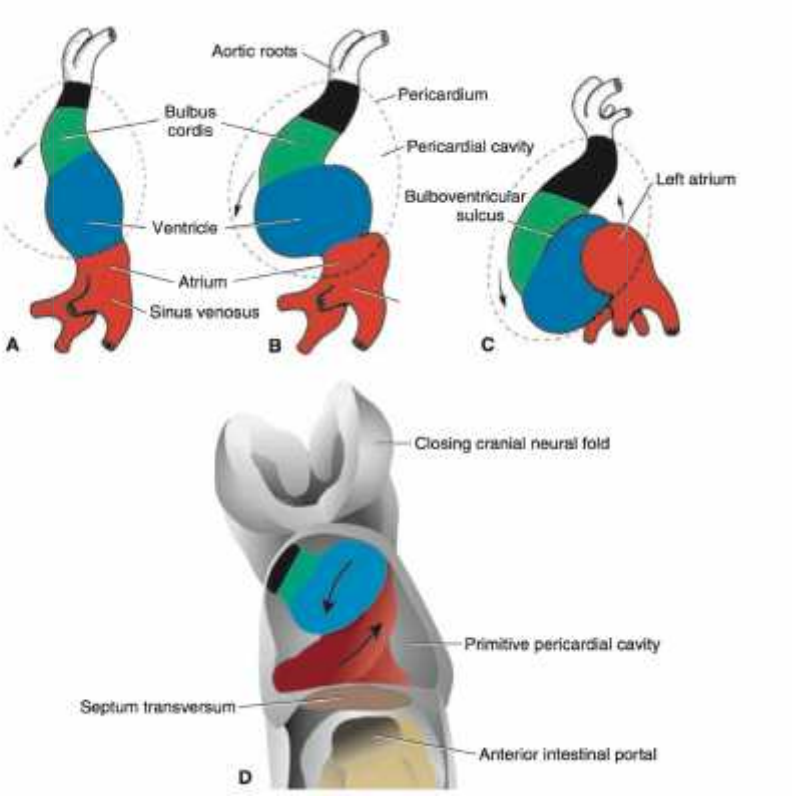


FIG 7: Formation of the cardiac loop A. 22days. B. 23 days. C. 24 days. D. Frontal view of the heart tube undergoing looping in the pericardial cavity. The primitive ventricle is moving ventrally and to the right, while the atrial region is moving dorsally and to the left.

Stage III:

Development of the heart consists of absorption of the bulbus cordis & sinus venosus. At this stage, atrioventricular loop begins to untwist & cardiac septa develop, forming a four-chambered heart. [17]

Formation of the septa within the heart results from the development of endocardial cushion tissue in the atrioventricular canal & the trunco-conal region. This occurs between 27th & 37th days of development, when the embryo is 4-14mm in length.

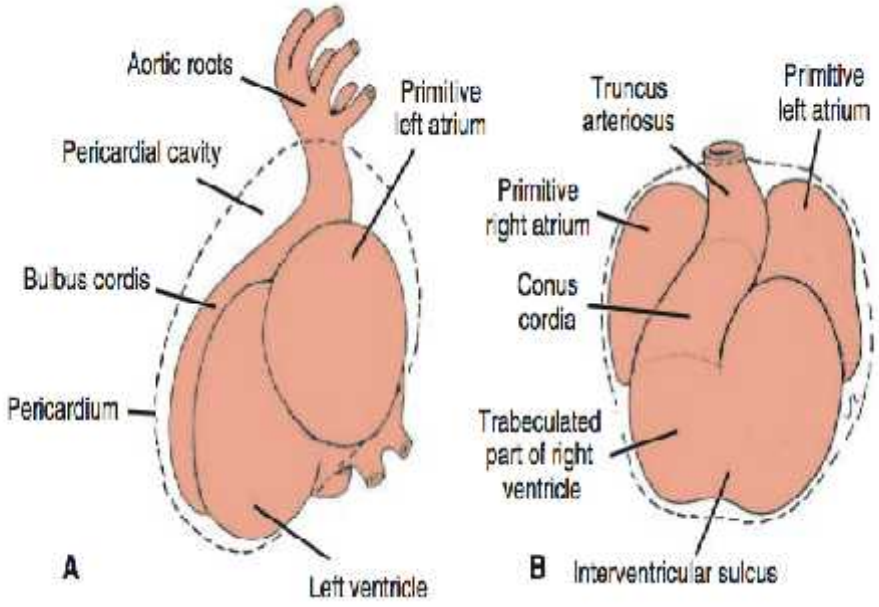


FIG 8: Heart of a 5 mm embryo (28 days). A. viewed from the left. B. Frontal view. The bulbous cordis is divided into the Truncus arteriosus, conus cordis and trabeculated part of the right ventricle.

Formation of atrial and ventricular septa:

In the atrium, the septum primum – a sickle shaped crest descending from the roof of the atrium- does not completely divide the atrium in two, but leaves an open ostium primum for communication between the two chambers. When the ostium primum is obliterated with the endocardial cushion, the ostium secundum forms within the septum primum.

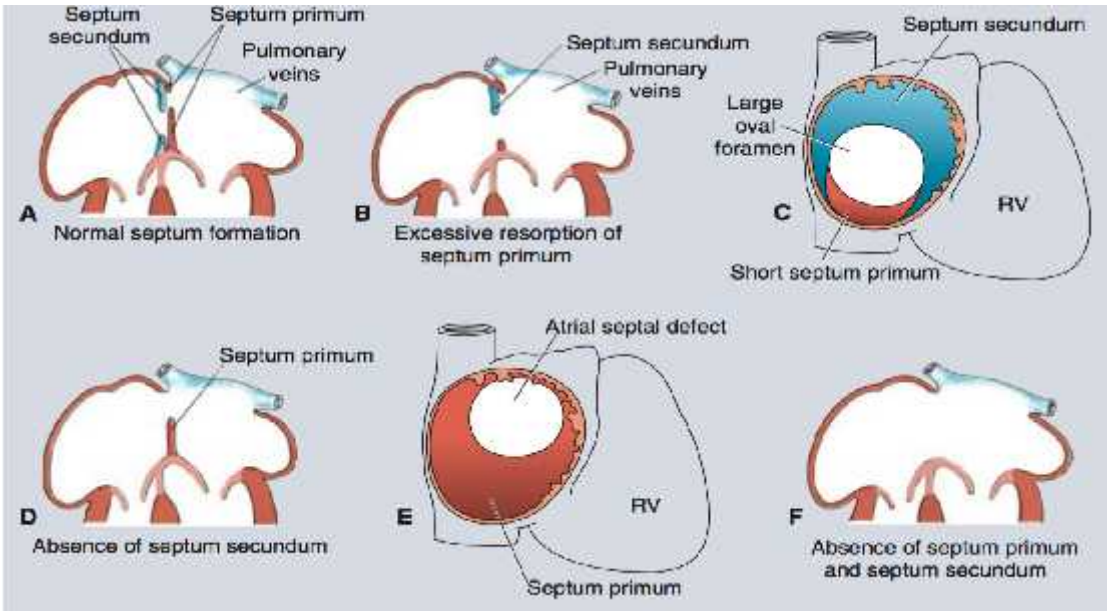


FIG 9: Formation of atrial septa A. Normal atrial septum formation. B,C. ostium secundum defect caused by excessive resorption of the septum primum. D,E. Similar defect caused by failure of development of the septum secundum. F. Common atrium, or cor trilocure biventriculare, resulting from complete failure of the septum primum and septum secundum to form.

Lastly, a septum secundum is formed, but an interatrial opening, the foramen ovale, remains until the birth when pressure in the left atrium increases, causing the two septa to press against each other and close this communication. [17]

Septum formation within the atrioventricular canal occurs when two large endocardial cushions fuse, resulting in right (tricuspid) and left (mitral) atrioventricular orifice. This usually occurs by 33rd day of development.

The interventricular system is formed by the end of seventh week of development. It results from the dilation of the two primitive ventricles (right & left conus swellings), which causes the medial walls to become apposed and fused together. This forms the muscular portion of the interventricular septum. Formation of the membranous portion follows. (17)

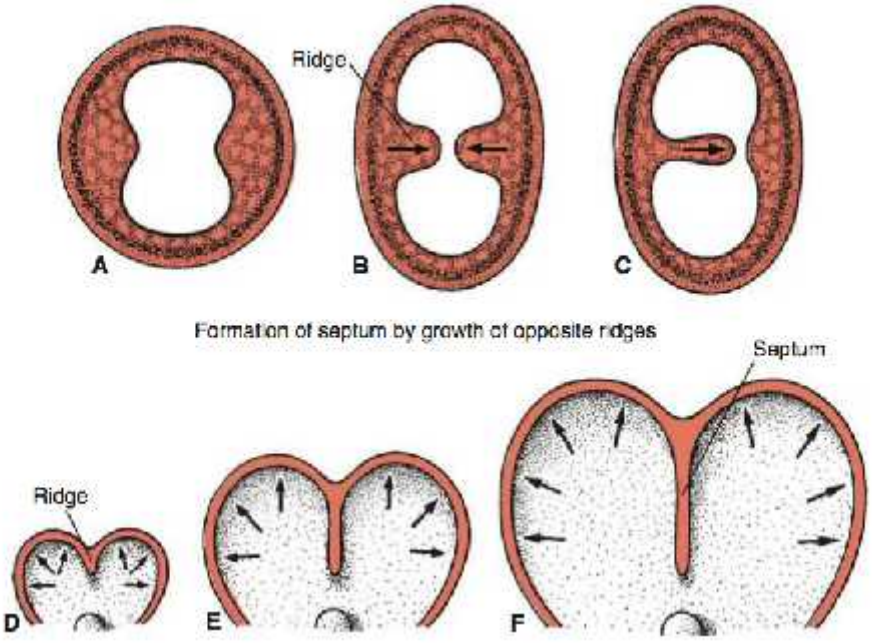


FIG 10: Formation of ventricular septa A,B. septum formation by two actively growing ridges that approach each other until they fuse. C. Septum formed by a single actively growing cell mass. D-F. Septum formation by merging two expanding portions of the wall of the heart. Such a septum never completely separates two cavities.

During eighth (8^{th}) week of the development, the Truncus swellings and twist around each other to form the aorticopulmonary septum. This septum divides the Truncus arteriosus into an aortic & a pulmonary channel. [17]

The cushions of the conus cordis develop simultaneously. These conus cushions unite with the aorticopulmonary septum. After this fusion occurs, the septum divides the conus into an anterolateral portion (the right ventricular outflow tract) and a posteromedial portion (the left ventricular outflow tract).

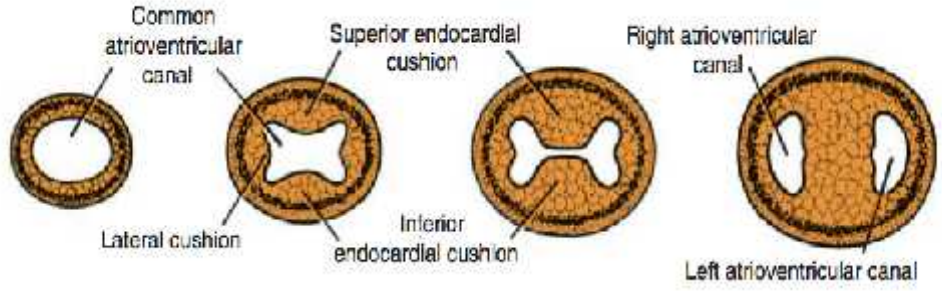


FIG 10: Formation of the septum in the atrioventricular canal. From left to right, days 23, 26, 31 and 35. The initial circular opening widens transversely.

Next, the opening that remained between the two ventricles closes as a result of the conus septum fusing with tissue from the inferior endocardial cushions along the top of the muscular interventricular septum. This becomes the membranous part of the interventricular septum. Between 5 and 7 weeks of development, the semilunar valves (aortic & pulmonary valves) are formed. [17]

Formation of the conducting system of the heart

Initially, the pacemaker for the heart lies in the caudal part of the left cardiac tube. Later, the sinus venosus assumes this function, and as the sinus is incorporated

into the right atrium, pacemaker tissue lies near the opening of the superior vena cava. Thus, the sinuatrial node is formed.

The atrioventricular node and bundle (bundle of His) are derived from two sources: (1) cells in the left wall of the sinus venosus and (2) cells from the atrioventricular canal. Once the sinus venosus is incorporated into the right atrium, these cells lie in their final position at the base of the interatrial septum. [17]

Vascular Development

Blood vessel development occurs by two mechanisms: (1) vasculogenesis in which vessels arise by coalescence of angioblasts and (2) angiogenesis whereby vessels sprout from existing vessels. The major vessels, including the dorsal aorta and cardinal veins, are formed by vasculogenesis. The remainder of the vascular system then forms by angiogenesis. The entire system is patterned by guidance cues involving vascular endothelial growth factor (VEGF) and other growth factors. [17]

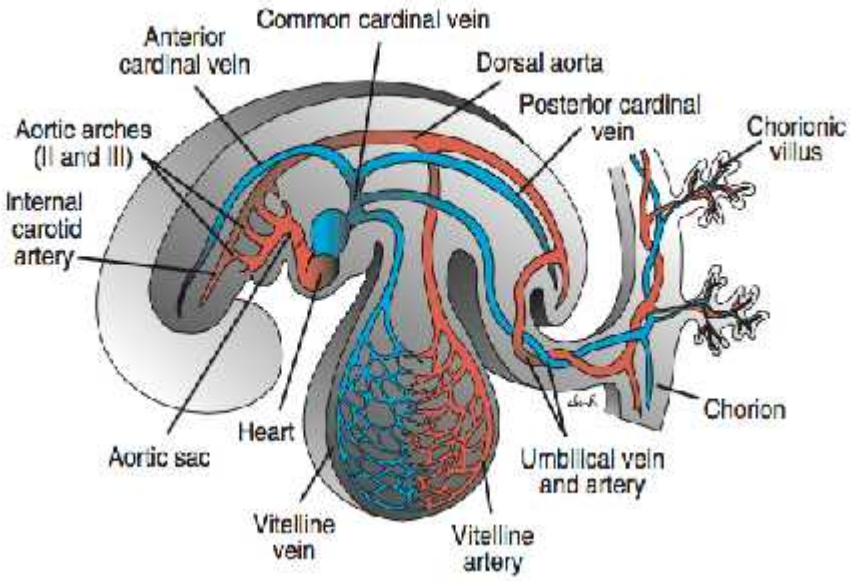


FIG 12: Main components of the venous and arterial systems in a 4mm embryo (end of the fourth week).

Arterial System

Aortic Arches

During the fourth & fifth weeks of development, six pairs of arteries arising from the most distal part of the Truncus arteriosus are formed.

TABLE 1: AORTIC ARCH DERIVATIVES

ARCH	ARTERIAL DERIVATIVE
1	MAXILLARY ARTERIES
2	HYOID & STAPEDIAL ARTERIES
3	COMMON CAROTID AND FIRST PART OF THE INTERNAL CAROTID ARTERIES
4th LEFT SIDE	ARCH OF THE AORTA FROM THE LEFT COMMON CAROTID TO THE LEFT SUBCLAVIAN ARTERIES
RIGHT SIDE	RIGHT SUBCLAVIAN ARTERY (PROXIMAL PORTION)
6th LEFT SIDE	LEFT PULMONARY ARTERY AND DUCTUS ARTERIOSUS
RIGHT SIDE	RIGHT PULMONARY ARTERY

- *Reminder of the internal carotid arteries are derived fro the dorsal aorta; the external carotid arteries sprout from the third aortic arch.*
- *The proximal portion of the aortic arch is derived from the left horn of the aortic sac; the right horn of this sac forms the brachiocephalic artery.*
- *The distal portion of the right subclavian artery as well as the left subclavian artery form from the seventh intersegmental arteries on their respective sides.*

The first pair of arches is formed when the embryo is approximately 1.3mm in length (day 19 to day 20), the second pair is formed when the length of the embryo is 3m (day 20 to day 23). These first and second arch pairs disappear as the third pair is formed when the embryo is approximately 4mm in length (day 45 to day 25). [17]

The dorsal aortas beyond the dorsal ends of the third pair of arches persist as the internal carotid arteries. This third pair of arches form the sterns of the internal carotid arteries. The external carotid arteries arise from these arches, which connect with the aortic sac to form the common carotid arteries.

The fourth pair of arches appear when the embryo reaches 5 to 6mm in length (day 26 to 30). When the embryo is about 14mm long (day 36 to day 42), the dorsal aorta between the subclavian artery and the common dorsal aorta disappears. Thus, the fourth left arch and the common dorsal aorta, and the fourth right arch becomes the proximal part of the right subclavian artery. Also at this time (when the embryo is between 14 and 16mm long), the right limb of the aortic sac elongates to form innominate artery. [17]

The distal segment of both the subclavian arteries and the proximal portion of the left subclavian artery develop from the seventh intersegmental artery. The fifth aortic arch pair never fully develops.

Finally, the right sixth aortic arch, which first appears when embryo is 6 mm in length (approximately day 30), becomes the right pulmonary artery, whereas the left sixth aortic arch persists as the left pulmonary artery and during intrauterine life, as ductus arteriosus.

Coronary arteries

The coronary arteries arise as thickenings of the aortic endothelium when the embryo approaches 10 to 12mm in length (day 35 to day 42). This occurs at the same time that the Truncus arteriosus divides into aortic and pulmonary segments. Both coronary arteries pass to the sides of the Truncus arteriosus, and the anterior descending coronary artery begins to be laid down. Both circumflex arteries have developed by the time embryo is 14mm long (day 42), by the time the length of the embryo is 20mm (day 43 to day 49) all the larger branches have formed. [17]

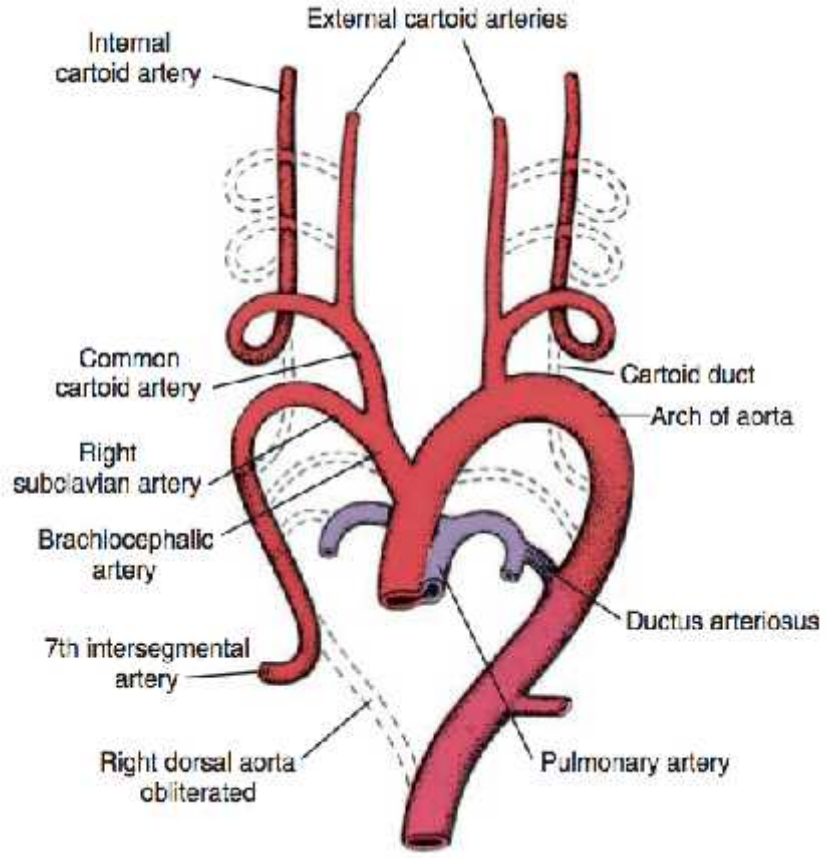


FIG 13: Changes from the original aortic arch system.

Venous system

Pulmonary veins

The pulmonary veins are thought to originate from the two sources: a presplanchnic source consisting of a channel formed from the confluence of the vascular plexus of the lung, which extends to the middle part of the sinus venosus without opening into it, and from the main pulmonary stem, which is an outgrowth of the heart tube.

The common pulmonary vein develops when the embryo is about 5 mm in length (day 29). This common pulmonary vein is eventually absorbed into the left atrium. Next, the right and left pulmonary veins are absorbed, resulting in four septate pulmonary veins entering the left atrium, two superior and two inferior. [17]

Systemic veins

When the embryo reaches 3 mm in length (day 21 to day 23), it contains three series of veins: the umbilical veins draining the chorion, the vitelline veins draining the yolk sac, and the cardinal veins, which are responsible for draining the embryo itself.

As the liver develops (usually when the embryo is 4 to 9 mm in length), the vitelline veins are converted to portal and hepatic veins.

The umbilical veins are rerouted through the hepatic sinusoids. At this time, the right and proximal parts of the left umbilical veins disappear. This results in a passage through hepatic sinusoids, which forms the ductus venosus. [17]

By the time embryo reaches 9 mm in length (day 31 to 35), the circulation proceeds from the placenta, through the umbilical vein, through the ductus venosus, and into the sinus venosus.

When the embryo is between 4 and 22 mm, the differentiation of the cardinal system and the development of the superior and inferior venacavae occur. The precardinal, postcardinal and subcardinal veins develop when the embryo is 4 mm long (day 26 to day 30).

By the time embryo is 11 mm in length (day 36 to day 42), the hepatic portion of the inferior venacava (IVC) develops from the vitelline veins. The supracardinal veins then develop, whereas the postcardinal veins begin to atrophy. When the embryo is 22 mm long (day 50 to day 56), the IVC has fully developed from the vitelline, subcardinal and supracardinal veins. [17]

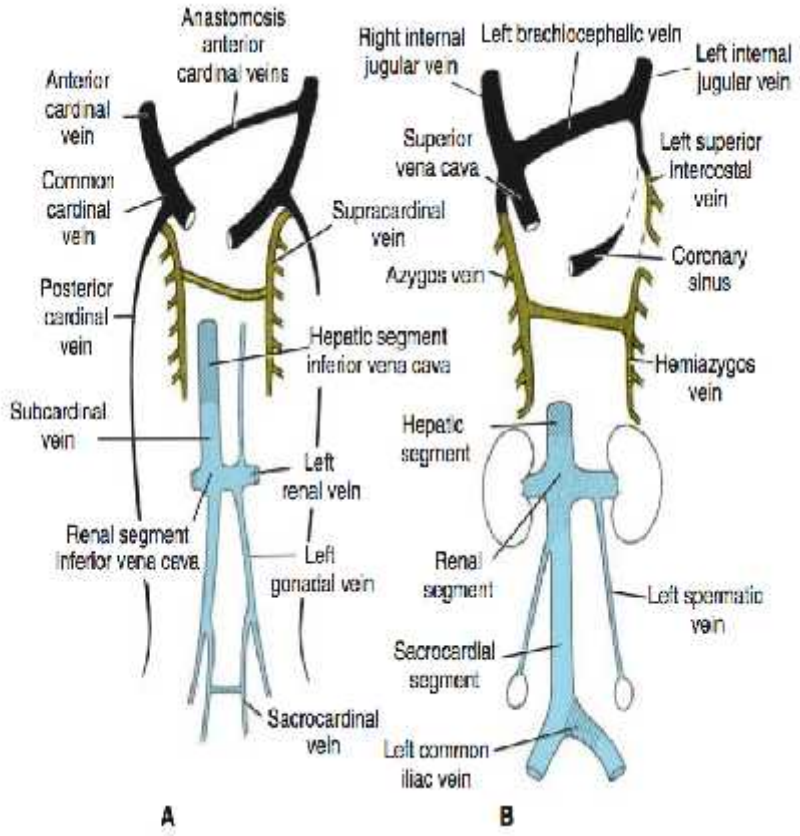


FIG 14: Development of the inferior venacava, azygous vein and superior venacava. A. seventh week.. The anastomosis lies between the sacrocardinals and anterior cardinals. B. the venous system at birth showing the three components of the inferior venacava.

At the embryo length of approximately 20 mm (day 43 to 49), the superior venacava has developed from the precardinal veins, whereas the azygous and hemiazygous form from the supracardinal veins. [17]

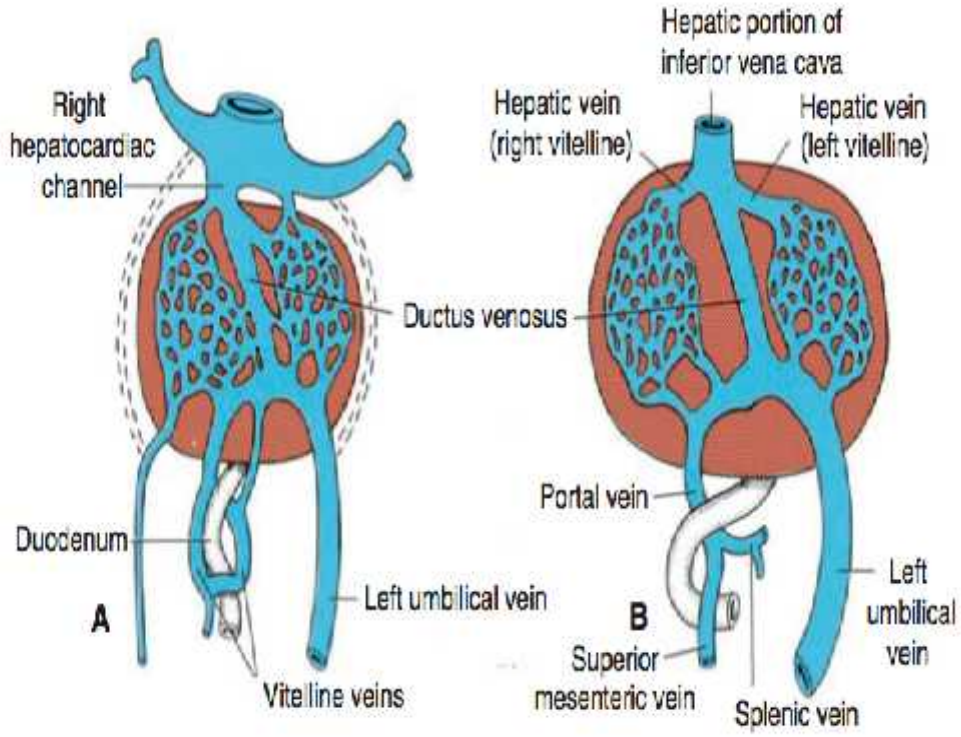


FIG 15: Development of vitelline and umbilical veins in the A second and B third months.

Note: formation of the ductus venosus, portal vein, hepatic portion of the inferior venacava. The splenic and superior mesenteric veins after the portal vein.

FETAL CIRCULATION

Unlike in the adult, fetal gas exchange takes place in the placenta. For oxygenated blood to reach the systemic circulation and deoxygenated blood to return to the placenta for oxygenation, the fetus has several sites of intercommunication: the ductus venosus, the foramen ovale, and the ductus arteriosus. [18]

In utero, oxygenated blood travels from the placenta to the fetus though the umbilical vein at an average rate of 175ml/kg fetal weight per minute. The oxygen saturation of this blood is about 85%. On entering the fetus, the majority of this blood flows through the ductus venosus, by passing the liver and entering the Inferior venacava (IVC). The reminder of this oxygenated blood enters the liver sinusoids & mixes with the portal circulation. The ductus venosus contains a sphincter at the level of umbilical vein, which presumably closes as a result of uterine contractions when venous return is too high, thus preventing sudden overloading of the heart. [18]

The blood that has entered the IVC mixes with the deoxygenated blood returning from the fetal lower limbs. It then enters the right atrium of the fetal heart. The majority of the blood that enters the IVC through the ductus venosus is shunted directly into the left atrium by way of the foramen ovale. This blood mixes with the desaturated blood returning from the fetal head and arms by way of the superior venacava and with the slower moving blood in the IVC coming from the hepatic veins. It then enters into the right ventricle and on into the pulmonary artery.

The resistance in the pulmonary vessels is high in utero; the main portion of the blood that enters the pulmonary artery passes directly through the ductus arteriosus to the descending aorta. The blood that has been shunted from the right atrium into the left atrium mixes with a small amount of desaturated blood, which is returned from the lungs by the pulmonary veins. [18]

FIG: FETAL CIRCULATION

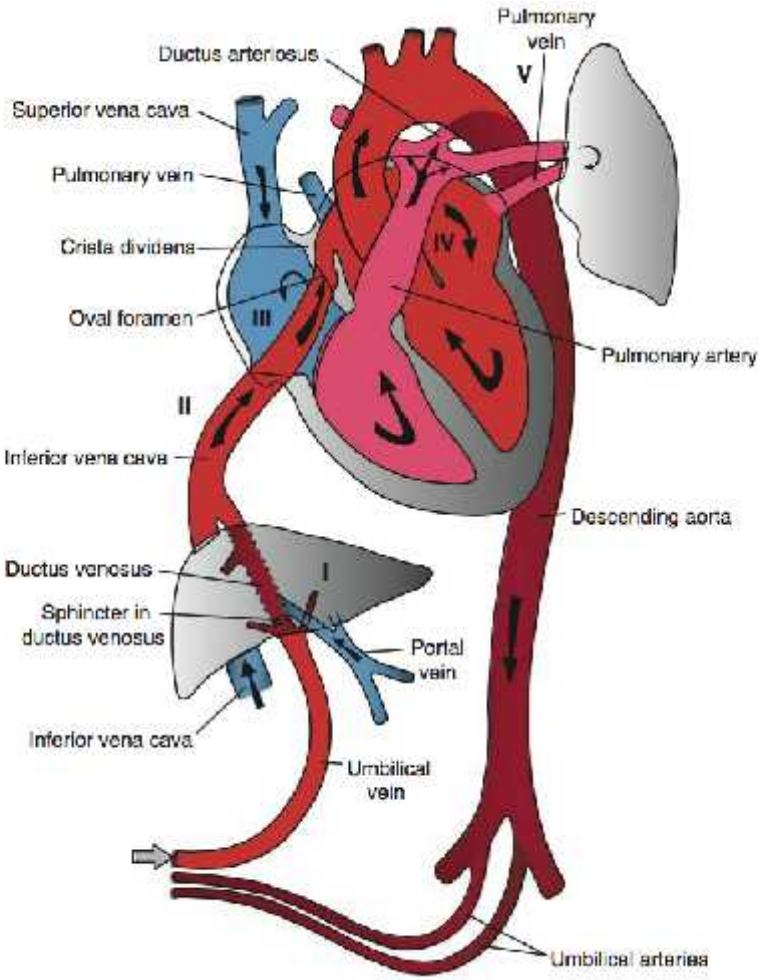


FIG 16: Fetal circulation before birth. Arrows, direction of the blood flow. Note where oxygenated blood mixes with the deoxygenated blood in the liver (I), inferior venacava (II), the right atrium (III), left atrium (IV) and the at the entrance of the ductus arteriosus into the descending aorta (V).

The left atrial blood enters the left ventricle and thus the ascending aorta. Most of the blood supplies the fetal head and upper extremities of the fetus through the vessels arising from the aortic arch. The reminder continues down the descending aorta, mixing with the blood that has been shunted through the ductus arteriosus. From this point, it flows out of the fetus by the way of the two umbilical arteries and

returns to the placenta. This returning blood has oxygen saturation of approximately 58%.

At birth, many changes occur in the cardiovascular system. These changes occur as a result of cessation of placental blood flow and the beginning of pulmonary respiration. [18]

The ductus arteriosus closes almost immediately after birth. Once obliterated the ductus arteriosus forms the ligamentum arteriosum. Closure of ductus arteriosus results in increased pressure in the left atrium. This increase in pressure, combined with a decrease in pressure within the right atrium resulting from the interruption of placental blood flow causes the septum primum and septum secondum to appose each other. This results in a functional closing of the foramen ovale. Complete fusion is usually complete by 1 year of age.

The umbilical arteries close immediately after birth closure occurs because of contraction of the smooth musculature within their walls and is thought to be caused by thermal & mechanical stimuli and a change in oxygen tension. The proximal portions of the umbilical arteries remains open and become the superior vesicle arteries. [18]

Closure of the umbilical vein and ductus venosus occurs shortly after closure of the umbilical arteries. The umbilical vein forms the ligamentum teres in the lower segment of the falciform ligament within the liver. The ductus venosus, which courses from the ligamentum teres to the inferior vena cava, is also obliterated and forms the ligamentum venosum.

NORMAL FETAL CARDIAC ANATOMY

The fetal heart is similar to that of the adult, with several anatomic and physiologic differences. The long axis of the fetal heart is perpendicular to the body, such that a transverse section through the fetal thorax demonstrates the four cardiac chambers in a single view. The adult heart, in contrast, is obliquely oriented with its long axis along a line between the left hip and the right shoulder. The four-chamber view is important because 10% to 96% of structural anomalies are detectable on this view. [19]

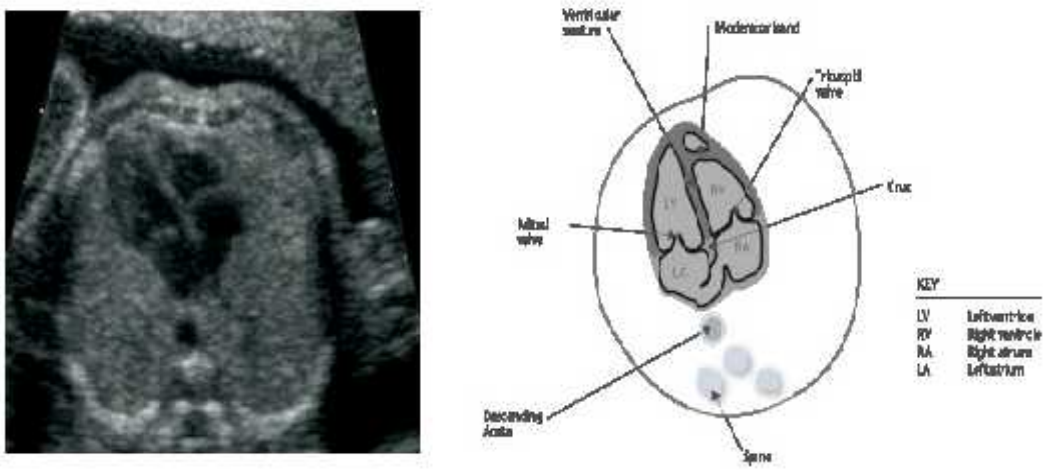


FIG 17: *Normal Fetal Cardiac Anatomy*

NORMAL CARDIAC AXIS

In cross sectional transverse view of the fetal chest, the correct orientation of fetal heart is with apex pointing to the left & bulk of the heart occupying the left side of the chest. This is levocardia. In mesocardia the heart is central with the apex pointing anteriorly. In dextrocardia the apex is directed right- ward, and the heart is primarily in the right chest. [19]

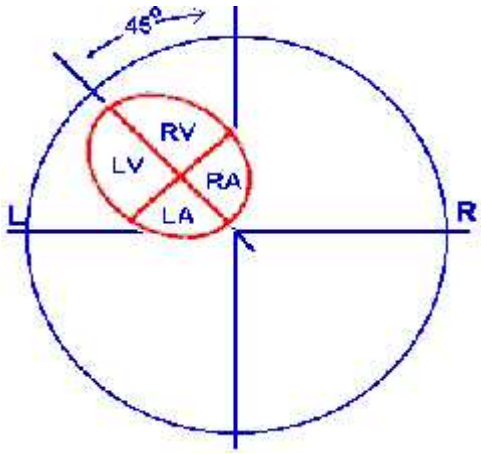


FIG 18: *Normal Cardiac Axis*

A line traversing the interventricular septum will fall between 25 & 65 degrees (normally 45^{o} +/- 20^{o}) leftward from a line extending between the spine & mid anterior chest wall. In this precise orientation, the left atrium is located closest to the fetal spine & right ventricle will be nearest to the anterior chest wall. This abnormality must be distinguished from dextroposition, in which the heart maintains a normal axis but is displaced to the right by an external process, such as a left chest mass or pleural effusion. Abnormal cardiac axis is associated with a 50% mortality and abnormal cardiac position with 81% mortality. [19]

FETAL ECHOCARDIOGRAPHY - SONOGRAPHIC TECHNIQUE

Before scanning the fetal heart, it is important to determine whether abdominal situs is normal because congenital heart diseases are frequently associated with abnormal abdominal situs. On the basis of fetal position, several planes can exist.

In a fetus with breech presentation, the left side of the fetus should be proximal to the transducer when the fetal occiput is on the left side of the mother. When the fetal occiput is on right side of the mother, the left side of the fetus should be distal to the transducer. When the fetus is positioned face up, its left side appears

on the right side of the screen, and when the fetus is lying face down, its left side appears on the same side of the screen. [20]

In a fetus with vertex presentation, determination of abdominal situs is reversed. While it can be difficult to determine abdominal situs when the fetus is in the transverse position, the right–hand rule of thumb can reliably determine fetal situs. With this simple approach, the palm of the right hand corresponds to the fetal abdomen, the dorsal side of the forearm to the fetal back, and the fist to the fetal head. The direction of the thumb always corresponds to the left side of the fetus regardless of the fetal position. [20]

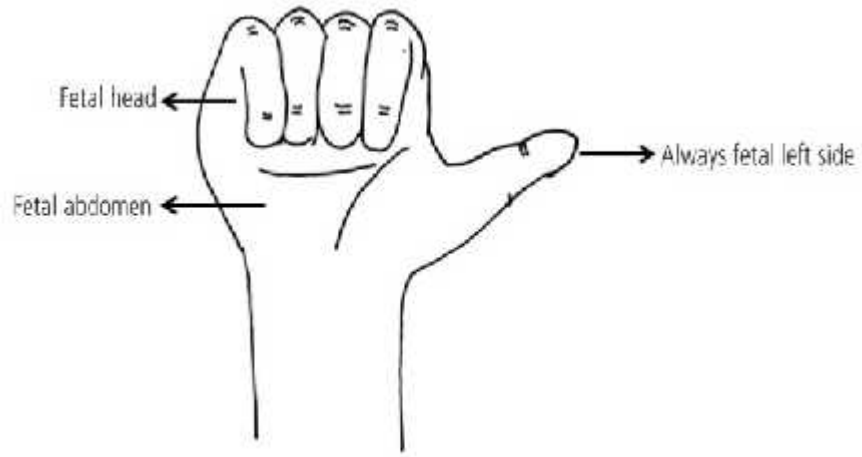


FIG 19: *Determination Of Abdominal Situs: Right-Hand Rule Of Thumb*

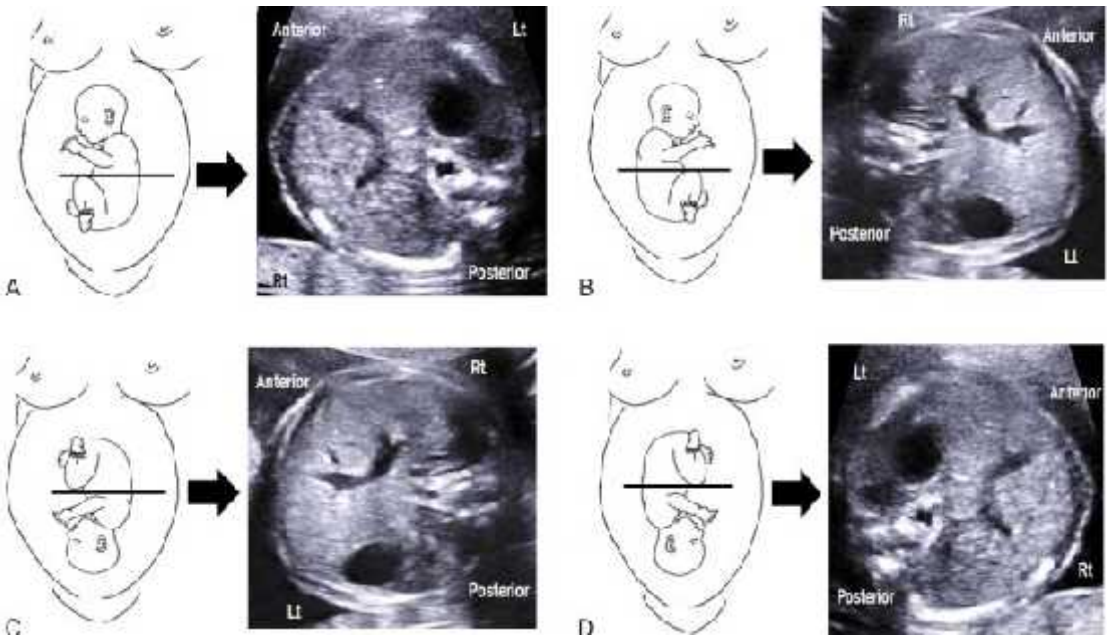


FIG 20: *Schematic Images And Transverse Abdominal Views Of Fetuses With Breech And Vertex Presentations*

After determining the abdominal situs, the fetal heart is then focused on, looking for particular structures. A number of structures are considered essential to identify fetal cardiac normality. These are listed below.

1. Two atria, and the mechanism of foramen ovale.
2. Two ventricles, with intact ventricular septum.
3. Two great arteries.
4. Inferior venacava draining into the anterior atrium.
5. Superior venacava draining into the anterior atrium.
6. Pulmonary veins draining into the posterio atrium.
7. The foramen ovale flap seen in the posterior atrium.
8. Differential ventricular trabeculation.
9. Differential atrioventricular insertion.
10. A complete muscular infundibulum in the anterior ventricle supporting the pulmonary valve.
11. Arterial - atrioventricular valve continuity in the posterior ventricular chamber.
12. The right ventricular outflow connected via the pulmonary artery and ductus arteriosus to the descending aorta.
13. The left ventricular outflow tract connected via the arch of the aorta to the descending aorta.

To identify all these above-mentioned structures in a systematic manner, different views have been used. The views are mentioned below.

1. Four chambered view (4CV)
2. Out flow tract views (OTVs)
    a. Left ventricular outflow tract (LVOT)
    b. Right ventricular outflow tract (RVOT)
3. Short axis view of the ventricles.
4. Short axis view of the great vessels.
5. View of the aortic arch. Aortic arch (AoA) view
6. View of the ductal arch.
7. View of Venoatrial connections (SVC & IVC) (Hammock view).
8. Three-vessel view. (3VV)

Between 18 and 28 weeks gestation, it was possible in every case to identify all the normal structures with the exception of pulmonary veins. Pulmonary veins in the fetus are very small. According to studies**,** even in the last trimester positive identification of pulmonary veins was only possible for the half cases they have studied. It is not advisable to examine the patient supine for longer than 30 minutes, partly because of the discomfort but also because pressure of uterine contents on the materna inferior venacava can cause hypotension. But during the 18-28 week gestational age range, in majority of cases, a complete real-time and complete M-mode echocardiogram could be readily achieved within this time.

In few cases, maternal obesity, oligohydramnios or unfavourable fetal position made the examination more difficult, but overall movement is advantageous, as the required real-time sections are “presented” to the examiner as movement of the fetal trunk occurs.

After 28 weeks gestation, the fetus becomes more fixed in position usually with the fetal spine anterior. This means that the fetal heart is some distance away from the transducer so that the penetration and therefore resolution of cardiac structures is reduced. Also rib shadowing becomes more marked in later pregnancy and this again limits the accuracy of the detailed examination of, for example, the ventricular septum. Because of these difficulties, our study is restricted to 18 – 28 weeks of gestational age range.

Firstly**,** Fetal echocardiographic interpretation of anatomical cardiac structures and then followed lastly by accurate identification of the transducer orientation, relative to the fetal trunk, required to produce a recognized cardiac section. In order to perform anatomical evaluation few echocardiographic sections were chosen for description and anatomical imitation. These structures were named according to the main structures displayed by them and taken together provide all the information necessary for the diagnosis of fetal cardiac normality. [21]

The technique is performed as a continuous process, involving the integrated study of the scan planes, seeking the specific structures to be identified.

1. Four-chamber view

This view is technically very easy to obtain. It is taken in the transverse section of the thorax. First, a good abdomen perimeter section is obtained; then the probe is slid cephalad to obtain the four-chamber view. There are three types of four-chamber views: apical, basal, and lateral. The most informative view is the apical or basal four-chamber view. The lateral four-chamber view is the best view to visualize the interventricular and interatrial septum. Necessary observations on the four-chamber view are the following:

- **Number of chambers**. Normally four chambers are seen.
- **Comparison of chamber sizes**. Normally both atria are of the same size. The ventricles should also be identical in size, with no evidence of wall thickening.
- **Identification of structures**

    1. The chamber closest to the spine is the left atrium and the most anterior chamber is the right ventricle.
    2. The echogenic moderator band is present in the right ventricle & flap of the foramen ovale opens into the left atrium.
    3. The crux of the heart is formed by the membranous part of the ventricular septum, the septum primum of the atrial septum, and the septal leaflets of the mitral and tricuspid valves.
    4. The septal leaflet of the tricuspid valve is inserted into the septum closer to the apex than that of the mitral valve. This offset is normally about 3 mm.
    5. The ventricular septum has to be examined right from the apex to the crux for any defects. An echo drop-out is often seen in the interventricular septum, which needs detailed examination. A true ventricular septal defect (VSD) generally has bright margins & An angle between the interventricular septum and the sound beam, clears this confusion as the echo drop-out disappears; however, any change due to a VSD will persist. (21)

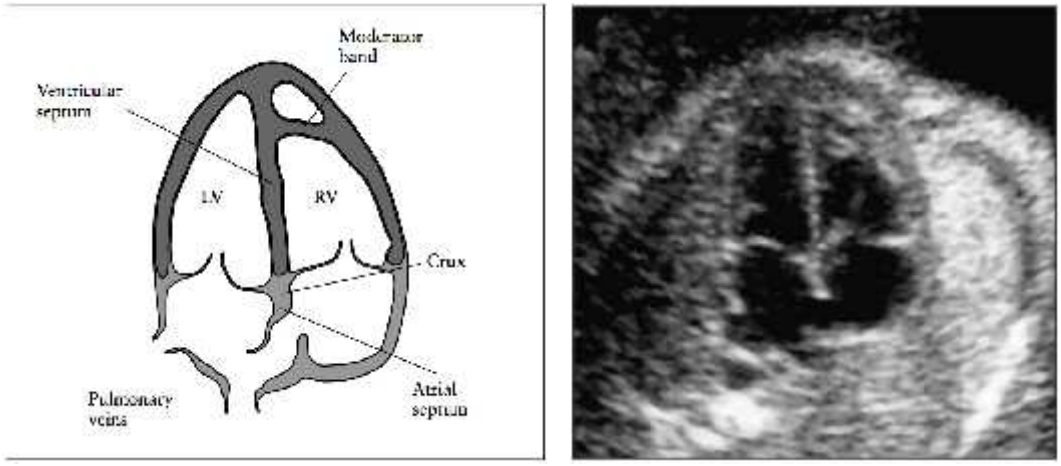


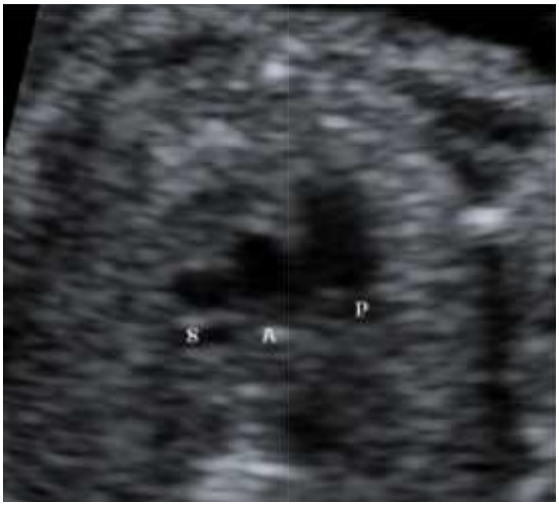
FIG 21: *Showing Four Chamber View Of Heart*

- Real-time evaluation of the two atrioventricular valves, the mitral and tricuspid, should be identified.
- Flow across the atrioventricular valves has to be examined with color Doppler. The direction of flow is from the atria to the ventricles. It is the same across both valves. No aliasing should be seen.
- Only one vessel, the descending aorta, should be seen between the left atrium and the spine in the area behind the heart.

Common anomalies seen on the four-chamber view are Two-chambered heart, Hypoplastic left heart, Ebstein's anomaly, Ventricular septal defects, Atrioventricular septal defect etc.,

2. Three-vessel view

From the position for a four-chamber view, the probe is slid cephalad for the three-vessel view. The three vessels seen on this view are the pulmonary artery in longitudinal section, seen anteriorly and to the left; the aorta in transverse section, seen in the center; and the superior vena cava (SVC) in transverse section, seen to the right.[21]



S- SVC,

A- Aorta,

P-Pulmonary Artery

FIG 22: *Showing Three Vessel View*

3. Outflow tract views:

A. **Right ventricular outflow tract (RVOT).** From the position for a four-chamber view, the probe is slid cephalad and rotated towards the left fetal shoulder to obtain the RVOT view. This reveals the RVOT in its long axis. [21]

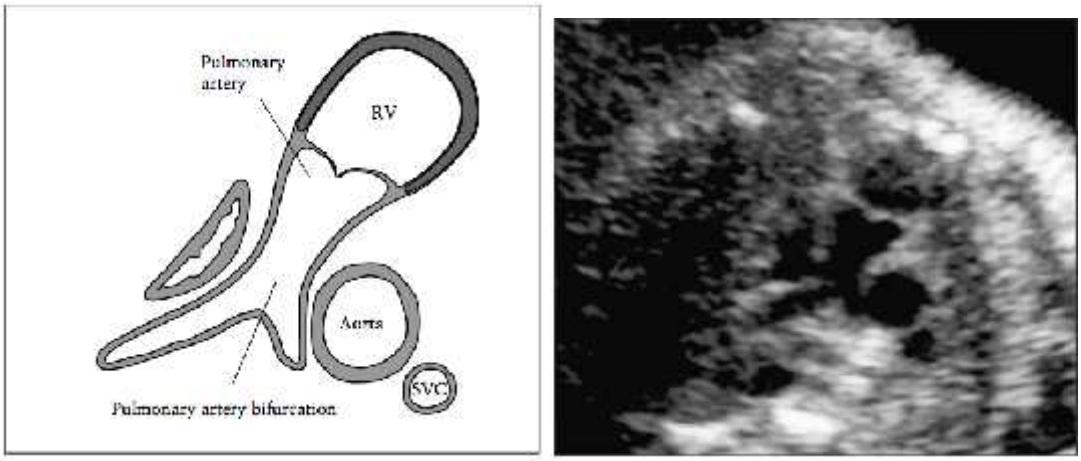


FIG 23: *Showing Right Ventricular Flow Tract*

The pulmonary valve motion can be appreciated within the RVOT. On rotation toward the left, the bifurcation of the pulmonary artery can be seen. Color and pulsed Doppler evaluation of the RVOT is done to look for aliasing at the pulmonary valve.

B. **Left ventricular outflow tract (LVOT).** The LVOT view position is obtained from the four-chamber view position, the transducer is angled towards the right shoulder of the fetus. In this view, 1) the septoaortic continuity needs to be confirmed and 2) the aortic valve motion has to be observed. [21]

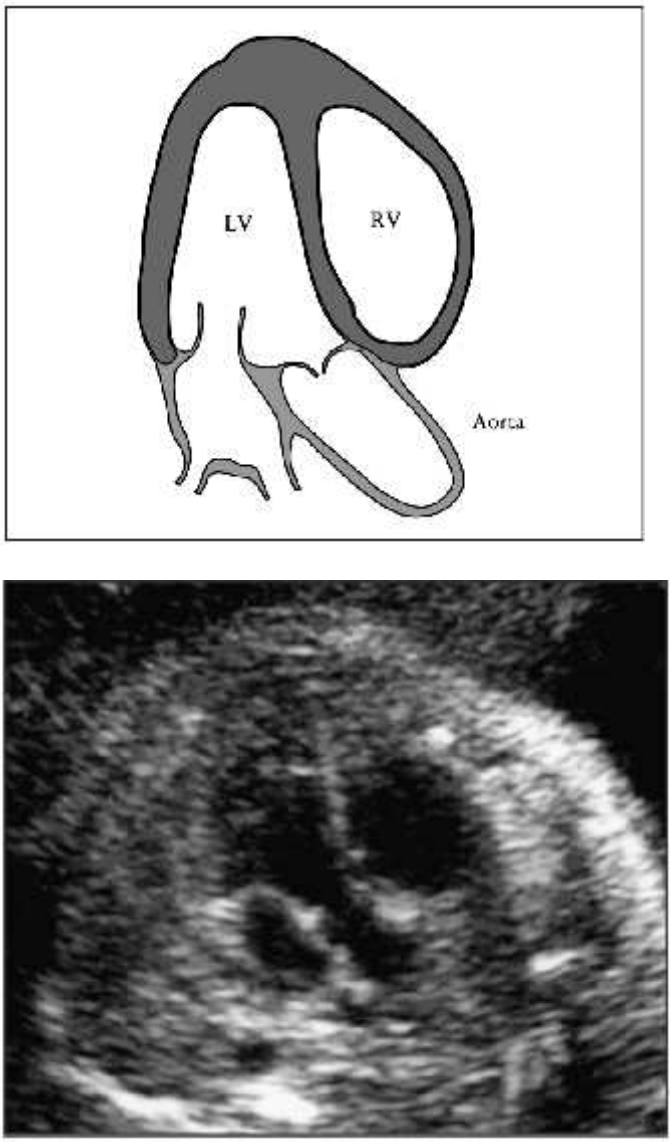


FIG 24: *Showing Left Ventricular Flow Tract*

Common anomalies seen on the outflow tract view are tetralogy of Falot, transposition of great arteries, double outlet right ventricle (DORV), pulmonary stenosis, rhythm abnormalities of the heart and Functional assessment of the fetal heart (two points to be assessed: size of the heart & squeeze of the heart).

5. Arch of aorta

The arch of aorta has to be visualized in a sagittal view. It is narrow and round. The great vessels arise from this arch.

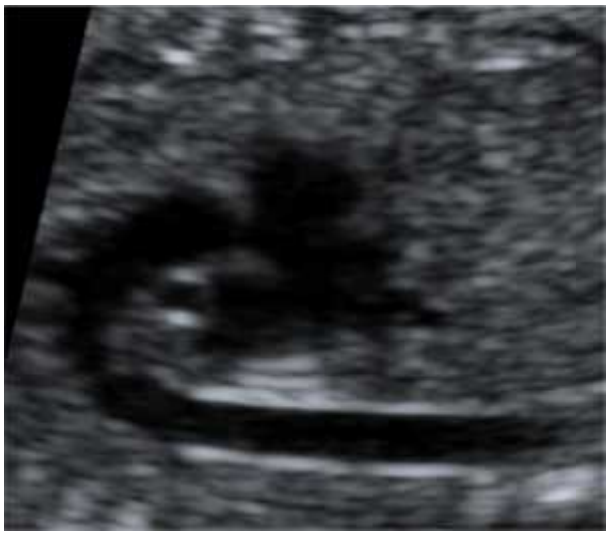


FIG 25: *Showing Aortic Arch View With Great Vessels Arising From The Arch*

On color Doppler, the direction of flow can be seen to be from the ascending aorta to the arch and then to the descending aorta. [21]

CONGENITAL CARDIAC ANOMALISES:

Description of individual anomalies have been described below:

Ventricular Septal Defect

Ventricular septal defect (VSD) is the most common congenital heart disease, seen in 1.5– 3.5 per 1000 live births, and accounting for 30% of all cardiac anomalies [22]. The defect is most commonly (80%) seen in the membranous septum and less commonly in the muscular, outlet, or inlet portions. Defects can be variable in size. VSD is best seen in a four-chamber view as discontinuity in the ventricular

septum, particularly the inlet defects. The ventricular septum is ideally evaluated in images acquired perpendicular to the interventricular septum because a pseudo- VSD, as a result of signal drop-out, can be seen in the superior aspect of images parallel to the ultrasound beam [22]. Membranous septum is also seen in the LVOT view. Out- let defects are best seen when the transducer is angled anteriorly [23]. Small defects can be difficult to detect, particularly in the peri-membranous portion, but Doppler imaging can show flow across the defect. In isolated VSD, bidirectional shunting with right-to-left shunt during systole and left-to-right shunting in diastole is seen, but in VSD associated with other anomalies, unidirectional shunting may be seen. Small defects may close, but large defects require surgical closure.

Atrial Septal Defect

Atrial septal defect (ASD) is characterized by defect in a portion of the atrial septum. It is the fifth most common congenital heart disease, seen in 1 of 1500 live births [24], and is caused by abnormal tissue resorption and deposition during development of the atrial septum. According to its location, it is classified as ostium secundum (midatrial septum), ostium primum (lower atrial septum), sinus venosus (outside the atrial septum in the wall separating the SVC or IVC from the LA), and coronary sinus defect, which can be partial or complete. ASD may be difficult to visualize in a fetus because of the presence of foramen ovale. However, with high-resolution ultrasound, the septum primum is seen in the four-chamber view as a circular or linear structure with a loose pocket configuration, and the septum secundum is seen as a thick stationary structure with the foramen ovale opening into it. Normal foramen ovale measures almost same as the aortic root, with the difference being 1 mm or less (24). The foraminal flap of the foramen ovale is seen moving into the LA at twice the heart rate. A secundum defect is seen as a larger defect in the

central portion of the atrial septum or a deficient foramen flap. A primum defect is seen in the lower part of the atrial septum.

AV Septal Defect

AV septal defect (AV canal defect or endocardial cushion defect) is caused by failure of fusion of the endocardial cushion, resulting in defects of the atrial ostium primum, the ventricular inlet septum common AV valve, and the biventricular AV connections. AV septal defect accounts for 2–7% of con- genital heart defects and is seen in 0.19–0.56 per 1000 live births [(22)]. It is associated with trisomy 21 syndrome, left atrial isomerism, hypoplastic left heart, pulmonary stenosis, coarctation, tetralogy, complete heart block, and extracardiac anomalies. There are two types: the complete type (97% of cases), with common valvular orifice, and the incomplete type, with separate right and left valve orifices. The valve of common AV junction has five leaflets, which are separate in the complete type, but two leaflets are connected by narrow tissue in the incomplete type. It is associated with a cleft in the anterior mitral leaflet. Free regurgitation is seen across the common AV valve [(22)]. Direct shunting may be seen from the LV into the RA. In severe forms, all four chambers communicate, causing left-to-right and right-to-left shunt. Ultrasound shows a defect in the endocardial cushion, with an inlet VSD and primum ASD associated with a single abnormal AV valve that has a T-shaped arrangement. Color Doppler shows open flow across the defect and abnormal AV valve.

Tetralogy of Fallot

Tetralogy of Fallot is characterized by narrowing of the RVOT, VSD, overriding aorta, and right ventricular hypertrophy. It accounts for 5–10% of congenital cardiac defects and is seen in 0.24–0.56 per 1000 live births [(22).] It is caused

by anterior displacement of the conotruncus, resulting in unequal division of conus into a small anterior RV portion and large posterior LV portion. The incomplete closure of the septum results in aortic overriding. It is associated with chromosomal and extra- cardiac abnormalities. On ultrasound, the aorta is seen straddling a large membranous VSD. Depending on the size of the PA, it may not be easily seen and the normal crossing of aorta and pulmonary arteries is not seen. The aorta may be dilated, and the pulmonary valve is stenosed or atretic with a dilated PA. Because of the presence of normal fetal shunts, RV hypertrophy is not seen in the fetus.

Transposition of Great Arteries

Transposition of great arteries is characterized by the abnormal origin of the great arteries from the ventricles because of abnormal spiraling of the conotruncal septum. It is broadly divided into D and L types. D-transposition accounts for 80% of transpositions and is characterized by the aorta originating from the morphologic RV and the PA originating from the morphologic LV. The pulmonary and systemic circulations operate in parallel, rather than serial, circuits. Oxygenation of systemic blood requires mixing via ASD, VSD, or patent ductus arteriosus. On ultra- sound, the morphologic RV is located on the right side of the morphologic LV. The artery originating from the morphologic RV (i.e., aorta) gives off branches to the head and neck, whereas the artery originating from the morphologic LV (i.e., PA) bifurcates and there is a sharp angle of the left PA with the ductus, giving the classic “baby bird beak” sign. The aorta and PA do not cross but are parallel to each other, with the aorta anterior and to the right of the PA [25].

In congenitally corrected transposition (L-transposition), in addition to the ventriculoarterial concordance, there is also AV discordance with the morphologic LA

connected to the morphologic RV and the morphologic RA connected to the morphologic LV. L-transposition accounts for 1% of con- genital heart defects and may be associated with VSD and pulmonic stenosis. Ultrasound shows parallel aorta and PA with the aorta anterior and to the left of the PA. The tricuspid valve may be deformed and inferiorly displaced. Differentiating this from D-trans- position of great arteries requires identification of the morphologic RV and LV.

Double Outlet RV

Double outlet RV (DORV) is characterized by the origin of more than 50% of both the aorta and PA from the RV and is caused by abnormal spiraling of the Truncus arteriosus and the arrest of membranous septal formation. It accounts for less than 1% of congenital heart defects and is seen in 0.08–0.16 per 1000 live births [22, 26]. There are four types of DORV: aorta parallel to the PA and to its right (64%), which resembles Tetralogy of Fallot; aorta anterior and to the right of the PA (26%), resembling D-transposition of great arteries; aorta anterior and to the left of the aorta (7%), resembling L- transposition of great arteries; and aorta posterior and to the right of the PA (3%). DORV is almost always associated with VSD, which provides the only outlet from the LV. It is associated with maternal diabetes or alcohol intake and other cardiac defects, such as LV hypoplasia, mitral valve stenosis or atresia, aortic valve stenosis, aortic coarctation or interruption, and coronary artery anomalies. DORV is best seen in short-axis views, where the aorta and pulmonary arteries do not cross and both the vessels arise from the RV and are parallel to each other. Demonstration of the origin of both the vessels from the same side of ventricular septum is essential to differentiate DORV from transposition of great arteries [22, 26].

Hypoplastic Left Heart Syndrome

Hypoplastic left heart syndrome is characterized by hypoplastic left-sided cardiac structures, including the LV, mitral valve, aortic valve, and aorta. It accounts for 2–4% of congenital cardiac defects and is seen in 0.16–0.25 per 1000 live births [22, 26]. It is more common in boys and is caused by decreased flow in and out of the LV during development (e.g., mitral or aortic stenosis or atresia). Blood flow to the systemic circulation (coronary arteries, brain, liver, and kidneys) in these patients is dependent on flow through the ductus arteriosus. It is associated with aortic coarctation in 80% of cases [27]. On ultrasound, the LV is small (LV:RV ratio < 1) in size; the ventricular septum makes an angle of 90° with the spino-sternal line, and the aortic outflow is smaller than the pulmonary outflow tract. Mitral and aortic valves are hypoplastic or atretic. A single area of flow is seen at the AV level and bidirectional flow at the proximal aorta because of distal aortic Coarctation [24].

Hypoplastic Right Heart Syndrome

Hypoplastic right heart syndrome is characterized by an underdeveloped right side of the heart, including the RV, tricuspid valve, pulmonary valves, and PA. It is seen in 1.1% of stillbirths and is rarer than the hypoplastic left heart syndrome. It is caused by decreased blood flow in and out of the RV during development (tricuspid atresia or pulmonary atresia). The most common cause is pulmonary atresia with an intact ventricular septum. Coronary arterial supply may be abnormal. Prenatal ultrasound shows a small RV with hypertrophy and a small or absent PA, with decreased or absent flow through the tricuspid valve and pulmonary valve [24]

Aortic Atresia or Stenosis

Aortic stenosis is narrowing of the LVOT that can be seen at the valvular, supravalvular, or subvalvular level, with an incidence of 5.2% in newborns [24]. Valvular stenosis may be associated with chromosomal abnormalities and bicuspid aortic valve, subvalvular stenosis with hypertrophic cardiomyopathy or inherited disorders, and supravalvular stenosis with William syndrome. Ultrasound diagnosis is difficult and may show thickening of the aortic valve leaflets, with hypertrophied LV and dilated aorta as a result and helps differentiate it from atresia.

Pulmonary Stenosis

Pulmonary stenosis can be seen at the valvular level or at the infundibulum. It is seen in 7.4% of newborns [24] and is associated with Noonan syndrome, maternal rubella, ASD, supravalvular aortic stenosis, tetralogy, and anomalous pulmonary vein return. Ultrasound diagnosis is difficult and may show thickening of the pulmonary valve leaflets, with hypertrophied RV and dilated PA as a result of poststenotic dilation. Doppler shows high velocities across the valve and differentiates it from atresia.

Echogenic Intracardiac Foci

Echogenic intracardiac foci is seen in 3–4% of fetal hearts, more commonly in the LV (93%) than the RV (5%), representing the reflection of the ultrasound waves off the small papillary muscle or chorda tendinae. It is usually insignificant but may be associated with chromosomal anomalies, such as trisomy 13 or 21, where papillary muscles or chordae may be calcified. It is seen in 13–18% of children with Down syndrome [28]. Detection of echogenic intracardiac foci increases

the likelihood of Down syndrome from 1.8% to 2.8% [29]. Calcification can also be seen in cardiac neoplasms, but neoplastic calcifications are larger, multiple, and not as echogenic as the echogenic intracardiac foci.

Ebstein Anomaly

Ebstein anomaly is characterized by displacement and attachment of one or more tricuspid leaflets (usually septal or posterior leaflets) toward the apex of the RV. The RV is divided into an "atrialized" portion above the leaflets and a muscular portion below the leaflets. It accounts for less than 1% of con- genital heart defects, occurs at a rate of 7% in the fetal population, and occurs in 1 per 20,000 live births [24]. It is associated with maternal lithium use, chromosomal abnormalities, ASD, patent foramen ovale, and pulmonary stenosis or atresia. Ultrasound shows apical displacement of the tricuspid valve into the RV, tethered leaflets, reduction in the size of the functional RV increase in the size of the RV (including the atrialized portion), and tricuspid regurgitation. Cardiomegaly, hydrops, and tachyarrhythmias may be seen. Intrauterine mortality is as high as 85%. Differential diagnosis includes UHL anomaly, tricuspid valve dysplasia, and idiopathic RA enlargement. [24]

Aortic Coarctation or Hypoplasia

Coarctation is discrete narrowing of the aortic arch, most commonly distal to the left subclavian artery. It accounts for 7% of all congenital cardiac defects [30] and 6% of cardiac anomalies seen prenatally (31). It can be associated with chromosomal abnormalities, maternal diabetes, bicuspid valve, aortic stenosis, Turner syndrome, intracranial aneurysms, VSD, ASD, Shone complex, trans- position, Taussig-Bing anomaly, and aortic hypoplasia. Coarctation may be difficult to visualize in ultrasound and is diagnosed when the distal arch is smaller than

normalized values. In hypoplasia, the entire arch is small. In addition, the ascending aorta is small (ratio of the PA to the ascending aorta, > 2 SD above normal). The LV can be small when it is part of the hypoplastic LV syndrome. Coarctation may progress in utero. [24]

Anomalous Pulmonary Venous Return

Anomalous pulmonary venous return can be total or partial. In total anomalous pulmonary venous return, all of the pulmonary veins drain into the RA or a systemic venous channel, which then drains into the RA. This can be at supracardiac, cardiac, or infracardiac levels. In partial anomalous pulmonary venous return, there is abnormal drainage of one to three pulmonary veins directly into the systemic venous circulation. It most commonly affects the left upper lobe (47%), followed by the right upper lobe (38%), right lower lobe (13%), and left lower lobe (2%) [32]. It is bilateral in 4% of cases and is associated with sinus venosus defect in 42% of cases. Ultrasound shows lack of visibility of the four pulmonary veins entering the LA. A small atrium, dilated RV and PA, and right- to-left flow ratio greater than 2.0 are suggestive of anomalous veins.

Single Ventricle

Single ventricle is characterized by a single or two AV valves opening into a single ventricle. It accounts for 2% of congenital heart defects and is caused by failure of development of the interventricular septum. The single ventricle may be the morphologic LV (85%) or the RV. The ventricle may not have an outflow tract. It may be associated with VSD, ASD, common atrium, pulmonary stenosis, and cardiosplenic syndromes. Ultrasound shows a single ventricle without an

interventricular septum. Differential diagnosis includes large VSD or hypoplastic RV or LV [24].

Truncus Arteriosus

Truncus arteriosus is characterized by a single arterial trunk that feeds the systemic pulmonary circulation and coronary arteries with a single semilunar valve. It accounts for 1–2% of congenital cardiac defects, is seen in 0.08–0.16 per 1000 live births [22,26], and is caused by failure of fusion and descent of the conotruncal ridge. It almost always straddles a VSD and receives blood from both the ventricles but rarely originates almost completely from the RV or LV. There are four types (Collett Edwards classification) based on the level of origin of the aorta and pulmonary arteries [33]. An admixture of oxygenated and deoxygenated blood in the common trunk results in subnormal systemic oxygenation. The ductus arteriosus is not necessary for systemic flow and therefore does not fully develop. On ultrasound, a single arterial trunk is seen overriding the interventricular septum, with an associated VSD, and there are several branches connecting with the aorta and pulmonary vasculature.

Ectopia Cordis and Pentalogy of Cantrell

Ectopia cordis is characterized by a heart located outside the thoracic cavity. The heart can be seen in the thoracic (60%), abdominal (30%), thoraco-abdominal (7%), or cervical (3%) locations.[24] Pentalogy of Cantrell is a congenital malformation caused by a continuous anterior defect in thoraco-abdominal wall as a result of developmental failure of the mesoderm between 14–18 days gestational age, resulting in lower sternal defect, anterior diaphragmatic defect, parietal pericardial defect, omphalocele, and congenital cardiac anomalies, with or without ectopia cordis

[34]. It is usually sporadic, with variable expression. Prognosis depends on the severity of the cardiac and extracardiac malformations [34].

Cardiac Neoplasms

Cardiac neoplasms are uncommon in the fetus, with most of them being primary rather than metastastic [35]. Approximately 50% of masses are intracavitary, with inflow or out- flow obstruction; 10% of these masses are malignant (24). Rhabdomyoma (60%), teratoma (25%), fibroma (12%), hemangioma, and hamartoma are the common masses [36]. Myxoma, oncocytic cardiomyopathy, lymphangioma, metastasis, epithelial cysts, mesothelioma of AV node, and valvular blood cysts are rare [35]. Rhabdomyoma is a sessile, smooth, lobulated, and round tumor that is homogeneously echogenic. It is more commonly multiple and typically located in the ventricular septum, atrial, or ventricular free walls. Rhabdomyomas usually do not cause hemodynamic compromise, grow be- cause of maternal hormones, and spontaneously regress after birth. Multiple rhabdomyomas are associated with tuberous sclerosis in 100% and solitary tumors in 50% of cases. Rhabdomyosarcoma, however, appears clustered, irregular, or fragmented. Teratoma can also originate from the pericardium or with- in the heart. Solid and cystic areas with foci of calcification can be seen. Pericardial effusion is always seen in these cases. Fibroma is less common and is seen as a smooth homogeneous mass that blends with the myocardium, often indistinguishable from rhabdomyosarcoma. It is commonly seen in the interventricular septum and less commonly in the ventricular free walls [35]. It is heterogeneous if there is cystic degeneration or calcification [36]. It may be associated with Beckwith-Wiedemann syndrome or Gorlin syndrome

Cardiomyopathies

Cardiomyopathies account for 8–11% of fetal cardiovascular abnormalities with one third of fetuses dying in utero [37]. Cardio- myopathies can be broadly classified as dilated, hypertrophic, and restrictive types. Intrinsic causes of primary cardiomyopathy are single-gene disorders (Noonan syndrome, familial cardiomyopathy, and metabolic abnormalities), mitochondrial and storage dis- orders, chromosomal abnormalities, and α- thalassemia. Extrinsic causes are intrauterine infections, maternal diseases (autoantibodies and diabetes), and twin-twin transfusion syndrome. Dilated cardiomyopathy is the end result of various cardiac disease processes and is characterized by the dilation of cardiac chambers and the reduction of systolic function. In hypertrophic cardiomyopathy, the LV-RV myocardial thickness is increased, without an underlying structural abnormality. It has been associated with maternal diabetes and often regresses during the first 6 months of life. Ventricular hypertrophy can also be seen because of increased afterload. Decreased LV compliance results in cardiac and respiratory distress. Restrictive cardiomyopathy is characterized typically by normal ventricular size and systolic function, but abnormal diastolic function and elevated filling pressure. Secondary cardiomyopathies have better prognosis than do idiopathic or familial cases (38).

Endocardial fibroelastosis is the most common type of restrictive cardiomyopathy and is characterized by diffuse ventricular endocardial thickening caused by proliferation of elastic and collagen fibers and occasional calcifications, predominantly involving the LV. It is sporadic, but can be familial in 10% of cases. It can be primary (infections, autoimmune, ischemia, impaired lymphatic drainage, carnitine deficiency, and mucopolysaccharidosis) when there are no structural cardiac anomalies or secondary to a structural cardiac anomaly (aortic stenosis or atresia, Coarctation,

mitral valve disease, anomalous coronary arteries, or VSD). In the initial stages, the LV is dilated with hypokinesis, but eventually, the LV chamber size decreases with increased wall thick- ness and echogenicity. Tricuspid re- gurgitation is one of the earliest findings [24]; 80% of children with endocardial fibroelastosis present with congestive heart failure during the first year of life [39].

Arrhythmia

Fetal arrhythmias are seen in 2% of cases [25]. Premature atrial and ventricular con- tractions account for 75% and 8% of fetal arrhythmias, respectively. Premature con- tractions are typically benign but may be associated with structural heart disease [24]. Arrhythmias are significant only when they are sustained (< 10%), resulting in supraventricular tachycardia severe bradyarrhythmia causing complete heart block. Fetal tachycardia is diagnosed when the heart rate is greater than 180 beats/ min. Supraventricular tachycardia (paroxysmal supraventricular tachycardia, atrial flutter, or atrial fibrillation) is more common than ventricular tachycardia, may be associated with structural cardiac disease and hydrops fetalis, and may require medications with careful fetal and maternal monitoring. Fetal bradycardia is diagnosed when the heart rate is less than 100 beats/min, lasting more than 10 seconds, and is often associated with fetal hypoxia or asphyxia. Complete heart block may be a result of complex cardiac malformation in the AV junction or by autoimmune disorders [40].

NEED FOR THE STUDY – FETAL ECHOCARDIOGRAPHY

In 1996, Ingrid Stümpflen et al described 46 cases with sonographically detected abnormalities between 18–28 weeks' gestation. 3085 consecutive women were screened: 2181 were with no known risk factor for congenital heart disease; 540 had maternal risk factors for congenital heart disease; 364 had sonographically

detected abnormalities. He described the four-chambered view, outflow-tract views and used color-flow mapping, Doppler and M-mode imaging. The findings obtained were detected prenatally by echocardiography—15 in the group with no risk factors, three in the group with maternal risk factors, and 28 in the group with sonographic abnormalities. (41)

In 2001, Garne.E et al has evaluated the prenatal diagnosis of congenital heart disease by ultrasound investigation in well-defined European populations (experience from 20 European registries). The results obtained were; 2454 cases with congenital heart disease with an overall prenatal detection rate of 25%. And in Western Europe 1694 cases with isolated congenital heart diseases of which 16% were diagnosed prenatally. Malformations affecting the size of the ventricles were detected prenatally in half of the cases. The presence of associated malformations significantly increases the prenatal detection rate. (42)

In 2002, Yagel et al described three vessels & tracheal view (3VT), a novel and simple method to examine the great vessels in the mediastinum, and its applicability in the clinical practice of fetal echocardiography, while establishing nomograms for cardiac vessel measurements obtained in this view. The three vessels and trachea view was examined in 379 low-risk gravidae between 14+0 and 23+6 weeks' gestation. Six parameters in this plane were measured to establish nomograms. (43)

In 2009, Nitin G. Chaubal et al conducted a study on fetal echocardiography and showed that four-chamber view; the three-vessel view and the outflow-tract view are sufficient to diagnose 80.85% of cardiac anomalies, and how fetal

echocardiography apart from identifying structural defects in the fetal heart, can be used to look at rhythm abnormalities and other functional aspects of the fetal heart. [44]

In 2011, Nuruddin Badruddin Mohammed et al described the methods available for Fetal echocardiography, Examination of the Fetal heart, Fetal echocardiography as a screening tool used for in- utero diagnosis of abnormal Fetal cardiovascular structure and physiology, and the role of early Fetal echocardiography. They have showed the importance of carrying out fetal echocardiography in second trimester in detecting congenital cardiac anomalies. [45]

In 2003, Vinals.F et al described a new tool for the prenatal screening of congenital heart defects known as STIC - Spatio-temporal image correlation to assess the feasibility and capability of STIC acquisition, performed by a general obstetrician performing antenatal ultrasound, to visualize fetal cardiac structures in women undergoing routine obstetric ultrasound examination, in order to obtain information to confirm normality of the fetal heart during intrauterine life. This was a prospective study of one hundred fetuses with echocardiographically confirmed normal hearts and no extracardiac anomalies with gestational ages ranging between 18 and 37 weeks. [46]

In 2013, Yihua He et al described the application of spatiotemporal image correlation technology in the diagnosis of fetal cardiac abnormalities & aim of this study was to investigate the clinical application value of STIC technology combined with traditional 2D ultrasound in the diagnosis of fetal cardiac abnormalities. A total of 1,286 fetuses were subjected to sequential echocardiographic examination, during which STIC technology was used to collect heart volume data and carry out image post-processing and off-line analysis. [47]

In 2013, Yifei Li et al described the performance of different scan protocols of fetal echocardiography in the diagnosis of fetal congenital heart disease: a systematic review and meta-analysis where there results have suggested that great diagnostic potential for fetal echocardiography detection as a reliable method of fetal congenital heart disease. But at least 3 sections view (4 CV, OTV and 3 VTV) should be included in scan protocol, while the STIC can be used to provide more information for local details of defects, and can not be used to make a definite diagnosis alone with its low specificity. [48]

In 2013, Mi-Young Lee et al described the technique of fetal echocardiography as a routine evaluation of normal fetus with the prenatal diagnosis of cardiac defects that depends on the knowledge, skill, and experience of practitioners. [49]

MATERIALS AND METHODS:

SOURCE OF DATA:

All pregnant women (between 18 weeks to 28 weeks of gestation) were randomly selected from the routine antenatal ultrasound scans to our department , B.L.D.E.U's Shri B.M. Patil Medical College Hospital and Research Centre, in whom the fetal echocardiography was screened for congenital anomalies. Fetal echocardiography was also performed in high-risk pregnancies.

METHOD OF COLLECTION OF DATA:

All pregnant women (falling under 18 weeks to 28 weeks of gestation) were randomly selected from the routine antenatal ultrasound scans to our institution from October 2013 & July 2015 for evaluation of fetal well-being by using transabdominal ultrasound.

The study protocol includes localization of fetal heart, identifying normal structures of the fetal heart by following a step wise procedure including different views such as four chamber view (4CV), outflow tract view (OTV), aortic arch view (AoA), three vessel view (3VV) & short axis view of ventricles.

Some of the pregnancies with 'high- risk' factors are noted during the study. These high risk factors are described below.

HIGH- RISK GROUPS:

Family history of congenital heart disease: The majority of the congenital heart disease follows pattern of mutli-factorial inheritance [50]. The incidence of recurrence in a family with one affected child will vary with the defect involved lies between 1 in 50 [51] for common defects to 1 in 80 for rarer defects. Where a patient is affected the incidence of recurrence is approximately 5%. [52] Three percent congenital heart

disease occur as a component in single gene disorders. Marfan's syndrome is a autosomal dominant connective tissue disorder. The most important cardiac manifestation of this syndrome is dilatation of the aortic root which can be readily appreciated echocardiographically in post natal life [53]. The Holt-Oram syndrome is a dominant disorder in which a common component of the syndrome is atrial septal defect. [54] Some disorders such as asplenia and polysplenia syndromes, have been described in sibships, [55] and may be transmitted in a recessive manner although the commonly quoted incidence of recurrence of these abnormalities is 1 in 20. [56]

Maternal diabetes: The incidence of fetal abnormality in general, including heart disease, is increased by a factor of two in maternal diabetes. [57] Transposition of great arteries is said to be up to 11 times more common. [58]

Fetal ascites: fetal ascites, or non-immune hydrops fetalis, has a known association with cardiac abnormalities. [59] In some cases hydrops may be due to cardiac failure which may be functional or structural in origin. [60,61]

Fetal arrythmia: These are of three main types, tachyarrthymias, bradycardias and irregularity of rhythm. Although none are strongly associated with increased structural cardiac abnormality there is increased risk of congenital heart disease particularly in the bradycardia group. [62]

Fetal anomaly: There is an increased incidence of congenital heart disease in association with other anomalies, an incidence of approximately 30%. [63] Five percent congenital heart disease occurs in association with chromosomal abnormalities. [64] Forty percent of children with down's syndrome have congential heart disease and between 90-100% of trisomy 13 and 18 are affected. [65]

Miscellaneous groups: This group of pregnancies studied included those exposed to infection or drug ingestion during the first trimester, where the infection or drug had a known association with the causation of congenital heart disease. Rubella virus, mumps and cytomegalovirus, are known to affect the fetal heart. [66] Steroid, oestrogen, lithium and anticonvulsant ingestion during early pregnancy are all said to increase the incidence of congenital heart malformations. [67]

Suspected cardiac anomaly: During the latter part of the study, other centres, aware of our interest in the fetal heart, referred cases where cardiac abnormality had been suspected on a routine antenatal ultrasound scan.

THE SCANNERS AND TRANSDUCERS USED.

Machines which were used in the study are SIEMENS ACUSON X700 (frequency range of 1-5 MHZ) & PHILIPS HD11-XE (frequency range of 2-5MHZ). The equipment used for performance of fetal echocardiography has an excellent B-mode, with a good cine-loop facility so that one can scroll back frame by frame and capture the frame of interest. The spatial and temporal resolution for these machines is good. The systems have a Color Doppler, Pulsed Doppler, and Continuous wave Doppler, M-mode & B-mode imaging capabilities.

FIG 26: PHILIPS HD11-XE

INCLUSION CRITERIA:

All pregnancies from 18 weeks to 28 weeks of gestation are screened for fetus with congenital cardiac anomalies on an obstetric scan.

EXCLUSION CRITERIA:

All pregnancies till 18 weeks of gestational age & after 28 weeks of gestational age.

STUDY DESIGN:

Descriptive study.

OBSERVATION AND RESULTS

A total of 4693 gravid women who had their routine obstetric scans, were screened for anomalies and 33 cardiac abnormalities were detected during the study. There were associated extracardiac anomalies noted during the study.

The study was carried out in patients with suspected cardiac anomalies in routine ANC visits falling under 18 weeks to 28 weeks of gestational age who were referred to department of radio-diagnosis.

The maternal age group in this study ranges between 16- 36 years. The mean maternal age in the study is 27. There was association seen between the congenital cardiac anomalies and the maternal age, where age group of 30 years and above showed increased frequency of cardiac anomalies.

Ventricular septal defect (VSD) was the most common anomaly identified (18.2%) and AVSD is the next most common anomaly identified (15.2%), in this study.

TABLE 2: ASSOCIATON BETWEEN ECHOCARDIOGRAPHIC FINDINGS AND MATERNAL AGEGROUPS

ECHOCARDIOGRAPHIC FINDINGS	AGEGROUP				Total
	20 AND BELOW	21-25	26-30	31 AND OLDER	
AORTIC STENOSIS	0	0	1	1	2
ASD	0	0	2	1	3
AVSD	0	1	2	2	5
COMPLETE TGA	0	0	1	1	2
DILATED RIGHT ATRIUM	2	0	0	0	2
DILATED RIGHT VENTRICLE	0	1	0	0	1
DORV	0	1	0	0	1
DORV WITH VSD	0	1	1	0	2
HLH	0	1	1	0	2
HRH	0	0	0	1	1
CARDIAC MALPOSITIONS	1	1	0	0	2
MULTIPLE IEF	1	0	0	0	1
TOF	0	0	0	3	3
VSD	0	0	2	4	6
TOTAL	4	6	10	13	33

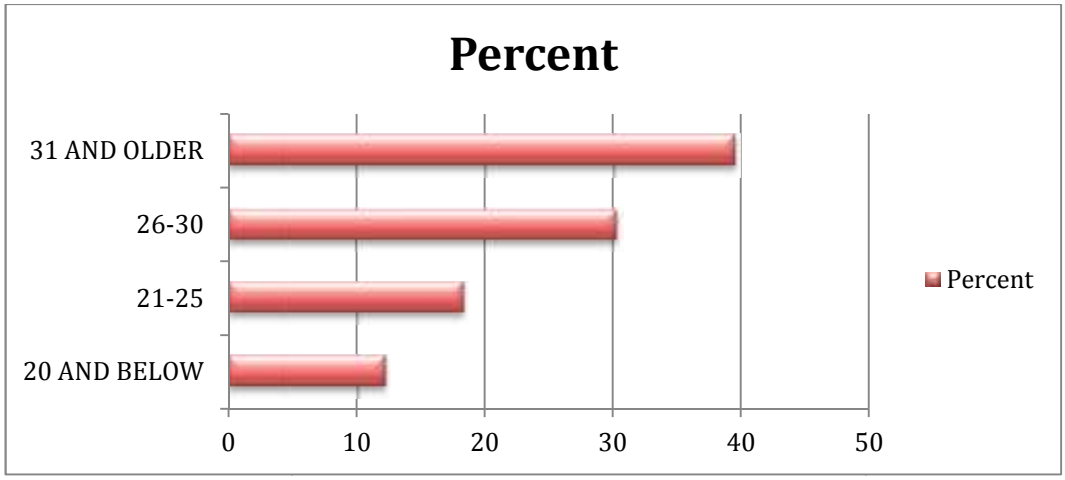
TABLE 3: MEAN MEDIAN MODE OF MATERNAL AGE AND GESTATIONAL AGE.

		AGE	GESTATIONAL AGE (USG)
N	Valid	33	33
	Missing	0	0
Mean		27.33	23.85
Median		27.00	24.00
Mode		27	24
Std. Deviation		5.224	2.635
Minimum		16	18
Maximum		36	28

TABLE 4: MATERNAL AGE AND FREQUENCY OF OCCURRENCE

	MATERNAL AGE IN YEARS	Frequency	Percent
Valid	20 AND BELOW	4	12.1
	21-25	6	18.2
	26-30	10	30.3
	31 AND OLDER	13	39.4
	Total	33	100.0

GRAPH 1: FREQUENCY OF ANOMALIES AND MATERNAL AGE

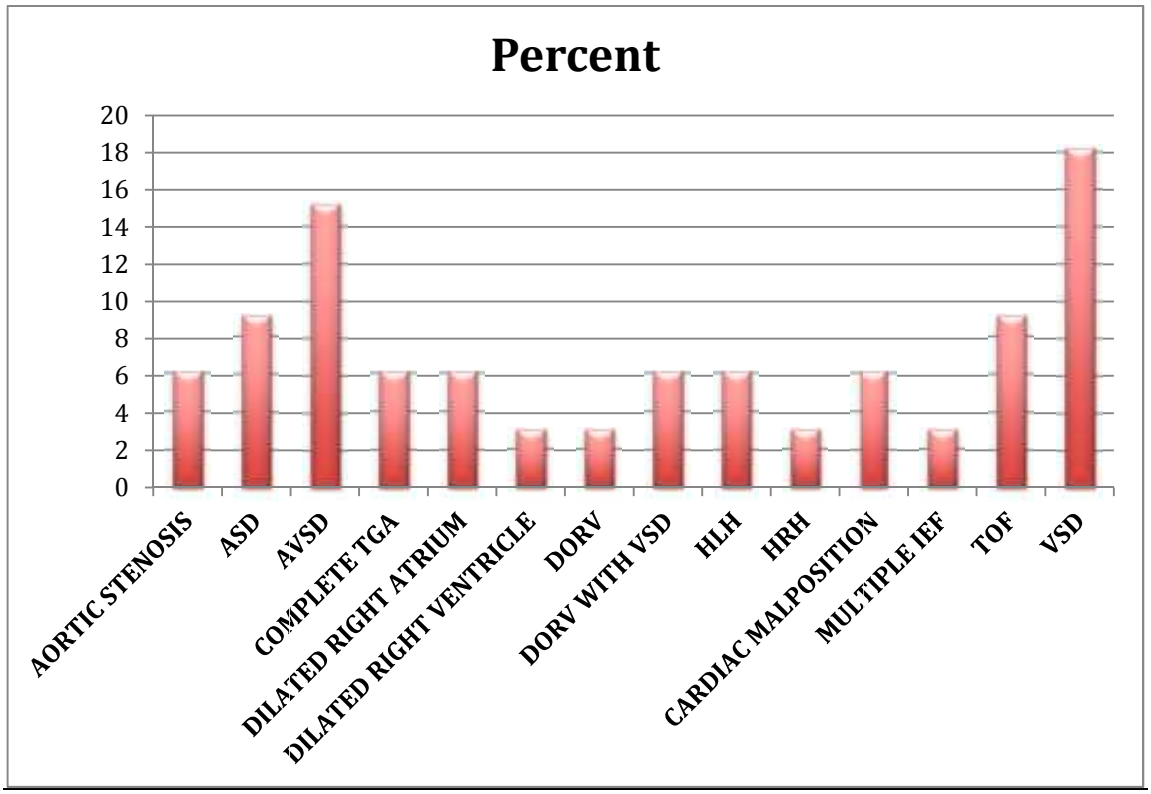


The frequency of anomalies found in the maternal age groups of 26-30 years (30.3%) & 31 years and above (39.4%) showed increased frequency of anomalies.

TABLE 5: FREQUENCY OF CONGENITAL CARDIAC ANOMALIES

ECHOCARDIOGRAPHIC FINDINGS	Frequency	Percent
AORTIC STENOSIS	2	6.1
ASD	3	9.1
AVSD	5	15.2
COMPLETE TGA	2	6.1
DILATED RIGHT ATRIUM	2	6.1
DILATED RIGHT VENTRICLE	1	3
DORV	1	3
DORV WITH VSD	2	6.1
HLH	2	6.1
HRH	1	3
CARDIAC MALPOSITION	2	6.1
MULTIPLE IEF	1	3
TOF	3	9.1
VSD	6	18.2
Total	33	100

GRAPH 2: FREQUENCY OF CONGENITAL CARDIAC ANOMALIES



In our study, ventricular septal defect is found to most common (18.2%) followed by atrioventricular septal defect (15.2%), the other common findings were ASD, Aortic Stenosis, Complete TGA, DORV, DORV with VSD, HLH, HRH, cardiac malpositions, TOF, Dilated right atrium and dilated right ventricle were detected.

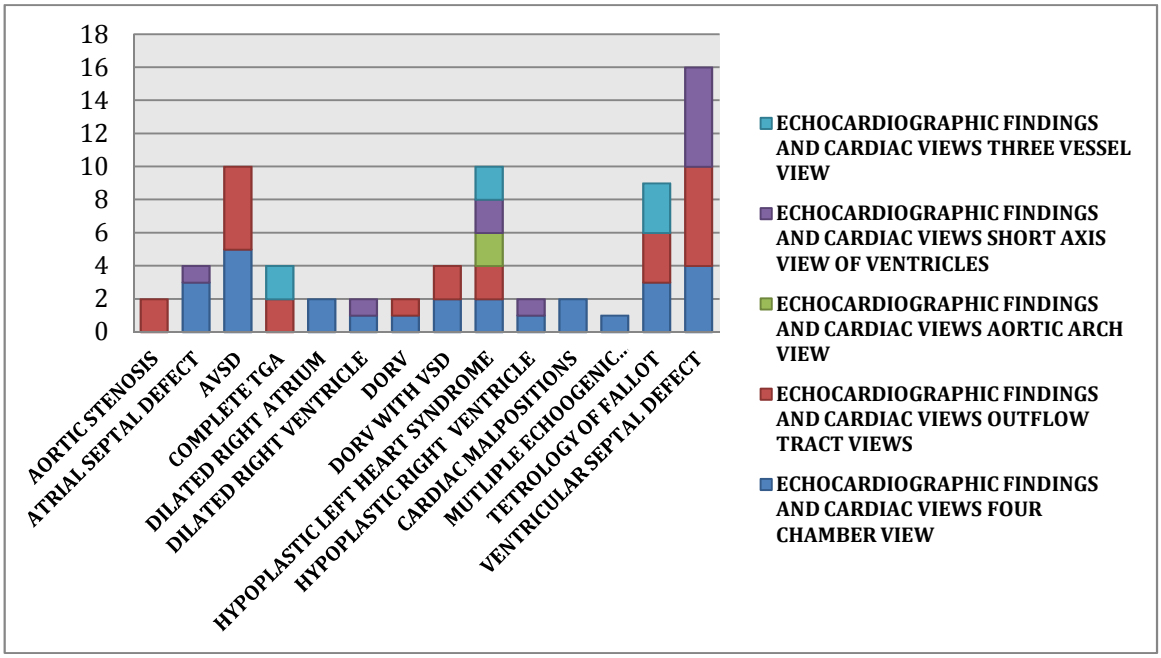
TABLE 6: SHOWING ASSOCIATION BETWEEN ECHOCARDIOGRAPHIC FINDINGS & CARDIAC VIEWS

ECHOCARDIOGRAPHIC FINDINGS	CARDIAC VIEWS									
	FOUR CHAMBER VIEW		OUTFLOW TRACT VIEWS		AORTIC ARCH VIEW		SHORT AXIS VIEW OF VENTRICLES		THREE VESSEL VIEW	
	N	%	N	%	N	%	N	%	N	%
AORTIC STENOSIS	0	0	2	8.7	0	0	0	0	0	0.0
ATRIAL SEPTAL DEFECT	3	11.1	0	0.0	0	0.0	1	9.1	0	0.0
AVSD	5	18.5	5	21.7	0	0.0	0	0	0	0.0
COMPLETE TGA	0	0	2	8.7	0	0	0	0	2	28.6
DILATED RIGHT ATRIUM	2	7.4	0	0.0	0	0.0	0	0	0	0.0
DILATED RIGHT VENTRICLE	1	3.7	0	0.0	0	0	1	9.1	0	0.0
DORV	1	3.7	1	4.3	0	0	0	0	0	0.0
DORV WITH VSD	2	7.4	2	8.7	0	0.0	0	0	0	0.0
HYPOPLASTIC LEFT HEART SYNDROME	2	7.4	2	8.7	2	6.1	2	18.2	2	28.6
HYPOPLASTIC RIGHT VENTRICLE	1	3.7	0	0	0	0.0	1	9.1	0	0.0
CARDIAC MALPOSITIONS	2	7.4	0	0	0	0.0	0	0	0	0.0
MUTLIPLE ECHOOGENIC INTRACARDIAC FOCI	1	3.7	0	0	0	0.0	0	0	0	0.0
TETROLOGY OF FALLOT	3	11.1	3	13.0	0	0.0	0	0	3	42.9
VENTRICULAR SEPTAL DEFECT	4	14.8	6	26.1	0	0.0	6	54.5	0	0.0
Total	27	81.8 %	23	69.7	2	6.1	11	33.3	7	21.2

In this study, four-chamber view was able to detect 81.8% of the cases described. 4CV was able to detect all cases of ASD, AVSD, dilated right atrium & right ventricle, DORV, DORV with VSD, Hypoplastic left heart syndrome, Hypoplastic right heart, increased levocardia, mesocardia, multiple IEF & TOF. The findings were confirmed from other views wherever required. Among six cases of VSD, the 4CV was able to detect 4 cases and the other two cases were detected with other views. The 4CV was not able to detect cases of aortic stenosis and complete TGA.

In this study, short axis view was able to detect 33.3% of the cases described. This view was able to detect all the six cases of VSD, two cases of HLHS, one case of ASD, one case of dilated right ventricle & one case of HRH. The findings were confirmed from other views wherever required. The short axis view was not able to detect cases of aortic stenosis, AVSD, complete TGA, dilated right atrium, DORV, DORV with VSD, increased levocardia, mesocardia, multiple IEF and TOF.

GRAPH 3: SIGNIFICANCE OF CARDIAC VIEWS



In this study, three-vessel view of ventricles was able to detect 21.2% of the cases described. Using this view we were able to detect all the cases of complete TGA, Hypoplastic left heart syndrome & TOF. The findings were confirmed from other views wherever required.

The 3VV was not able to detect cases of aortic stenosis, ASD, AVSD, dilated right atrium, dilated right ventricle, DORV, DORV with VSD, HRH, increased levocardia, mesocardia, multiple IEF and VSD.

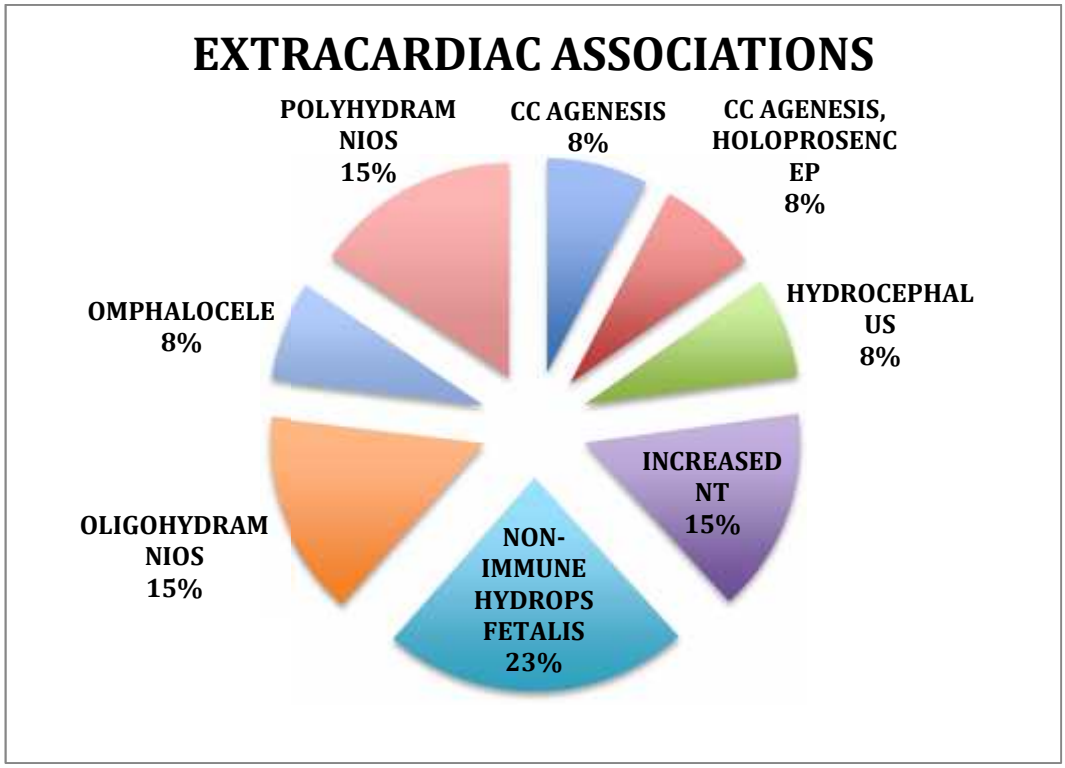
In this study, OTV was able to detect 69.7% of the cases described. Using this view we were able to detect all the cases of aortic stenosis, complete TGA, DORV, DORV with VSD, Hypoplastic left heart syndrome, TOF, AVSD & VSD. The findings were confirmed from other views wherever required.

The OTV was not able to detect cases of, ASD, dilated right atrium, dilated right ventricle, HRH, increased levocardia, mesocardia and multiple IEF.

In this study, aortic arch view was able to detect 6.1% of the cases described. Using this view we were able to detect all the cases of Hypoplastic left heart syndrome. The findings were confirmed from other views wherever required.

The aortic arch view was not able to detect any of the other anomalies.

GRAPH 4: PERCENTAGE OF EXTRACARDIAC ASSOCIATIONS



In our study, we found the following extracardiac associations:

Corpus callosal agenesis was seen in association with HLH in one case.

Corpus callosal agenesis with holoprosencephaly was also seen in association with HLH in another case.

Hydrocephalus was seen in association with DORV with VSD in one case.

Increased nuchal translucency was found associated with one case each of ASD & AVSD.

Non-immune hydrops fetalis was found associated with one case each of complete TGA & AVSD.

Oligohydramnios was found associated with one case each of Aortic stenosis & VSD.

Omphalocele was seen in association with VSD in one case.

Polyhydramnios was found associated with one case each of TOF and VSD.

DISCUSSION

Congenital cardiac abnormalities are one of the most important causes for perinatal mortality and mortality. Delay in diagnosis of a defect is an important contributor to morbidity & mortality in early infancy. Prenatal diagnosis of a defect will therefore optimize the chances of survival of an affected infant. Hence it is important to detect the above anomalies with the help of of fetal echocardiography. A total of 4693 gravid women who had their routine obstetric scans, were screened for anomalies & 33 cardiac abnormalities were detected during the study.

As in guidelines for American Institute of Ultrasound in Medicine (AIUM), the incidence rate was 6 per 1000 live births. [68]

Chaubal et al (2009) in fetal echocardiography have showed the incidence of CHD is 8 per 1000 live births. [69]

The incidence found in the study was 0.7% with detection of cardiac anomalies prenatally by using fetal echocardiography.

Maternal age and cardiac anomalies:

We detected 33 cases of cardiac anomalies in the maternal age group ranged from 16-36 years with mean age of 27 years. Of these 13 cases (39.4 %) were above 30 years of age, 10 (30.3%) cases between 26-30 years of age, 6 cases between 21-25 years and 4 cases below age group of 20 years.

In a study by **Assia Miller et al (2011)** in Maternal Age and Prevalence of Isolated Congenital Heart Defects in an Urban Area of the United States found that there is increased prevalence of several congenital cardiac diseases with maternal Age >30 years in the study which consisted of 5289 subjects. [70]

In an another study by **Reefhuis J et al (2004)** in maternal age and non-chromosomal birth defects studied 1,050,616 subjects from 1968- 2000 and found that advanced maternal age (35-40 years) was associated with all congenital heart defects. The above study closely resembles our study with increased incidence of congenial cardiac anomalies with advancing maternal age. [71]

Frequency of anomalies:

Among 33 cases of cardiac anomalies, ventricular septal defect was the most common anomaly detected in our study which was seen in 6 cases, followed by AVSD in 5 cases (15.2%), ASD in 3 cases (9.1%), TOF in 3 cases (9.1 %), DORV and DORV with VSD in 3 cases (9.1%). We also found two cases (6.1%) each of aortic stenosis, hypoplastic left heart syndrome, complete TGA and dilated right atrium and one case (3%) each of dilated right ventricle, HRH, increased levocardia, mesocardia and multiple IEF.

Mark D. Reller et al (2008) studied Prevalence of Congenital Heart Defects in Metropolitan Atlanta from 1998–2005 with 3240 subjects having congenital cardiac diseases and found that VSD is the most common congenital cardiac anomaly. [72]

In a study by **Mark S. Sklansky et al (2009)** to determine the relative importance of the 4-chamber view (4CV) compared with the outflow tract views (OTVs) in prenatal screening for major congenital heart disease (CHD), the incidence of TOF was 17%, coarctation of aorta 13%, TGA 12%, HLHS 10%, VSD and AVSD accounting for 8% each. This study results resembles our study but in our study the most common cardiac abnormality was VSD accounting to 18.2% and frequency of TOF was 9.1%. [73]

Similarly in a study by **Hoffman et al (1995)** to determine the incidence of CHD; prenatal incidence shows VSD as the most common cardiac anomaly with a frequency of 35.7%, followed by coarctation of aorta (8.9%), ASD (8.2%), AVSD (6.7%) and TOF (6.2%). The increased incidence of VSD in this study was due to inclusion of still born babies and aborted fetuses whereas our study was completely based on prenatal incidence using fetal echocardiography. [74]

Cardiac views and pathologies detected:

Four chamber view :

In our study, 4 chambered view detected 27 out of 33 cases (81%) of cardiac anomalies. It gave a clue regarding some abnormality in all cases of ASD, AVSD, dilated right atrium and dilated right ventricle, TOF, DORV, DORV with VSD, HLHS, HRH, cardiac malposition and multiple IEF. Among the patients with VSD, four chamber view was able to detect 4 out of 6 cases (66.6%).

Copel JA, Pilu G, Green J et al (1987) in Fetal echocardiographic screening for congenital heart disease: The importance of the four-chamber view have showed that 92% of cardiac defects may be discovered by some positive findings in the four-chamber view and proposed routine screening for CHD. [75]

Vergani P, Mariani S, Ghidini A et al (1992) in Screening for congenital heart disease with the four chamber view of the heart have studied and showed that they improved their CHD diagnosis sensitivity from 43% to 81% by adding four chambered view in their screening protocol. [76]

Sharland and Allan et al (1992) in screening for congenital heart disease prenatally, results of 2 and 1/2 year study in the south east Thames region showed that after routinely including the four-chamber view in obstetric studies, 53 fetuses were referred for possible CHD over a period of 32 months; previously, from 1980 to 1988,

only 8 fetuses had been referred. They reported a 77% sensitivity for the four-chamber view in the diagnosis of CHD. [77]

Hence introduction of the 'four-chamber view' analysis of the fetal heart during obstetric anomaly scans improves the detection rate of major cardiac lesions, as evident in our study with detection rate of 81%.

In a study by **chaubal et al (2009)** from India on fetal echocardiography the 4 chamber view was useful in detecting two chambered heart, hypoplastic left heart, Ebstein anomaly, VSD and AVSD. Additionally, in our study the four chambered view was able to detect some abnormality in ASD, DORV, DORV with VSD, hypoplastic right heart. (78)

Outflow tract view:

Ogge et al (2006) in his study on prenatal screening for congenital heart diseases with 4 chamber view and outflow tract views was able to detect hypoplastic right and left ventricles, AVSD, ASD, aortic stenosis and pulmonary stenosis, coarctation of aorta, pulmonary atresia with VSD, double inlet ventricle in four chamber view. Similarly in our study four chamber view was not able to detect complete TGA and in few cases of small VSD. [79]

In our study the outflow tract view was able to detect 23 out of 33 cases (69.7%). It detected aortic stenosis, avsd, complete TGA, DORV with VSD, DORV, HLHS, TOF and VSD. Undetetected cases were ASD, dilated right atrium and right ventricle, hypoplastic right heart, cardia malpositions and multiple IEF.

In a study by **Ogge et al (2006)** on prenatal screening for congenital heart diseases with four chamber view and outflow tract views was able to detect TOF, complete TGA, DORV, pulmonary stenosis, coarctation of aorta, Truncus Arteriosus on outflow tract view.[79]

In a study by **Rajiah et al (2011)** in ultrasound of fetal cardiac anomalies, LVOT and RVOT views was helpful in detecting VSD, TOF, aortic atresia and stenosis. [80]

In our study using four chamber view and outflow tract views, we could detect some positive findings in all cases of congenital anomalies. Our study closely reflects the studies done by **Bromley et al (1992)** [81] in fetal echocardiography: accuracy and limitations in a population at high and low risk for heart defects where the study shows 83% of sensitivity of both the views together; and **Stumpflen et al (1996)** [82]conducted a study on effect of detailed fetal echocardiography as a part of routine prenatal ultrasonographic screening on detection of congenital heart disease and showed that using four chamber view, outflow tract view along with color flow imaging the sensitivity of the study was 88.5%.

And a study done **by carvalho et al (2002)** in improving the effectiveness of routine prenatal screening of major congenital heart defects showed that using four chamber and outflow tract views they could detect most of the cardiac defects with a sensitivity of 76%. [83]

Three vessel view:

In our study using four chamber view and three vessel views, we could detect some positive findings in all cases of congenital anomalies. Our study closely reflects the following study.

Qingqing Wu et al (2009) in Application of the 3-Vessel View in Routine Prenatal Sonographic Screening for Congenital Heart Disease described that diagnostic accuracy of prenatal screening for congenital heart disease (CHD) based on a combination of the 4-chamber view and 3-vessel view in an unselected population Twenty-one cases were identified by the 4-chamber view and 5 by an abnormal 3-

vessel view. The sensitivity of the 4-chamber view alone was 65.6% and the specificity was 99.9%. The sensitivity of the combination of the 4-chamber view and 3-vessel view was 81.3%, and the specificity was 99.9%. [84]

Short axis view of ventricles:

In our study using short axis view of ventricles we could detect 33.3% of cases and when this view was combined with four chambered view we could detect all the cases of VSD, two cases of HLHS, one case of ASD, dilated right ventricle and one case of HRH. However there were no studies in the literature associated with these findings.

Extracardiac associations:

In a study conducted by **Glauser et al (1990)** in congenital brain anomalies associated with hypoplastic left heart syndrome studied described 3 cases of corpus callosal agenesis and one case of corpus callosal agenesis with holoproscencephaly was seen in hypoplastic left heart syndrome.[85]

In our study hypoplastic left heart syndrome is associated with corpus callosal agenesis in one case and corpus callosal agenesis with holoproscencephaly in another case.

In a study by **Layangool et al (2014)** in survival analysis of down syndrome with congenital heart disease described in two hundred and seventy cases had an initial echocardiographic diagnosis of CHD which included 91 cases of patent ductus arteriosus, 49 VSD, 34 AVSD, 34 secundum type ASD, 6 TOF, 2 coarctation of aorta and 11 other combined lesions. And they considered the spontaneous closure of some cardiac lesions. They showed that downs syndrome was associated with VSD and AVSD. [86]

Kurdi et al (2007) in Non- Immune Fetal Hydrops: Are We Doing The Appropriate Tests Each Time?; described in etiology of non-immune hydrops that structural cardiac defects like hypoplastic left or right heart, atrioventricular septal defects, aortic stenosis or atresia, TOF. [87]

In our study we found the association of atrioventricular septal defect with non-immune fetal hydrops.

Gibin. C et al (2003) in Abdominal Wall Defects And Congenital Heart Disease conducted a study to determine the incidence of cardiac disease associated with abdominal wall defects of 48 fetuses & cardiac findings were identified along. The study showed that abnormal cardiac findings were found in 10 of 22 cases of omphalocele (45%) with one VSD, two ASDs, one ectopia cordis, one dysplasia of the tricuspid valve, one large pericardial effusion and four cases of persistent pulmonary hypertension. [88]

In our study, there is one case of omphalocele associated with VSD.

In our study hydrocephalus was seen associated with DORV with VSD; polyhydramnios with TOF & VSD; oligohydramnios with aortic stenosis & VSD; However, there were no studies in the literature associated with these findings.

SUMMARY

- A descriptive Fetal echocardiography study was done wherein a total of 4693 gravid women who had their routine obstetric scans coming to Shri B M Patil Medical college, were screened for anomalies & 33 cardiac abnormalities were detected.
- The incidence of cardiac anomalies found was 0.7%.
- We detected 33 cases of cardiac anomalies in the maternal age group ranged from 16-36 years with mean age of 27 years. Of these 39.4 % were above 30 years of age & rest of the cases were below 30 years of age.
- The most common cardiac anomaly detected was ventricular septal defect contributing to 18.2% of the cardiac anomalies; followed by AVSD, ASD, TOF, DORV, DORV with VSD, Aortic stenosis, Hypoplastic left heart syndrome (HLHS), complete TGA, dilated right atrium, dilated right ventricle, HRH, increased levocardia, mesocardia and multiple IEF.
- Four-chamber view detected 81% of cardiac anomalies. It detected cases of VSD, ASD, AVSD, dilated right atrium, dilated right ventricle, TOF, DORV, DORV with VSD, HLHS, HRH, cardiac malposition and multiple IEF.
- By using Outflow Tract View, Three Vessel View, Short Axis View of Ventricles, and Aortic Arch View detected complete TGA, Aortic stenosis, aortic atresia as a part of HLHS, VSD and findings of four-chamber view was also confirmed.
- Extracardiac anomalies such as corpus callosal agenesis, corpus callosal agenesis with Holoproscencephaly, omphalocele, hydrocephalus, oligohydramnios & polyhydramnios were associated with cardiac anomalies.

CONCLUSION

Fetal echocardiography is an important diagnostic tool in fetuses at risk of heart diseases, which in the hands of trained professional / fetal echocardiologist has high accuracy. Moreover, the imaging modality (ultrasound) is readily available. The early diagnosis provides professionals as well as families with precious time to prepare for aneuploidy assessment and also prenatal & postnatal management.

Our data indicate that routine antenatal assessment of the four chambers and great vessels between 18 and 28 weeks is effective in the prenatal detection of major CHD. The radiologist performing the routine anomaly scan remains the major contributor to prenatal detection of major CHD. Proper equipment to allow optimization of cardiac views, adequate examination time, and ease of access to tertiary level fetal cardiology services obviously influence the extent to which such a service is successful.

In our study, the most common anomaly detected was ventricular septal defect.

To conclude, it may be difficult or time consuming to perform a dedicated fetal echocardiogram on all patients. However, it may be worthwhile looking at the four-chamber view, the outflow tracts, and the three-vessel view; this would sufficiently diagnose 85-90% anomalies.

Detecting the anomalies prenatally, will assert the value of fetal echocardiography not only as a diagnostic tool but most importantly as a means to improve the outcomes of fetuses affected by cardiac diseases, by serial

prognostication and also in monitoring fetal therapy in certain cases and ruling out associated multisystem anomalies and aneuploidies.

LIMITATIONS

- The true incidence of congenital cardiac anomalies could not be calculated, since postnatal follow-up was not part of the study.
- Our results may not correspond with the conditions prevalent in the general population, as it was a cross sectional descriptive study with a small sample size and duration.
- STIC volume imaging was not incorporated in the study due to technical reasons.

Bibliography

1. *Mitchell Sc, Korones Sb, Berendes Hw. Congenital Heart Disease In 56,109 Births. Incidence And Natural History. Circulation 1971;43:323–32.*

2. *Garrett, W. J. & Robinson , D.E. Fetal Heart Size Measures In Vivo,1970. By Ultasound. Pediatrics 46:25.*

3. *Winsberg, F. Echocardiography Of The Fetal And New Born Heart. 1972 Investigative Radiology, 7, 152-158.*

4. *Kaye, H.H., Tynan, M. & Hunter, S. The Validity Of The Echocardiographic Estimates Of The Left Ventricular Size And Performance In Infants And Children. 1975 British Heart Journal. 37: 371-375.*

5. *Linhart, J. W.,Mintz,G.S., Segal, B. L., Kawai, N. & Kotler, M N. Left Ventricular Volume Measurements Of Echocardiography. Fact Or Fiction? 1975. American Journal If Cardiology 36, 114.*

6. *Egblad, H., Bang, J. & Northeved, A. Ultrasonic Identification And Examination Of Fetal Heart Structures. 1975. Journal Of Clinical Ultrasound 3, 95-105.*

7. *Lee,F.Y.L, Batson, H.W.K., Alleman, N. & Yamaguchi, D. J. Fetal Cardiac Struture Identification And Recognition. 1977. American Journal Of Obstetrics And Gynecology. 129:503.*

8. *Baars, A. M. & Merkus, J.M.W.M. Fetal Echocardiography: A New Approach To The Study Of The Fetal Heart And Its Component Parts. 1977. European Journal Of Obstetric And Gynecologic Reproductive Biology, 7, 91-100.*

9. *Henrion, R. & Aubry, J. P. Fetal Cardiac Abnormality And Real Time Ultrasound Study: A Case Of Ivermerk Syndrome. 1979. Contributions To Gynecology And Obstetrics, 6, 119-122.*

10. *Sahn, D. J., Lange, L.W., Allen, H. D., Goldberg, S. J ., Anderson, C., Giles, H. & Haber, K. Quantitative Real- Time Cross Sectional Echocardiography In The Developing Normal Human Fetus And New Born, 1980, Circulation 62, 588- 597.*

11. *Allan, L. D., Tynan, M. J., Campbell, S., Wilkinson, J. L. & Anderson, R.H.*

Echocardiographic And Anatomical Correlates In The Fetus. 1980, British Heart Journal. 44, 441-451.

12. *Kleinman, C. S., Hobbins, J.C., Jaffe, C. C . , Lynch, D. C. & Talner , N.S. Echocardiographic Studies Of The Human Fetus: Prenatal Diagnosis Of Congenital Heart Disease And Cardiac Dysrhythmias. 1980. Pediatrics 65, 6 , 1059- 1067.*

13. *Allan, L. D., Little, D. , Campbell, S. & Whitehead, M. I. Fetal Ascites Associated With Congenital Heart Disease. 1981. British Journal Of Obstetrics And Gynecology 88, 453 -456.*

14. *Kleinman, C. S., Donnerstein, R. L., Devore, G . R., Jaffe, C .C ., Lynch, D. C ., Berkowitz, R. L., Talner, N. S. & Hobbins, J. C. Fetal Echocardiography For Evaluation Of In Utero-Congestive Heart Failure. 1982. The New Engalnd Journal Of Medicine 306, 568-575.*

15. *Wells, P. N. T. Biomedical Ultrasonics. Chapter 1. Wave Fundamentals. Pg 1-25. 1977. London Academic Press.*

16. *Wells, P. N. T. Biomedical Ultrasonics. Chapter 4. Velocity Absorption And Attenuation In Biological Materials. Pg 110-144. 1977. London Academic Press.*

17. *Langman's Medical Embryology -12th Ed; Part II, Chapter-13 Cardiovascular System, Sadler, T. W. (Thomas W.) Pg 162- 201. 2012. Lippincott Williams & Wilkins.*

18. *Fetal echocardiography – 2nd edition; Julia A. Drose. 2010, Saunders Elsevier.*

19. *Diagnostic ultrasound / [edited by] Carol M. Rumack … [et al.].—4th ed. Pg1294-1396.*

20. *Mi-Young Lee, Hye-Sung Won (2013). Technique Of Fetal Echocardiography Korean Society Of Obstetrics And Gynecoogy. 2013;56(4):217-226*

21. *Nitin G. Chaubal, Jyoti Chaubal ,Thane Ultrasound Centre, Thane, 1jaslok Hospital And Research Centre, Mumbai, India ; Fetal Echocardiography. Indian J Radiol Imaging / February 2009 / Vol 19 / Issue 1/60-68.7856342.*

22. *Barboza Jm, Dajani Nk, Glenn Lg, Et Al. Prena- Tal Diagnosis Of Congenital Cardiac Anomalies: A Practical Approach Using Two Basic Views. Radio-Graphics 2002; 22:1125–1138.*

23. *Carvalho Js, Ho Sy, Shinebourne Ea. Sequential Segmental Analysis In Complex Fetal Cardiac Ab- Normalities: A Logical Approach To Diagnosis. Ultrasound Obstet Gynecol 2005; 26:105–111.*

24. *Stamm Er, Drose Ja. The Fetal Heart. In: Rumack Ca, Wilson Sr, Charboneau Wj, Eds. Diagnostic Ultrasound, 2nd Ed. St. Louis, Mo: Mosby, 1998:1123–1159*

25. *Naderi S, Mcgahan Jp. A Primer For Fetal Cardiac Imaging: A Stepwise Approach For 2-D Imaging. Ultrasound Q 2008; 24:195–206 .*

26. *Barboza Jm, Dajani Nk, Glenn Lg, Et Al. Prena- Tal Diagnosis Of Congenital Cardiac Anomalies: A Practical Approach Using Two Basic Views. Radio-Graphics 2002; 22:1125–1138.*

27. *Hawkins Ja, Dody Db. Aortic Atresia: Morpho- Logic Characteristics Affecting Survival And Opera- Tive Palliation. J Thorac Cardiovasc Surg 1984; 88:620–626 .*

28. *Bromley B, Lieberman E, Shipp Td, Richardson M, Benacerraf Br. Significance Of An Echogenic Intracardiac Focus In Fetuses At High And Low Risk For Aneuploidy. J Ultrasound Med 1998; 17:127–131.*

29. *Nyberg Da, Souter Vl, El-Bastawissi A, Young S, Luthhardt F, Luthy Da. Isolated Sonographic Markers For Detection Of Fetal Down Syndrome In The Second Trimester Of Pregnancy. J Ultrasound Med 2001; 20:1053–1063*

30. *Kimura-Hayama Et, Melendez G, Mendizabal Al, Et Al. Uncommon Congenital And Acquired Aortic Diseases: Role Of Multidetector Ct Angiog- Raphy. Radiographics 2010; 30:79–98.*

31. *Allan Ld, Crawford Dc, Anderson Rh, Et Al. The Spectrum Of Congenital Heart Disease Detected Echocardiographically In Prenatal Life.* Br Heart J *1985; 54:523–526*

32. *Ho Ml, Bhalla S, Bierhals A, Gutierrez F. Mdct Of Partial Anomalous Pulmonary*

Venous Return In Adults. J Thorac Imaging 2009; 24:89–9

33. *Collett Rw, Edwards Je. Persistent Truncus Arte- Riosus: A Classification According To Anatomic Types. Surg Clin North Am 1949; 29:1245–1270*

34. *Gao Z, Duan Qj, Zhang Zw, Ying Ly, Ma Ll. Pentalogy Of Cantrell Associated With Thoracoab- Dominal Ectopia Cordis. Circulation 2009; 119: E483–E485*

35. *Isaacs H. Fetal And Neonatal Cardiac Tumors. Pediatr Cardiol 2004; 25:252–273*

36. *Lacey Sr, Donofrio Mt. Fetal Cardiac Tumors: Prenatal Diagnosis And Outcome. Pediatr Cardiol 2007; 28:61–67*

37. *Pedra Srff, Smallhorn Jf, Ryan G, Et Al. Fetal Cardiomyopathies: Pathogenic Mechanisms, He- Modynamic Findings, And Clinical Outcome. Circulation 2002; 106:585–591*

38. *Yinon Y, Yagel S, Hegesh J, Et Al. Fetal Cardiomyopathy: In Utero Evaluation And Clinical Signifi- Cance. Prenat Diagn 2007; 27:23–28*

39. *Trastour C, Bafghi A, Delotte J, Et Al. Early Prena- Tal Diagnosis Of Endocardial Fibroelastosis. Ultra- Sound Obstet Gynecol 2005; 26:303–306*

40. *Kleinman Cs, Nehgme Ra. Cardiac Arrhythmias In The Human Fetus. Pediatr Cardiol 2004; 23:234–251*

41. *Ingrid Stümpflen, Andreas Stümpflen, Maria Wimmer, Gerhard Bernaschek Departments Of Prenatal Diagnosis And Therapy , University Of Vienna Medical School, A-1097 Vienna, Austria; Effect Of Detailed Fetal Echocardiography As Part Of Routine Prenatal Ultrasonographic Screening On Detection Of Congenital Heart Disease The Lancet: 1996/September 28/Vol 348 /The Lancet /854-857.*

42. *Garne. E, Stoll. C, Clementi. M And The Euroscan Group : Dr.Garne. E, Pediatric Department, Kolding Hospital, 6000 Kolding, Denmark Has Conducted A Study The Evaluation Of Prenatal Diagnosis Of Congenital Heart Diseases By Ultrasound: Experience From 20 European Registries; Ultrasound Obstet Gynecol 2001; 17: 386–391.*

43. *S. Yagel, R. Arbel, E. Y. Anteby, D. Raveh And R. Achiron (2002) The Three Vessels And Trachea View (3vt) In Fetal Cardiac Scanning. Blackwell Science, Ltd. Ultrasound Obstet Gynecol 2002; 20: 340–345*

44. *Nitin G. Chaubal, Jyoti Chaubal ,Thane Ultrasound Centre, Thane, 1jaslok Hospital And Research Centre, Mumbai, India ; Fetal Echocardiography. Indian J Radiol Imaging / February 2009 / Vol 19 / Issue 1/60-68.7856342.*

45. *Nuruddin Badruddin. Mohammed, Anandakumar Chinnaiya (201l) Evolution Of Foetal Echocardiography As A Screening Tool For Prenatal Diagnosis Of Congenital Heart Disease. The Journal Of Pakistan Medical Association Vol. 61, No. 9, 904- 909*

46. *F. Vinals, P. Poblete And A. Giuliano Spatio-Temporal Image Correlation (Stic): A New Tool For The Prenatal Screening Of Congenital Heart Defects Ultrasound Obstet Gynecol 2003; 22: 388–394*

47. *Yihua He, Junlan Wang, Xiaoyan Gu, Ye Zhang, Jiancheng Han, Xiaowei Liu And Zhian Li Application Of Spatio-Temporal Image Correlation Technology In The Diagnosis Of Fetal Cardiac Abnormalities Experimental And Therapeutic Medicine 5: 1637-1642, 2013.*

48. *Yifei Li, Yimin Hua, Jie Fang, Chuan Wang, Lina Qiao, Chaomin Wan, Dezhi Mu, Kaiyu Zhou (2013). Performance Of Different Scan Protocols Of Fetal Echocardiography In The Diagnosis Of Fetal Congenital Heart Disease: A Systematic Review And Meta-Analysis. Plos One Vol 8(Issue 6): E65484.*

49. *Mi-Young Lee, Hye-Sung Won (2013). Technique Of Fetal Echocardiography Korean Society Of Obstetrics And Gynecoogy. 2013;56(4):217-226*

50. *110. Nora, J . J & Nora A. H. (1979). Genetics And Counseling In Cardiovascular Diseases. P 6. Springfield. Illinois. Charles C. Thomas.*

51. *Nora, J. J. (1968). Multifactorial Inheritance Hypothesis For The Etiology Of Congenital Heart Diseases. Circulation. 38, 604.*

52. *Czeizel, A., Pornoi, A., Peterfly, E. & Tarcal, E. (1982). Study Of Children Of Parents Operated On For Congenital Cardiovascular Malformations. British*

Heart Journal 47: 290-293.

53. *Donaldson, R. M., Emmanuel, R.E., Olsen, E.G.J. & Ross, D. N. (1980). Management Of Cardiovascular Complications In Marfan Syndrome. Lancet Ii, 1178-1181.*

54. *Mckusick, V.A. (1964). A Genetical View Of Cardiovascular Disease. Circulqtion, 30, 326.*

55. *Simpson, J. & Zellweger, H. (1973). Case Report. Familial Occurrence Of Ivermark Syndrome With Splenic Hypoplasia And Asplenia In Sibs. Journal Of Clinical Genetics, 10: 303. 4.*

56. *Rose, V., Izukawa, R. & Moes, C. A.F. (1975). Syndromes Of Asplenia And Polysplenia. British Heart Journal, 37, 840 – 852.*

57. *Miller, H. C. (1946). The Effect Of Diabetic And Prediabetic Pregnancies On The Fetus And New Born Infant. Journal Of Pediatrics 29: 455.*

58. *Liebman, J., Cullum, L. & Belloc, N. B. (1969). Natural History Of Transposition Of The Great Arteries. Anatomy And Birth And Death Characteristic. Circulation 40: 237.*

59. *Beischer, N. A., Fortune, D.W. & Macafee, J. (1971). Non – Immunologic Hydrops Fetal And Congenital Abnormalities. Obstetrics And Gynecology. 38: 86.*

60. *Allan, L.D., Little, D., Campbell, S. & Whitefield, Mi. (1981). Fetal Ascites Associated With Congenital Heart Disease. British Journal Of Obstetrics And Gynecology. 88: 453-456.*

61. *Kleinman, C.S., Donnerstein, R.L., Devore, G.R., Jaffe, C.C., Lynch, D., Berkowitz, R. L. Talner, N. S. & Hobbins, J. D. (1982). The New England Journal Of Medicine. 306: 568-575.*

62. *Shenker, L. (1979). Fetal Cardiac Arrhythmias. Obstetrical And Gynecological Survey. 34: 561-572.*

63. *Hoffman, J.I.E. & Christianson, R. (1978). Congenital Heart Disease In A Cohort Of 19,502 Births With Long -Term Follow-Up. The American Journal Of*

Cardiology. 42: 641 – 647.

64. *Nora, J . J & Nora A. H. (1979). Genetics And Counseling In Cardiovascular Diseases. P 6. Springfield. Illinois. Charles C. Thomas.*

65. *Polani, P. E (1968). Chromosomal Abnormalities And Congenital Heart Diseases. Guy's Hospital Report. 117:323.*

66. *Hardy, J.B. (1968). Viruses And The Fetus. Postgraduate Medicine. 43:156.*

67. *Nora, J . J & Nora A. H. (1979). Genetics And Counseling In Cardiovascular Diseases. P 135-140. Springfield. Illinois. Charles C. Thomas.*

68. *American Institute Of Ultrasound In Medicine. Aium Practice Guideline For The Performance Of Fetal Echocardiography. J Ultrasound Med 2011; 30:127–136.*

69. *Nitin G. Chaubal, Jyoti Chaubal ,Thane Ultrasound Centre, Thane, 1jaslok Hospital And Research Centre, Mumbai, India ; Fetal Echocardiography. Indian J Radiol Imaging / February 2009 / Vol 19 / Issue 1/60-68.7856342.*

70. *Miller A, Riehle-Colarusso T, Siffel C, Frías Jl, Correa A. Maternal Age And Prevalence Of Isolated Congenital Heart Defects In An Urban Area Of The United States. Am J Med Genet A. 2011 Sep;155a(9):2137-45.*

71. *Reefhuis J1, Honein Ma. Maternal Age And Non-Chromosomal Birth Defects, Atlanta--1968-2000: Teenager Or Thirty-Something, Who Is At Risk?; Birth Defects Res A Clin Mol Teratol. 2004 Sep;70(9):572-9.*

72. *Mark D. Reller, Md, Matthew J. Strickland, Phd, Tiffany Riehle-Colarusso, Md, William T. Mahle, Md, And Adolfo Correa, Md, Phd; Prevalence Of Congenital Heart Defects In Metropolitan Atlanta, 1998–2005 J Pediatr. 2008 Dec; 153(6): 807–813.*

73. *Mark S. Sklansky, Md, Darren P. Berman, Md, Jay D. Pruetz, Md, Ruey-Kang R. Chang, Md; Prenatal Screening For Major Congenital Heart Disease Superiority Of Outflow Tracts Over The 4-Chamber View, J Ultrasound Med 2009; 28:889–899*

74. *J.I.E Hoffman; Incidence Of Congenital Heart Disease: II. Prenatal Incidence,*

1995, Pediatr Cardiol 16: 155-165 Vol 16. Issue No 4.

75. *Copel Ja, Pilu G, Green J, Hobbins Jc, Kleinman Cs. Fetal Echocardiographic Screening For Congenital Heart Disease: The Importance Of The Four-Chamber View. Am J Obstet Gynecol. 1987 Sep;157(3):648-55.*

76. *Vergani P, Mariani S, Ghidini A, Et Al. Screening For Congenital Heart Disease With The Four-Chamber View Of The Fetal Heart. Am J Obstet Gynecol 1992; 167: 1000–1003*

77. *Gurleen K. Sharland And Lindsey D. Allan Screening For Congenital Heart Disease Prenatally. Results Of A 2½-Year Study In The South East Thames Region Volume 99, Issue 3, Pages 220–225, March 1992.*

78. *Nitin G. Chaubal, Jyoti Chaubal ,Thane Ultrasound Centre, Thane, 1jaslok Hospital And Research Centre, Mumbai, India ; Fetal Echocardiography. Indian J Radiol Imaging / February 2009 / Vol 19 / Issue 1/60-68.7856342.*

79. *Oggè G, Gaglioti P, Maccanti S, Faggiano F, Todros T. Prenatal Screening For Congenital Heart Disease With Four-Chamber And Outflow-Tract Views: A Multicenter Study. Ultrasound Obstet Gynecol. 2006 Nov;28(6):779-84.*

80. *Prabhakar Rajiah, Ceayee Mak, Theodore J. Dubinksy, And Manjiri Dighe. Ultrasound Of Fetal Cardiac Anomalies; American Journal Of Roentgenology 2011 197:4, W747-W760*

81. *Bromley B, Estroff Ja, Sanders Sp, Parad R, Roberts D, Frigoletto Fd Jr, Benacerraf Br. Fetal Echocardiography: Accuracy And Limitations In A Population At High And Low Risk For Heart Defects. Am J Obstet Gynecol. 1992 May;166(5):1473-81.*

82. *Stümpflen I, Stümpflen A, Wimmer M, Bernaschek G. Effect Of Detailed Fetal Echocardiography As Part Of Routine Prenatal Ultrasonographic Screening On Detection Of Congenital Heart Disease. Lancet. 1996 Sep 28;348(9031):854-7.*

83. *J.S. Carvalho, E Mavrides, E A Shinebourne, S Campbell, And B Thilaganathan. Improving The Effectiveness Of Routine Prenatal Screening For Major Congenital Heart Defects. Heart. 2002 Oct; 88(4): 387–391.*

84. *Qingqing Wu, MD, Man Li, PhD, Lirong Ju, BS, Weiyuan Zhang, MD, Xinghua Yang, PhD, Prenatal Sonographic Screening for Congenital Heart Disease; JUM October 1, 2009 vol. 28 no. 10 1319-1324*

85. *Glauser Ta1, Rorke Lb, Weinberg Pm, Clancy Rr. Congenital Brain Anomalies Associated With The Hypoplastic Left Heart Syndrome. Pediatrics. 1990 Jun;85(6):984-90.*

86. *Layangool T, Sangtawesin C, Kirawittaya T, Prompan W, Prachasilchai P, Pechdamrongsakul A. Survival analysis of Down syndrome with congenital heart disease: a 5-years registry at QSNICH. J Med Assoc Thai. 2014 Jun;97 Suppl 6:S108-14.*

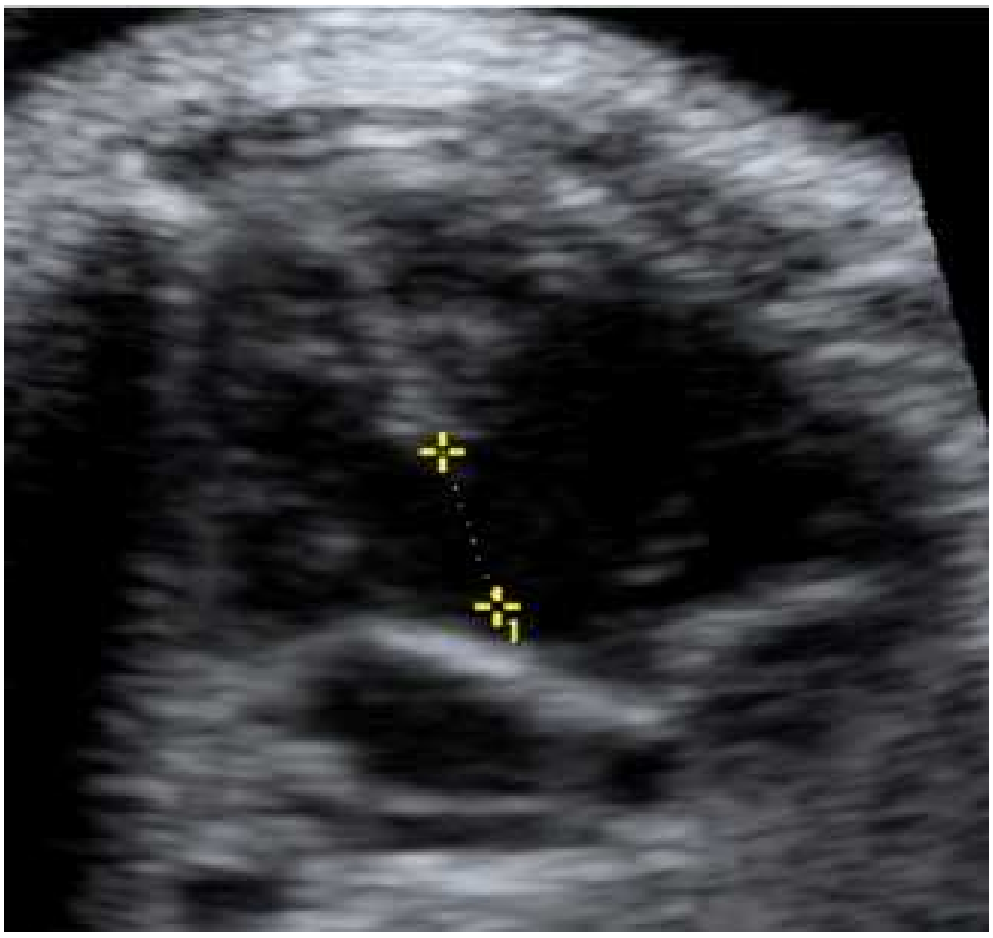
87. *W. Kurdi. Non-Immune Fetal Hydrops: Are We Doing The Appropriate Tests Each Time? J Prenat Med. 2007 Jan-Mar; 1(1): 26–28.*

88. *Gibbin C1, Touch S, Broth Re, Berghella V. Abdominal Wall Defects And Congenital Heart Disease. Ultrasound Obstet Gynecol. 2003 Apr;21(4):334-7.*

REPRESENTATIVE CASES

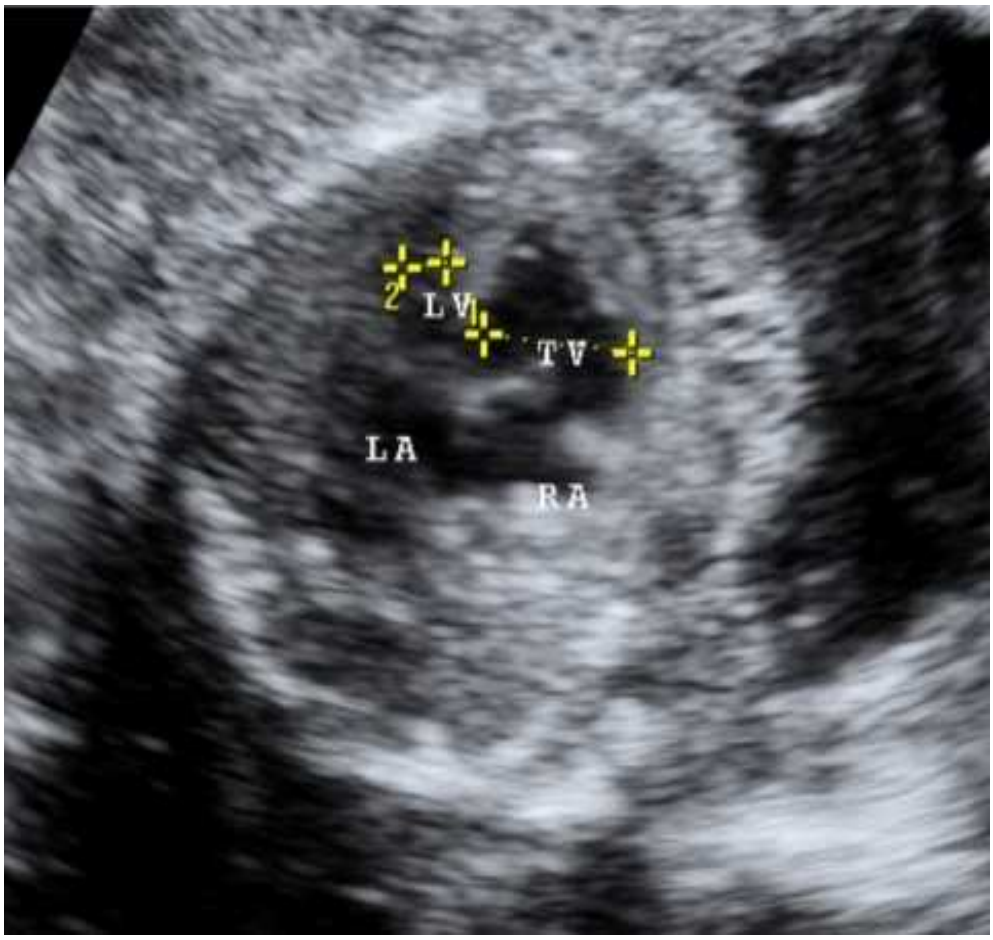
CASE 1

FOUR CHAMBER VIEW SHOWING VENTRICULAR SEPTAL DEFECT



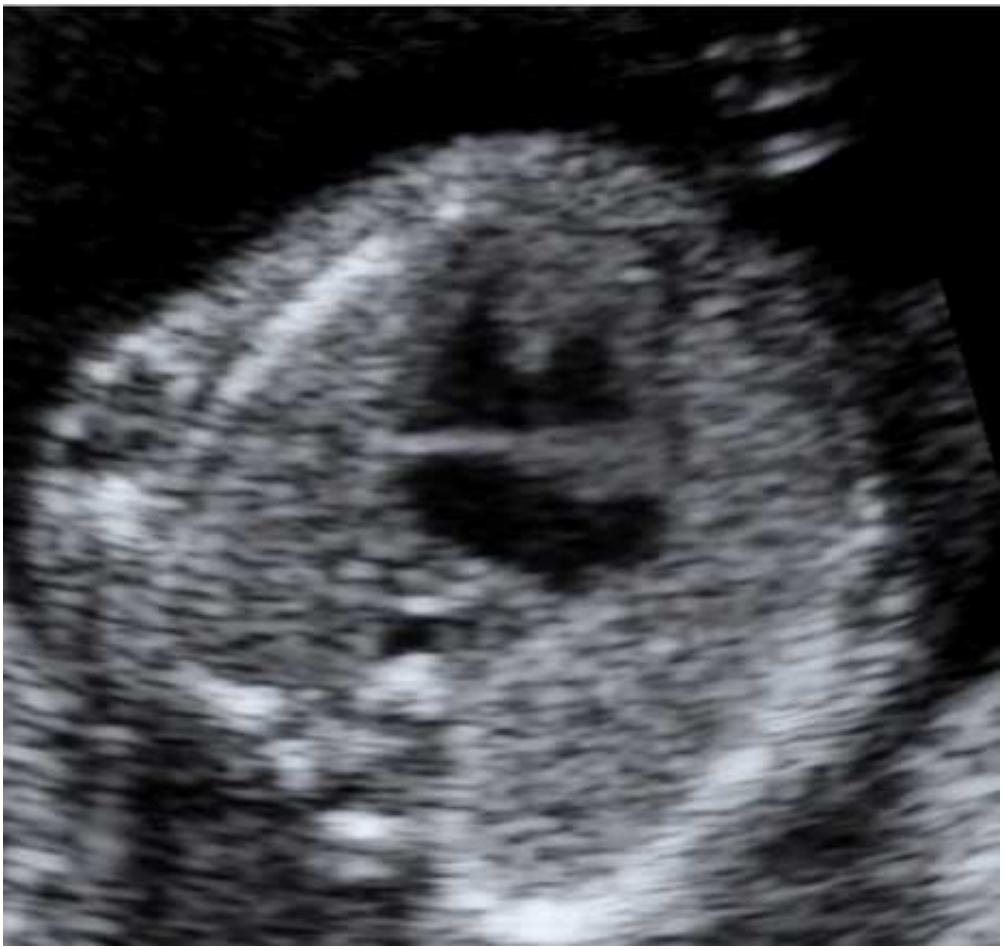
CASE 2

FOUR CHMABER VIEW SHOWING DILATED RIGHT VENTRICLE



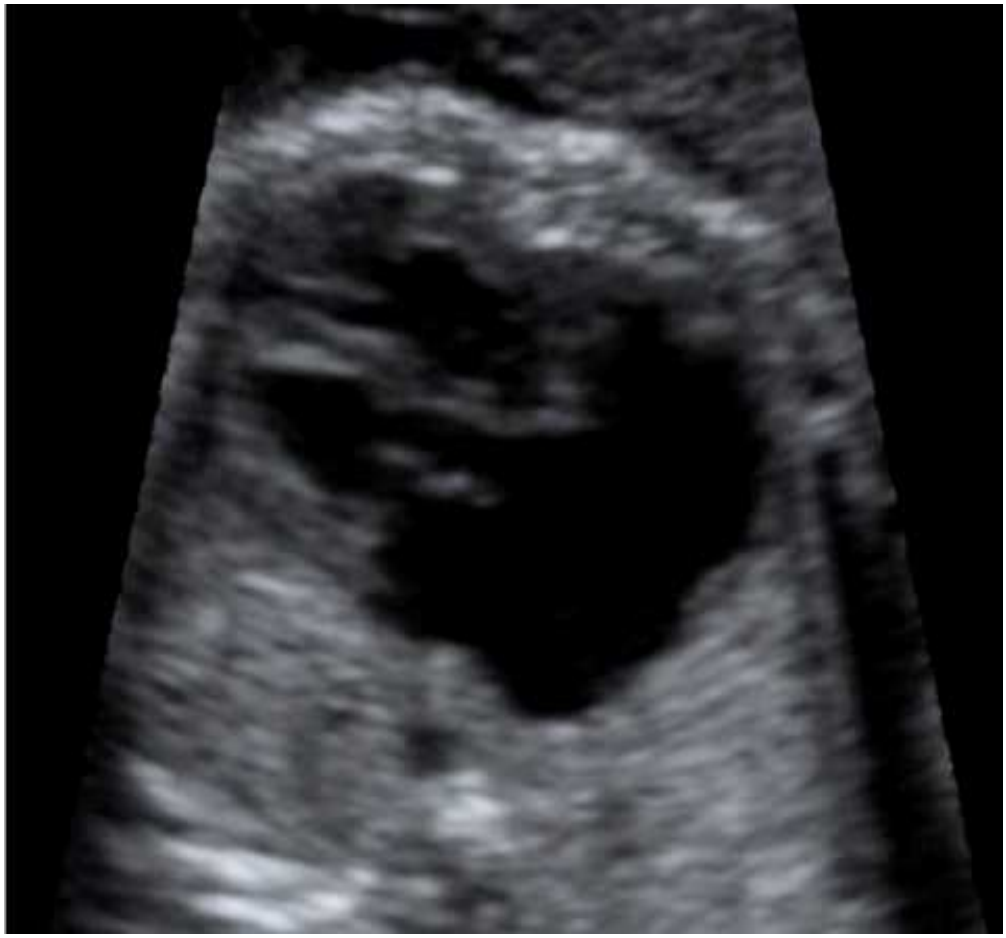
CASE 3

FOUR CHAMBER VIEW SHOWING ATRIOVENTRICULAR SEPTAL DEFECT



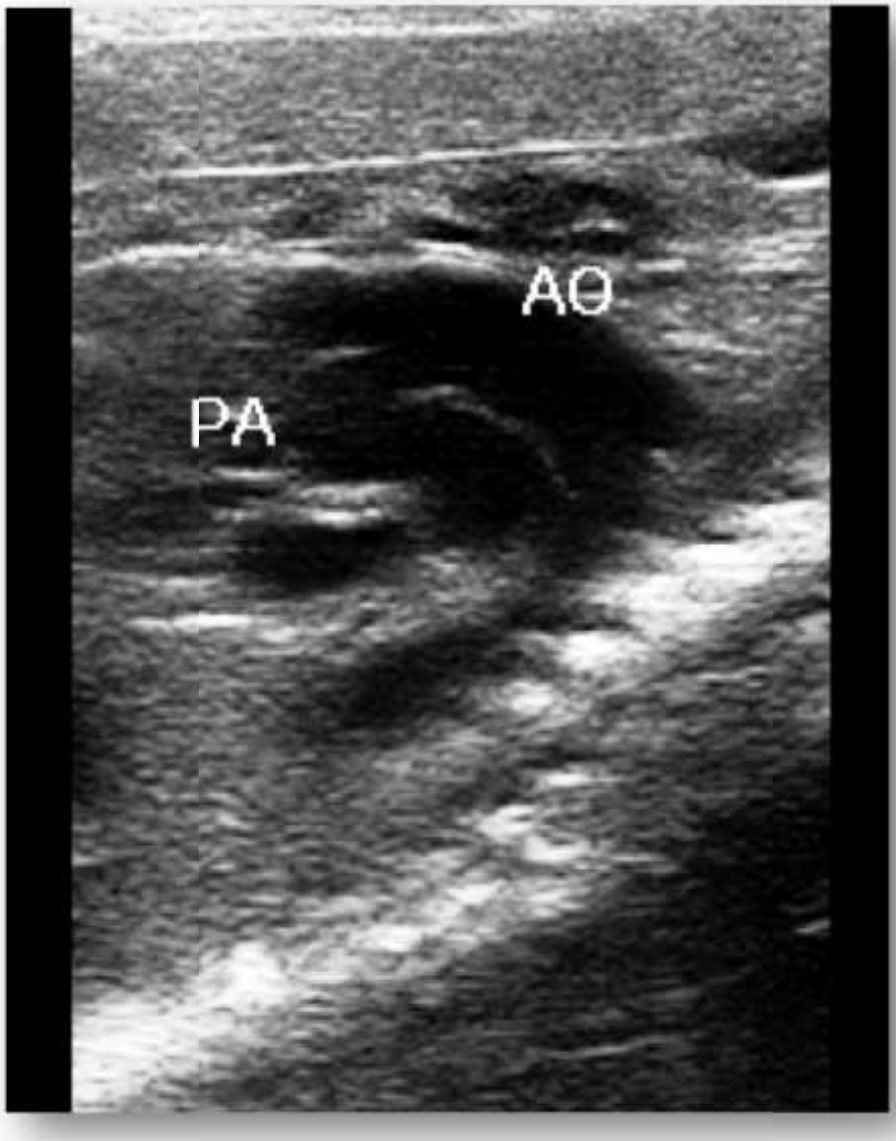
CASE 4

CARDIAC MALPOSITION- INCREASED LEVOCARDIA



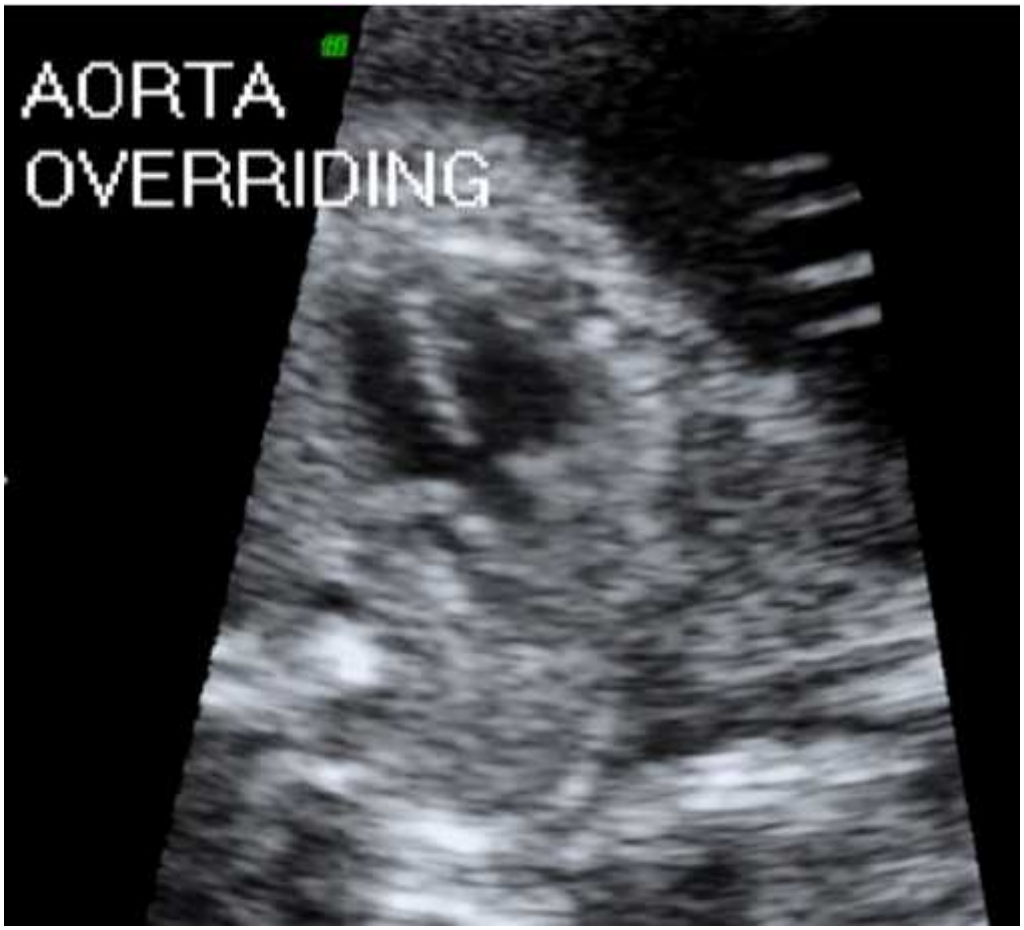
CASE 5

TRANSPOSITION OF GREAT AFTERIES SHOWING PARALLELISM BETWEEN AORTA AND PULMONARY ARTERY.



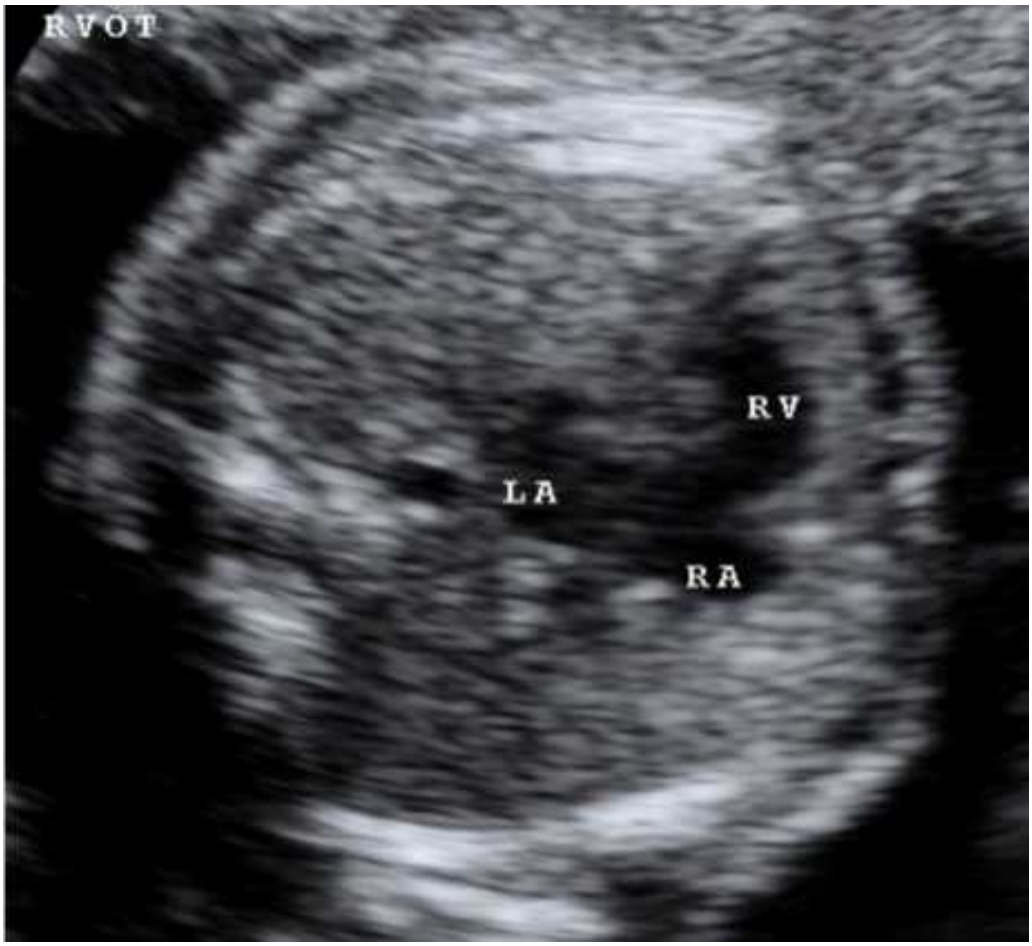
CASE 6

CASE OF TETROLOGY OF FALLOT SHOWING OVER RIDING OF AORTA.



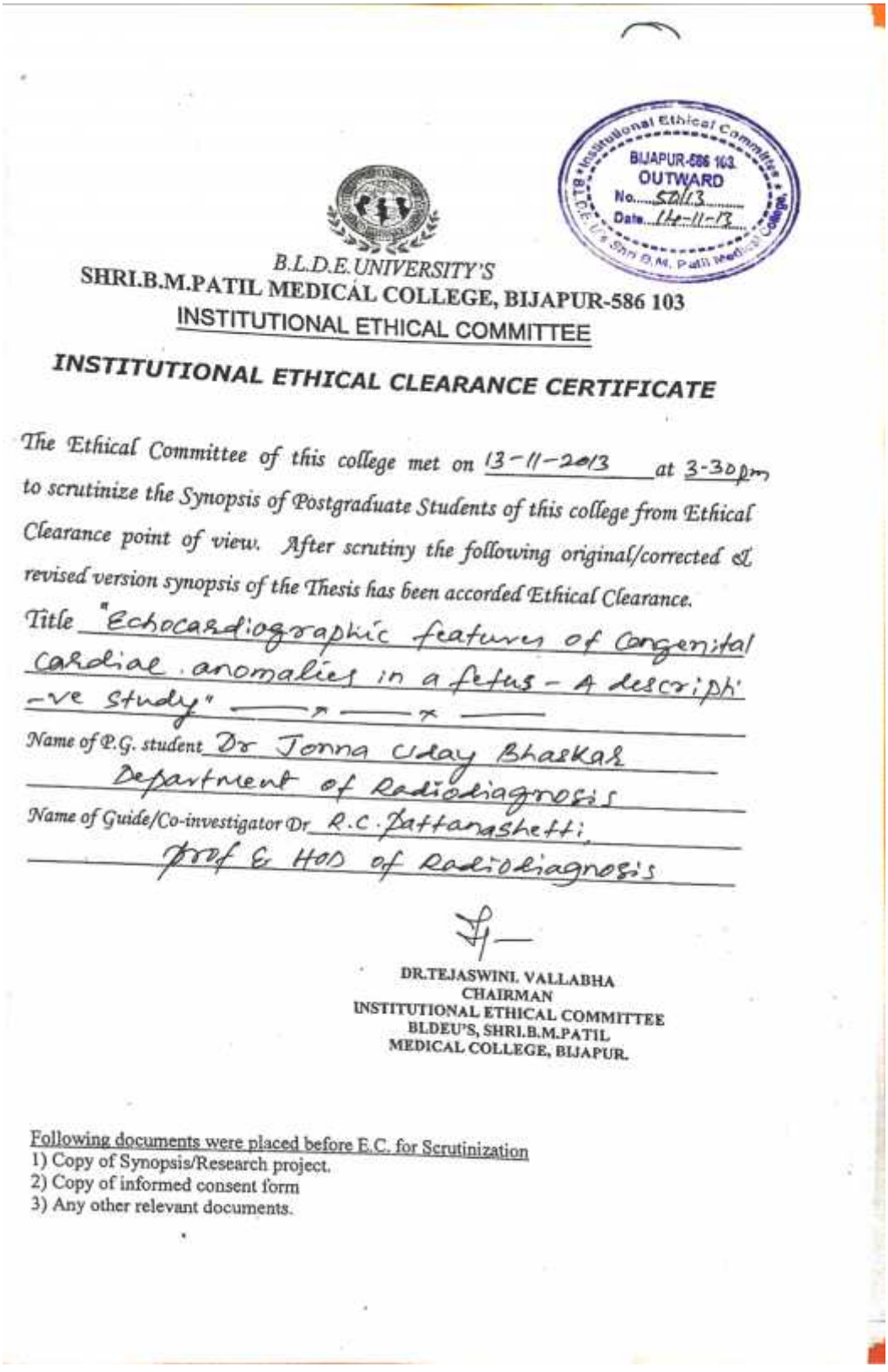
CASE 7

HYPOPLASTIC LEFT VENTRICLE IN RVOT VIEW



ANNEXURE – I

ETHICAL CLEARANCE CERTIFICATE



B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

BIJAPUR-586 103. OUTWARD No. 5213 Date 14-11-13

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2013 at 3-30pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title "Echocardiographic features of Congenital Cardial anomalies in a fetus - A descriptive study"

Name of P.G. student Dr Jonna Uday Bhaskar
Department of Radiodiagnosis

Name of Guide/Co-investigator Dr R.C. Jattanashetti
Prof & HOD of Radiodiagnosis

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

ANNEXURE – II

CASE SHEET PROFORMA

CASE NO:

MATERNAL AGE: **HOSPITAL NUMBER:**

MEAN GESTATIONAL AGE (USG):

EXAMINATION - FETAL ECHOCARDIOGRAPHY

CARDIAC VIEWS	FINDING DETECTED/ UNDETECTED
Four chambered view (4CV)	
Out flow tract views (OTVs)	
Short axis view of the ventricles	
View of the aortic arch (AoA view)	
Three-vessel view (3vv)	

ASSOCIATED ANOMALIES:

ECHOCARDIOGRAPHIC FINDINGS:

ANNEXURE – III

SAMPLE INFORMED CONSENT FORM

B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR – 586103, KARNATAKA

TITLE OF THE PROJECT: ECHOCARDIOGRAPHIC FEATURES OF CONGENITAL CARDIAC ANOMALIES IN A FETUS- A DESCRIPTIVE STUDY.

PRINCIPAL INVESTEGATOR: DR. JONNA UDAY BHASKAR
DEPARTMENT OF RADIO DIAGNOSIS
Email: jonna6@gmail.com

PG GUIDE: DR. R C PATTANASHETTI$_{MD,RD}$
PROFESSOR
DEPARTMENT OF RADIO-DIAGNOSIS
Shri B.M. Patil Medical College & Research Centre, Sholapur Road, VIJAYAPUR - 586103

PURPOSE OF RESEARCH:

I have been informed that this study will evaluate causes of low back pain in young adult patients.

I have been explained about the reason for doing this study and selecting me/my ward as a subject for this study. I have also been given free choice for either being included or not in the study.

PROCEDURE:

I/my ward have been explained that, I/my ward will be subjected to obstetric scans and detailed study of the fetal heart will be done by ultrasonography.

RISKS AND DISCOMFORTS:

I/my ward understand that I/my ward may experience some claustrophobic sensation during the procedure. I/my ward understand that necessary measures will be taken to reduce these complications as and when they arise.

BENEFITS:

I/my ward understand that my participation in this study will help to prenatally screen, allows physicians and families the greatest number of therapeutic options, and can improve the postnatal outcome& decreasing morbidity and mortality.

CONFIDENTIALITY:

I/my ward understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr. Jonna Uday Bhaskar is available to answer my questions or concerns. I/my ward understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me and that a copy of this consent form will be given to me for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I/my ward understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I/my ward also understand that Dr. Jonna Uday Bhaskar will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date: Dr. R.C. Pattanshetti (Guide)

Dr. Jonna Uday Bhaskar (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I/my ward confirm that DR. Jonna Uday Bhaskar has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I/my ward have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

MASTER CHART

Serial no	Age in Years	Gestational age in Weeks	Four Chamber View	Short Axis View of Ventricles	Three vessel view	Outflow Tract View	Aortic Arch View	Cardiac Anomaly	Extracardiac Findings
1	20	24	DETECTED	UNDETECTED	UNDETECTED	UNDETECTED	UNDETECTED	MULTIPLE IEF	NONE
2	32	26	DETECTED	DETECTED	UNDETECTED	DETECTED	UNDETECTED	VSD	NONE
3	27	20	DETECTED	DETECTED	UNDETECTED	DETECTED	UNDETECTED	VSD	OMPHALOCELE
4	16	27	DETECTED	UNDETECTED	UNDETECTED	UNDETECTED	UNDETECTED	MESOCARDIA	None
5	33	24	DETECTED	UNDETECTED	DETECTED	DETECTED	UNDETECTED	TOF	POLYHYDRAMNIOS
6	26	27	DETECTED	UNDETECTED	UNDETECTED	UNDETECTED	UNDETECTED	ASD	None
7	27	27	DETECTED	DETECTED	DETECTED	DETECTED	DETECTED	HLH	CC AGENESIS, HOLOPROSENCEPHALY
8	26	20	UNDETECTED	DETECTED	UNDETECTED	DETECTED	UNDETECTED	VSD	NON-IMMUNE HYDROPS FETALIS
9	28	25	UNDETECTED	UNDETECTED	DETECTED	DETECTED	UNDETECTED	COMPLETE TGA	NONE
10	21	21	DETECTED	DETECTED	UNDETECTED	UNDETECTED	UNDETECTED	DILATED RIGHT VENTRICLE	NONE
11	27	24	UNDETECTED	UNDETECTED	UNDETECTED	DETECTED	UNDETECTED	AORTIC STENOSIS	OLIGOHYDRAMNIOS
12	27	26	DETECTED	UNDETECTED	UNDETECTED	DETECTED	UNDETECTED	AVSD	NONE
13	33	22	DETECTED	UNDETECTED	DETECTED	DETECTED	UNDETECTED	TOF	NONE
14	24	25	DETECTED	UNDETECTED	UNDETECTED	DETECTED	UNDETECTED	AVSD	NONE
15	20	25	DETECTED	UNDETECTED	UNDETECTED	UNDETECTED	UNDETECTED	DILATED RIGHT ATRIUM	NONE
16	18	24	DETECTED	UNDETECTED	UNDETECTED	UNDETECTED	UNDETECTED	DILATED RIGHT ATRIUM	NONE
17	33	22	DETECTED	DETECTED	UNDETECTED	DETECTED	UNDETECTED	VSD	OLIGOHYDRAMNIOS

18	33	20	DETECTED	UNDETECTED	UNDETECTED	DETECTED	UNDETECTED	AVSD	INCREASED NT
19	34	25	DETECTED	UNDETECTED	DETECTED	DETECTED	UNDETECTED	TOF	NONE
20	27	24	DETECTED	UNDETECTED	UNDETECTED	UNDETECTED	UNDETECTED	ASD	NONE
21	32	24	UNDETECTED	UNDETECTED	DETECTED	DETECTED	UNDETECTED	COMPLETE TGA	NON-IMMUNE HYDROPS FETALIS
22	31	25	UNDETECTED	DETECTED	UNDETECTED	DETECTED	UNDETECTED	VSD	POLYHYDRAMNIOS
23	36	21	DETECTED	DETECTED	UNDETECTED	UNDETECTED	UNDETECTED	ASD	INCREASED NT
24	34	27	UNDETECTED	UNDETECTED	UNDETECTED	DETECTED	UNDETECTED	AORTIC STENOSIS	None
25	23	28	DETECTED	DETECTED	DETECTED	DETECTED	DETECTED	HLH	CC AGENESIS
26	26	21	DETECTED	UNDETECTED	UNDETECTED	DETECTED	UNDETECTED	DORV WITH VSD	NONE
27	26	19	DETECTED	UNDETECTED	UNDETECTED	DETECTED	UNDETECTED	AVSD	NONE
28	22	18	DETECTED	UNDETECTED	UNDETECTED	DETECTED	UNDETECTED	DORV	NONE
29	21	26	DETECTED	UNDETECTED	UNDETECTED	UNDETECTED	UNDETECTED	INCREASED LEVOCARDIA	NONE
30	25	25	DETECTED	UNDETECTED	UNDETECTED	DETECTED	UNDETECTED	DORV WITH VSD	HYDROCEPHALUS
31	32	27	DETECTED	DETECTED	UNDETECTED	UNDETECTED	UNDETECTED	HRH	None
32	31	24	DETECTED	UNDETECTED	UNDETECTED	DETECTED	UNDETECTED	AVSD	NON-IMMUNE HYDROPS FETALIS
33	31	24	DETECTED	DETECTED	UNDETECTED	DETECTED	UNDETECTED	VSD	None