Ventricular Septal Defect and its Hemodynamic Correlates *

By

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Ventricular septal defect is by far the commonest Congenital Cardiac malformation Comprising 20-25 percent of all Children with Congenital heart disease (1). Small defects measuring less than 5 m.m. in diameter Cause little hemodynamic changes, however, large defects greater than 1 cm in diameter may produce congestive .cardiac failure and marked reduction in growth during infancy (2).

The purpose of this report is to present hemodynamic, angiocardiographic and echo-cardiographic correlates; and to discuss medical and surgical management of patients with ventricular septal defect at the NICVD with a view to evolve a rational policy of management which would provide optimum Care of these children within the frame work of our developing Pediatric medical and surgical cardiac services.

METERIAL OF METHODS.

Forty one patients, age 2.2-18 years, who presented at the NICVD during 1980-1982 period were included in the study. (Table I) All of these patients had been studied by cardiac Catheterization. This of necessity invoked patient selection since infants under 2 years of age were not studied. Furthermore cardiac catheterization was only undertaken when the defect was considered to be moderate or large and associated with some degree of pulmonary arterial hypertension. Thus the study was biased towards larger defects. Diagnostic work up included clinical examination, Electrocardiography. chest roentgenogram, M-mode and Two dimensional real time sector scan echocardiography (SSE).

Echocardiographic examination was done in supine position. Single crystal M-mode echocardiograms were obtained by using commercially available Ekoline Echocardiograph. The recordings were obtained on light sensitive strip chart or pallaroid film. Assesment of left to right shunt was made by determining the LA/AO ratioand a ratio, greater than 1.25 suggested significant shunt (3). Pulmonary arterial pressure was evaluated by PEP/RVET ratio. A value $\succ 0.28$ and abnormal right ventricular wall thickness suggested an elevated pulmonary arterial pressure (4).

Two Dimensional real time sector scan echocardiography was performed using an A.T.L. (Advanced technology Laboratories) echocardiograph, A 3.25 M HZ transducer was used to image the ventricular septum from various locations by utilising different planner views (5,6). The images were video-taped and hard copies of the stop frame image were obtained using 130 A-Line scan recorder.

* From the NICVD, Karachi.

The diameter of ventricular septal defect (VSD) image was measured in various views obtained from different locations in Systole and diastole and largest diameter measurment was used for correlations with hemodynamic data.

Cardiac Catheterization studies were done after 4-6 Hour fasting in infants and over night fasting in older childrens. Pethidine, (25mg) cholorpromazine (6.25mg) and phenergan (6.25mg) mixture was used as a preme dication, in the dose of IML/151b body weight to a maximum of 2 ML The dose was administered intra muscularly 45 min to one hour prior to catheterization. Pressure measurements were made by using P 23 ID statham Gauge pressure transducer and E for M (Electronics for Medicine) recorder. The records of pressure tracings were obtained on a strip chart. All studies were done by percutaneus technique. Right heart pressures including pulmonary arterial wedge pressure, were obtained. Left atrial, left ventricular and aortic pressures were obtained with the venous catheter via a patent Foramen ovale or through V.S.D. If left heart could not be so entered systemic pressure and saturation were obtained by Femoral arterial puncuture. Retrograde left heart catheterization was performed when V.S.D. could not be visualised on levophase or there was a suspicion of a supracristal defect. oxygen saturations were determined by an American optical oximeter.

Angiograms were obtained by injecting 1.5ml of urografin dye through an NIH catheter with side holes. On angiogram, the size of ventricular septal defect was measured and corrected for X-Ray magni fication by using Catheter image (7).. Axial angiographic Technique was used to image the ventricular Septum. The most common view to image ventricular Septum was the elongated right anterior oblique projection. Patient's right Shoulder was elevated by 15-20°. and the patient was moved 20-25° in cranio Caudal Oxis. Biplane cine angiograms were obtained. (8). Lateral view cine angiograms using horizontal X-Ray beam showed the limits of an anteriorly located ventricular septal defect.

Puomonary blood flow was calculated by assuming oxygen consumption value of 180 ML/min/M² for all studies. (9).

Oxygen content was calculated by multiplying the oxygen saturation value with 1.36ml.

Pulmonary and systemic blood flows were calculated by using Ficks principle;

$$CO/M^2 = \frac{VO2\ ML/MIN/M^2}{A\text{-}V\ O2\ Content\ (L)}$$

Where CO= Cardiac out put, A= arterial Oxygen content, V= Venous Oxygen conent, Vo2=oxygen consumption ML/min/M².

Pulmonary vascular resistence was estimated, by the equation;

$$\frac{PAP\text{-}LAP}{QP} = u/M^2$$

Where LAP=left atrial mean pressure, QP=Pulmonary blood flow L/min/M².

PAP=mean pulmonary arterial pressure systemic resistence was;

$$\frac{AOP-RAP}{QS} = u/M^2$$

were AOP=mean Aortic pressure RAP=mean Right atrial pressure, QS=systemic blood flow L/min/M². Left to right shunt was expressed

as QP/QS ratio. In patents with bidirectional shunting, effective pulmonary blood flow (QE) was calculated by the equation,

$$\frac{V\,O2}{PVc - SVc}$$

where VO_2=Oxygen Consumption ML/min/M^2 PVc= oxygen content of pulmonary Venous Blood, SVC=Superior vena caval oxygen content. The magnitude of L-R shunt was then QP-QE and right to left shunt QS-QE.

RESULTS

Forty one Patients age 2.2-18 years were included in the study. seven (17%) had supracristal or sub pulmonary ventricular septal defect, (fig 1), four (9.7%) had defect in the trabecular septum (muscular) one of these had 2 defect in the muscular septum. (fig 2). The largest number of patients, 30/41 (73%), had membranous or perimembranous ventricular-septal defect (Fig. 3, 4, 5, 7).

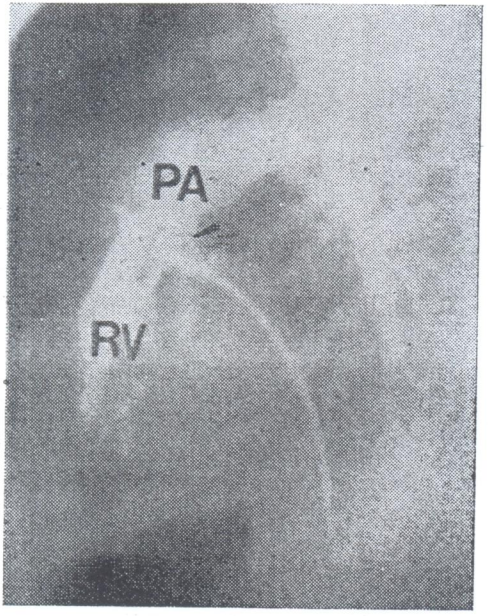

Fig. 1*A*

Right ventricular (RV) cine angiogram (lateral view) in a patient with subpulmonary ventricular septal defect. Ventricular septal defect is located below the pulmonic vlave (Arrow) AP = Pulmonary artery.

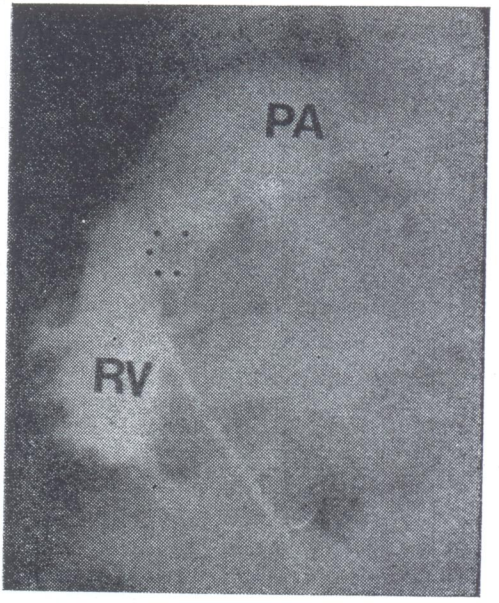

Fig. 1*B*

Right ventricular cine angiogram of patient in panel A. A circular filling defect (dots) in the right ventricular outflow tract is due to prolapsing aortic valve leaflet.

Clinically, early diastolic murmur of Aortic insufficency was present in 4/7 patients with supracirstal defects. Echocardiographic diagnosis of a ventricular septal defect was made prospectivily in all 19/41 patients who

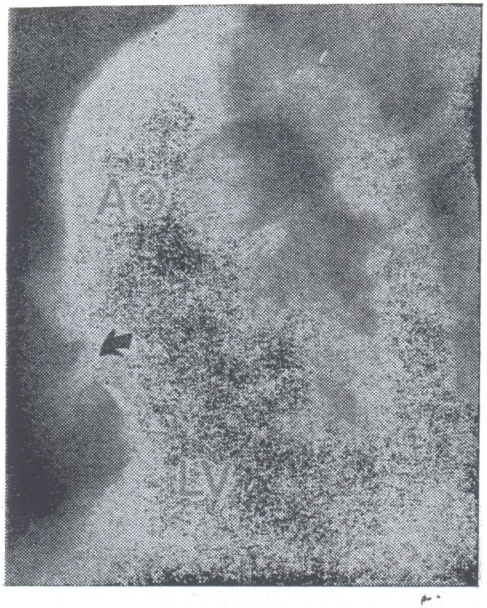

Fig. 1C

Left ventricular (LV) cineangiogram of a patient showing subpulmonary ventricular septal defect (arrow). Anterior aortic root overhangs above the ventricular septum. Ao = Aorta.

had 2-DSSE. There were no false positive diagnosis. Membranous or perimembranous defects were more often imaged from the left parastesnal location in an obligue left ventricular long axis plannar view Fig. 4A. Once a defect was imaged from one location imaging was attempted from all other possible locations such as apical, subxiphoid (Fig 4B) and high parasternal sites. Supracristal ventricular septal defect could be imaged on m-mode echocardiogram as an interrupted septal echo space bellow the aortic root and right aortic coronary leaflet was imaged as an anomalous echo in the right ventricular outflow tract (10).

On SSE Supracristal defect could be seen from parasternal long axis and subxiphoid short axis (sagittal planner) views(12). Fig. 6.

In left ventricular long axis view obtained from the left para sternal location the characteristic features of supracristal defect were (a) enlarged aortic root over hanging the ventricular septum: (b) Anterior aortic leaflet cusp located within the defect or at times prolapsing into the right ventricular out flow tract (10).

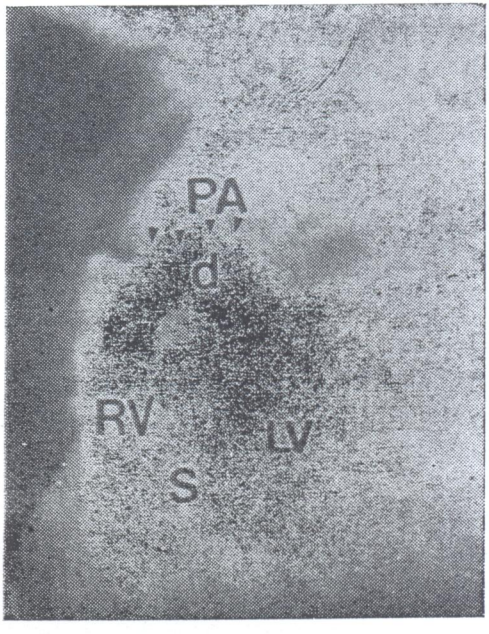

Fig. 1D

Right Ventricular (RV) cineangioram, Lateral view in the a patient with Supra cristal ventricular septal defect (d) which is located above the Crest (C) of the ventricular septum (S) and bellow the pulmonic Valve. (arrow) L.V. = Left Ventricle.

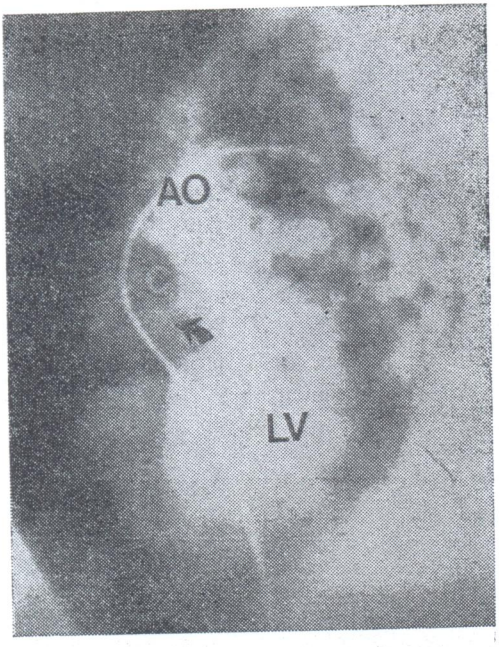

Fig. 2A

Lateral view left ventricular (L.V.) angiogram in an extended right Ventricular Oblique plane. Moderate Size high muscular Ventricular septal defect is seen (arrow). AO = Aorta, L.V. = Left Ventricle, S = Septum, C = Crista Supraventricularis.

Quantitation of the size of the ventricular septal defect showed that (VSD)i mage from any location was larger in diastole. The validation of echocardiographic method of measuring the size of V.S.D. was done by correlating the size of the defect by SSE with that measured by the angiographic technique. There was a highly significant linear correlation (n=13, r=0.88 (Y=0.817 X + 1.42) Fig. 8). Angiographic and echographic size correlation was not done for supracristal defects since the defect was blocked by the prolapsing aortic leaflet in most of the angiographic views. (Fig. 1C).

Cardiac catheterization data. Patients were separated in two Groups based on the level of pulmonary vascular resistance (Table I (Fig 9-10). Group I contained 29/41 patients in whom the PVR was normal i.e. less than 4 units/M^2 (mean = 1.61 + 0.66). Age was 7.4+4.11 years and the QP/QS ratio in 11/29 was $>2:1$ (2.98 + 0.9) and $<2:1$ (1.44+0.8) in 18/29. In three patients QP/QS ratio was $<1.5:1$. Group I was further subvided to Group IA and IB. Group IA

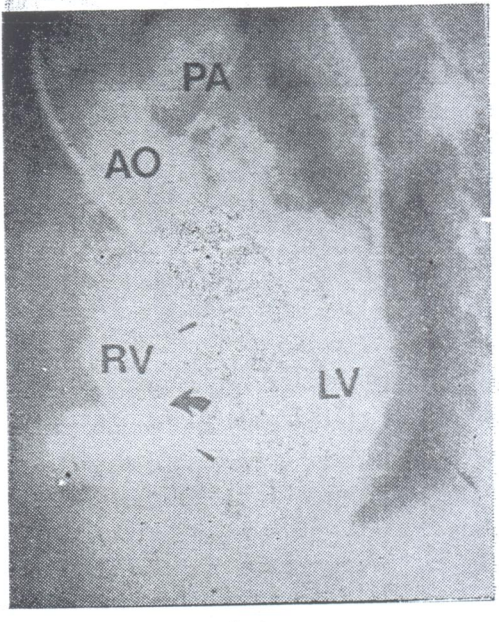

Fig. 2B

Left ventricular cine-angiogram in a lateral view of an extended Right Ventricular oblique plane in a patient with mid muscular septal defect (arrow).

A = Aorta, RV = Right Ventricular.

Table I: Hemodynamic data of 41 Patients with Ventricular septal defect.

GROUP	*No.*	*PAP(m) mmHg*	*PAP(S) mmHg*	*QP/QS*	*RVP/SP*	*P.V.R u/M²*
Group I A	13	16.1± 3.4	27.0± 5.0	1.60± 0.36	0.23± 0.05	1.46± 0.61
Group I B	16	37.5± 11.2	55.0± 13.8	2.98± 0.92	0.42± 0.14	1.78± 0.70
Group II	12	71.0± 15.0	94.0± 17.1	1.98± 0.8	0.87± 0.11	10.8± 8.7

PAP = Pulmonary arterial pressure.
m = Mean, S = Systolic QP/QS = Pulmonary blood flow/Systemic Blood flow ratio.
RVP = Right Ventricular pressure.
PVR M^2 = Pulmonary Vascular resistence per $Meter^2$ ± one standard deviation.
SP = Systemic pressure.

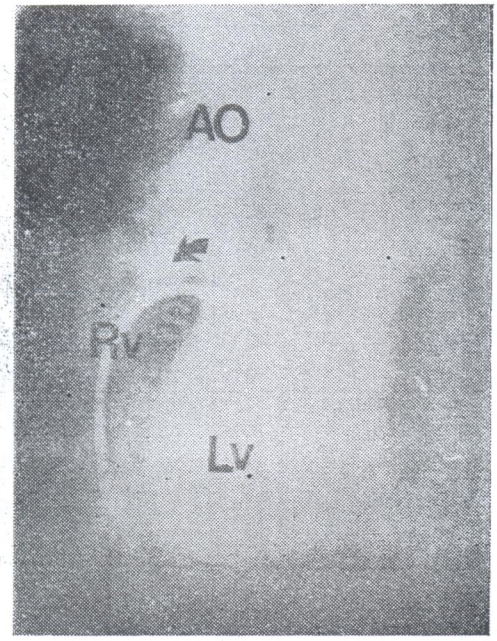

Fig. 3A

Left Ventricular cine angiogram on extended RV oblique plane (Lateral View). Note a large Subaortic (Ao) Ventricular Septal defect (arrow).

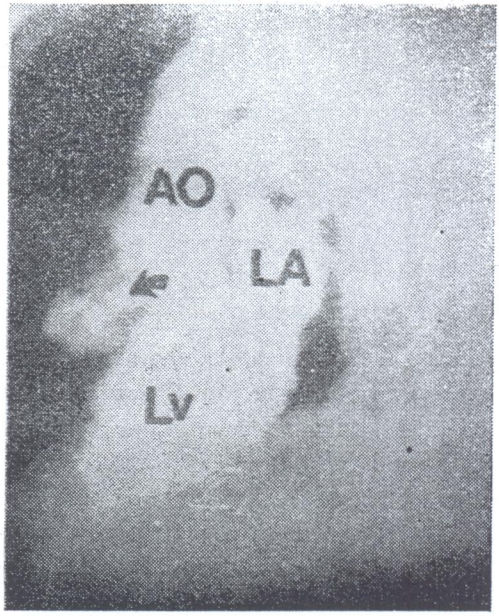

Fig. 3B

Subaortic ventricular septal defect with aneurysm formation is seen (arrow). LA = Left Atrium, LV = Left Ventricle S = Septum, RV = Right Ventricle.

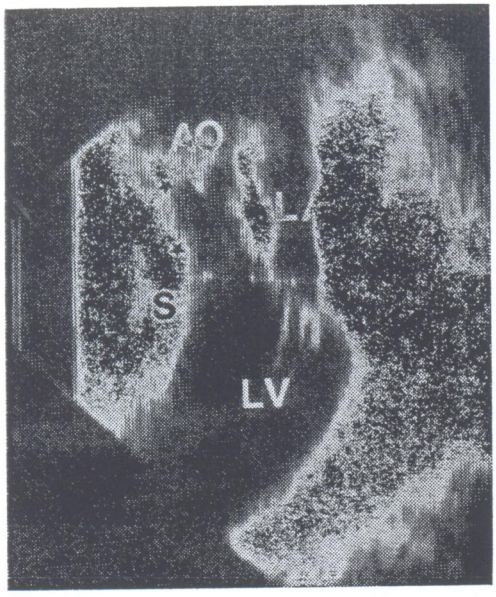

Fig. 4A

Long axis two dimensional Sector scan View obtained at left Parasternal Location. A moderate size ventricular septal defect is imaged (Stars) bellow the aortic root (AO) The defect is Covered by the tricuspid valve (arrow).
LV = Left Ventricle.

contained 13/29 patients with normal pulmonary artrial pressure (<25mmHg mean) All but 2 patients in Group I A had QP/QS ratio was <2.1 the mean for the Group was (1.4+ 0.28). The RVP/SP ratio was 0.23+0.05. Group IB had 16 patients with mean pulmonary arterial pressure greater than 25 mm Hg. The QP/QS ratio for the group was 2.42+0.14 and the RVP/SP ratio was 0.42+0.14.

Group 11 contained 12 patients, age 5.7 + 2.6 years, in whom mean pulmonary arterial pressure was elevated i.e. greater than 25mm Hg (71.0+8.7) and the RVP/SP ratio was 0.87±0.11 Table I. In 8/12 patients the QP/QS ratio was >2:1 and PVR <8 units. Three of the twelve patients had QP/QS ratio <2:1 and Bidirectional shunting with markedly elevated PVR (>20 units). One remaining patient had QP/QS ratio of 1.4 and PVR of 7.4 units. Peak Systolic pulmonary arterial pressure for Group II was 94±17 mmHg and mean systemic pressure was 71±15 Hg Fig. 10.

In 29/41 patients V.S.D. diamater measurements were obtained from echocardiograms and from angiograms when echoes were not avai..b... The V.S.D. diameter showed a

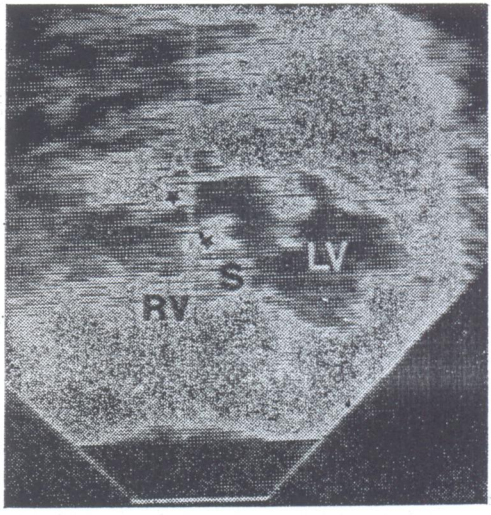

Fig. 4B

Long axis two dimensional secter scan view Obtained from subxiphoid location in another patient. Moderately large Sub Aortic ventricular septal defect is imaged between the Stars. RV = right Ventricle, S=Septum, L.V.= left Ventricle.

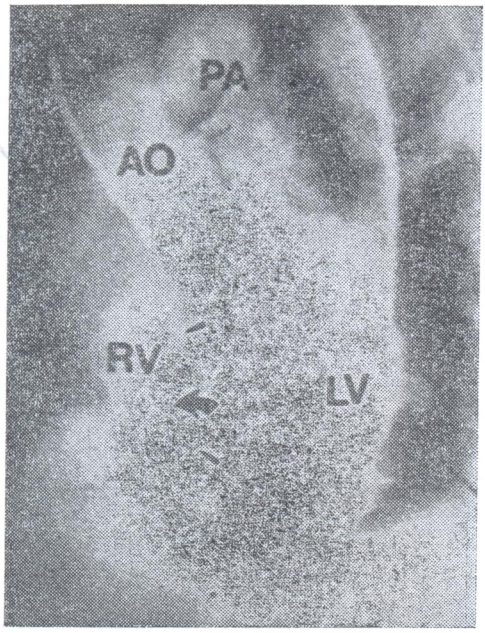

Fig. 5A

Left ventricular cineangiogram (Lateral view) Obtained in extended Right ventricular Oblique plane. Note a large mid muscular ventricular septal defect with some Opacification of the right Ventricle in a patient with clinical cyanosis and pulmonary vascular Resistance of ≥ 20 units/M^2.

linear correlation with the peak Systolic pulmonary arterial pressure (r=0.77, Y=7.587 X +4.11) Fig. 11.

Further more V.S.D. diameter was also linearly related to the RVP/SP ratio (n—27, r=0.76:, Y=0.062 X+0.068). Fig 12.

In patients with normal pulmonary arterial pressure V.S.D. diameter was 4.26+1.1 and was statistically larger ($P < .0.001$) than with moderatly elevated PAP (V.S.D. diameter 8.2±1.48). The patients with near systemic level PAP had ventricular Septal defect diameter of 10.2±2.6, which was larger compared to those with moderatly elevated PAP ($P < 0.10$) although at a lower degree of statistical significance. Table II.

The group with normal RVP/SP ratio (< 0.3) showed a significantly smaller V.S.D diameter (4.6 ± 1.4) compared to those with moderately elevated ratio (0.46+0.05) and V.S.D diameter of 7.79+2.0 mm.). ($P < 0.01$) The V.S.D. diameter in patients with near systemic pressure right ventricle (RVP/SP ratio 0.82 ± 0.14) was signigicantly larger (10.2 ± 2.8mm) compared with V.S.D. diameter associated with moderately elevated RVP/SP ratio ($P < 0.01$) Table III.

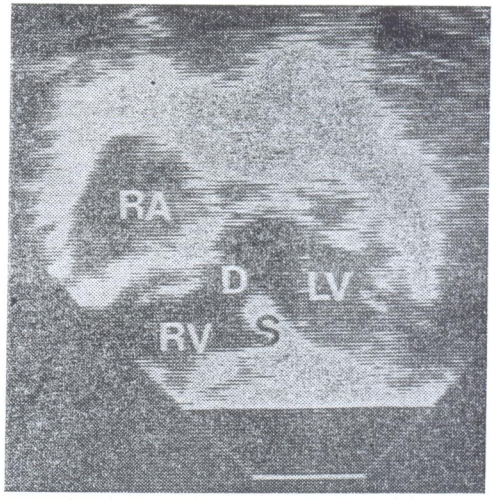

Fig. 5B

Correlative left ventricular long axis oblique view Sector scan obtained from subxiphiod location in the same patient as in Pannel A. A large defect is seen in the muscular Septum.

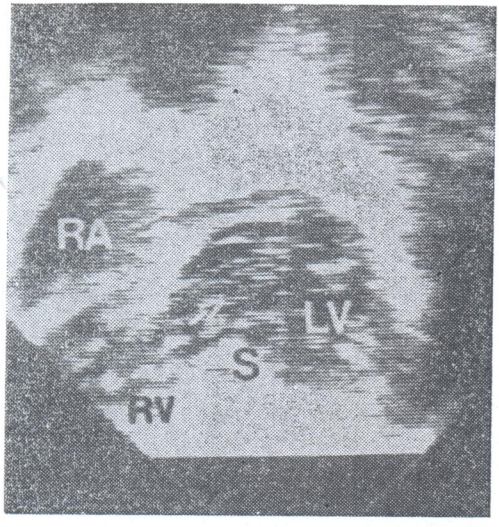

Fig. 5C

Four chamber oblique view sector scan from subxiphoid location in the same patient. Ten ml of Saline is injected in the peripheral vein. Clouds of echoes appear in the right Ventricle and are shunted into the left ventricle (arrow) through the ventricular Septal defect.

Table II. Haemodynamic Correlates of Ventricular Septal Defect.

PAP. mm HG	*No.*	*V.S.D. Diameter mm*	*P. Value*
≤35			
28.8± 5.0	6	4.26± 1.10	
36—60			<0.001
51.6± 9.7	8	8.2± 1.48	
>61			
93.7± 17.3	15	10.2± 2.6	<0.10

PAP = Peak Systolic pulmonary arterial pressure; V.S.D. = Ventricular Septal defect; ± = One Standard Deviation; < = Less than; > = Greater than.

Table III. Haemodynamic Correlates of Ventricular Septal Defect.

RVP/SP	*No.*	*VSD. Diameter m.m.*	*P. Value*
<0.3			
0.26± 0.06	7	4.6± 1.4	
0.31—0.5			<0.01
0.46± 0.05	7	7.97 2.0±	
>0.51			
0.82± 0.14	13	10.2± 2.8	<0.01

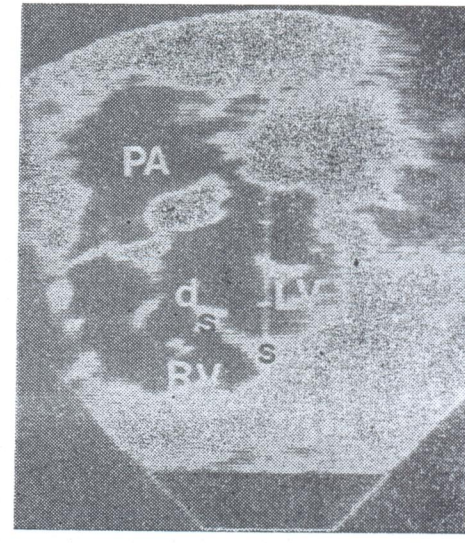

Fig. 5D

Two dimensional sector Scan Obtained from subxiphoid Location in Sagittal plane. Large muscular defect (d) is covered on the right ventricular aspect (RV) by the tricuspid valve. S = Septum, LV = left Ventricle, PA= Pulmonary artery.

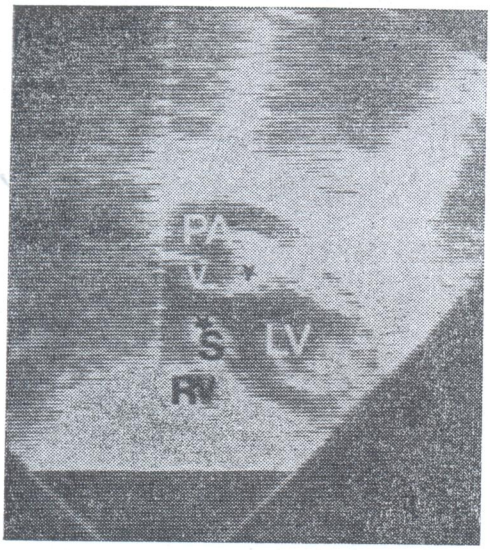

Fig. 6

Subxiphoid Sector scan view in sagittal plane in a patient with subpulmonary ventricular septal defect. Large defect is present (between the stars) bellow the pulmonic valve (V) and the crest of the ventricular septum "S" marks the lower limit of the defect. LV = Left Ventricle, RV = right ventricle.

Patients in whom QP/QS ratio was $<2:1$, V.S.D diameter was 5.4.$\pm$1.6 mm significantly lower ($P<0.001$) than those with QP/QS ratio greater than 2:1 (9$\pm$2.9 mm) (Table IV).

Table IV. Haemodynamic Correlates of Ventricular Septal Defect.

wP/QS	*No.*	*VSD Diameter mm*	*P. Value*
$<2:1$			
1.68$\pm$ 0.69	9	5.35$\pm$ 1.60	
$>2:1$			<0.001
2.83$\pm$ 0.80	16	9.69$\pm$ 2.89	

Pulmonary vascular resistence plotted against age showed that abnormally elevated, resistence had developed even at 2.2 year age and in significant number between 2.2—6 years age. Fig. 13.

Cardiac Surgery:

Fourteen of 29 patients in group I had surgery. Of the remaining 15 patients, 9 with QP/QS >2.1 were awaiting surgery, and six with a small defect did not require surgery. Thirteen had primary closure of ventricular septal defect; suture closure with proline in five and closure with Dacron patch in 8. surgical closure of ventricular septal defect was achieved on Cardipulmonary bypass using deep Hypothermia and Cardioplegia. There was no surgical death in this group. Four of these 13 patients had supracristal ventricular

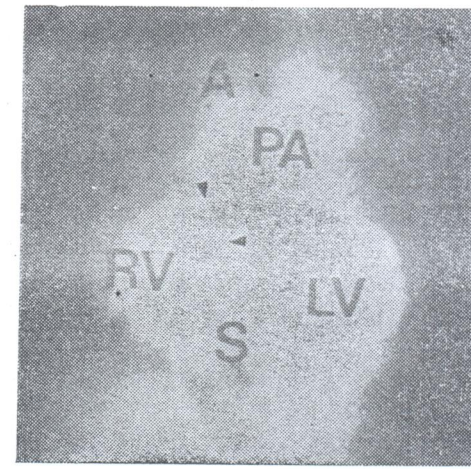

Fig. 7*A*

Left anterior oblique view of the left ventricular cine angiogram shows a moderate size Subaortic ventricular septal defect (arrows).

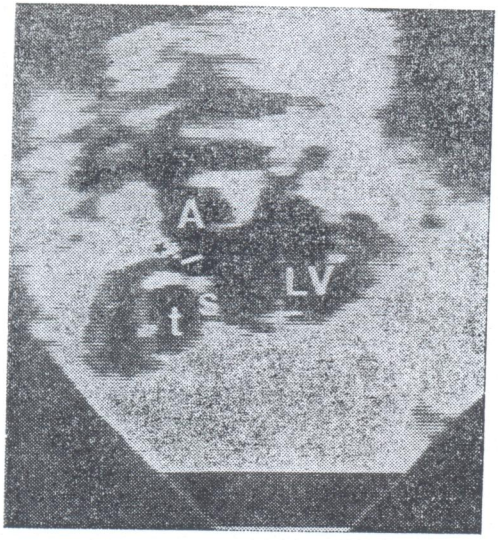

Fig. 7B

Correlative two dimensional sector Scan in left ventricular longaxis shows the position of the sub aortic ventricular septal defect. (starr & arrow).

t= tricuspid valve S= Septum L.V. = Left Ventricle.

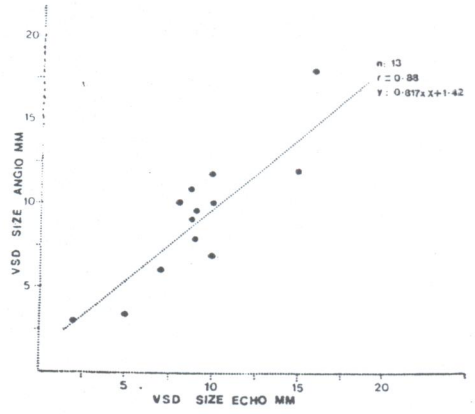

Fig. 8

Ventricular septal defect (V.S.D.) diameter measured by 2D sector scan echocardiography and V.S.D. diameter measured by angiography shows statistically significant linear correlation in 13 patients.

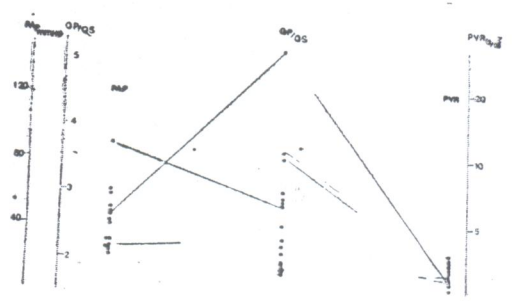

Fig. 9

Hamodynamic data of Group I patients. who had pulmonary vascular resistence (PVR) value than $\succ 5$ units/M^2. The QP/QS ratio varied as did the systolic pulmonary arterial pressure (PAP).

Septal defect and prolapsing Aortic right Coronary leaflet. Two of these continued to have moderate aortic regurgitation post operatively. The V.S.D. was closed with Dacron patch in 2 and suture closure was obtained in other two; the aortic valve was not repaired in any.

One of 13 patient's in Group I had 2 defects in the ventricular septum which were closed with Decron patch. Eight patients had membranous V.S.D. Three had Suture closue and 5 required a decron patch. The remaining one patient in Group I had a large left to right Shunt and pulmonary artery hypertension and underwent pulmonary artery banding operation with reduction in distal pulmonary arterial pressure to within normal limits and remains well.

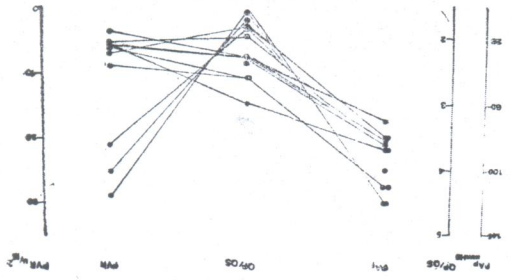

Fig. 10

Hemodynamic data of Group II patients. The pulmonary vascular resistance (PVR) was elevated $\succ 4$ units. All had pulmonary arterial hypertension i.e. systolic pulmonary arterial pressure (PAP) greater than 35 mmHg The QP/QS ratio varied.

Note: Eisenmenger reaction was present in 3 patients ie PVR greater than 10 units/M^2.

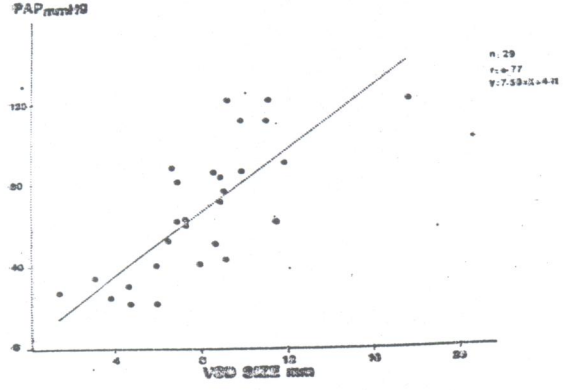

Fig. 11

Ventricular septal defect (V.S.D.) diameter determined either Echocardiographically or by angiography showed a statistically significant linear correlation to the systolic pulmonary artery pressure (PAP); (r = 0.77) in 29 patients.

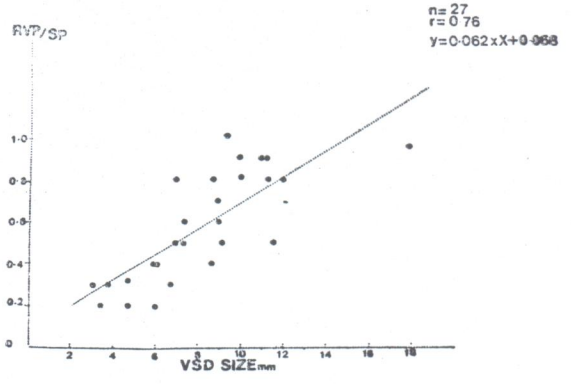

Fig. 12

Ventricular septal defect diameter showed statisfically significant linear relation to the Right Ventricular systolic pressure/ systemic pressure ratio (RVP/SP) in 27 patients.

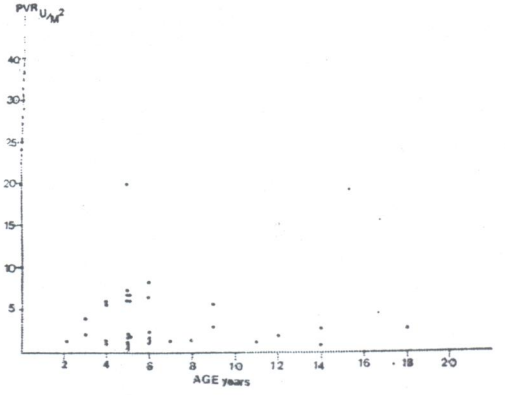

Fig. 13

Pulmonary vascular reistence (PVR/U/M^2) was plotted against age. Patient's less than 2 years age were not studied. A significant number of patient's between age three and six years showed elevated pulmonary vascular resistence (PVR) ie greater than $\succ 4$ units, suggesting that large defects with pulmonary artery hypertension should undergo VSD closure within 2 years of age.

Six patients in group II had surgery, four underwent primary closure of V.S.D. with Dacron patch and two patients had pulmonary arterial banding operation, One of these two had a relatively smaller right ventricle and severe pulmonary arterial hypertension, and the other had Aortic override above the ventricular Septum (DORV type). Both are now free of Congestive cardiac failure. There was no surgical death this group. Two of six remaining patients were inoperable, due to development of clinical cyanosis and PVR $\succ$ 20 units/M^2. Four are awaiting surgery.

DISCUSSION

Our study and the reported experience shows that defects in ventricular septum can be confidently imaged by 2 dimensional sector scan echocardiography. (11, 12) Our study validates the V.S.D. Size determination by echocardiography by showing that ventricular septal defect diameter determined by echocardiography was Comparable to that determined by axial angiography.

The most important determinant of hemodynamic and physiologic effects of ventricular septal defect is its size (13). Smaller defects are said to be 1-2mm in diameter but less less than 0.5cm and Large defects to be greater than Icm in diameter. Hemodynamicaly Small defects are associated with QP/QS ratio of $\prec$1.5:1 and normal PAP. Moderate size defects are associated with QP/QS ratio $\succ$ 2:1, modest increase in pulmonary artery pressure, and RVP to systemic artery pressure ratio RVP/SP of (0.3-0.7) Large defects are associated with 2/3 systemic leval pulmonary arterial pressure and RVP/SP ratio of $\succ$ 0.75 (14) These relationships are approximations and hemodynamics and V.S.D. size correlates have not been firmly established in vivo. 2D-echocardiography allows for accurate determination of V.S.D. size and our study Confirms that V.S.D. size can be measured by echocardiography with accuracy comparable to the angiographic technique. ventricular Septal defect diameter and hemodyanamic correlations suggest that the pulmonary arterial pressure, QP/QS ratio and RVP/SP ratio can be evaluated by determining the size of the ventricular septal defect i.e. defects $\prec 0.46 \pm 1.1$ were associated with normal PAP, RVP/SP ratio ($\prec$0.3) and QP/QS $\prec$1.5. Defects with PAP $\succ$ 61.mm Hg and RVP/SP ratio $\succ$ 0.57 were larger than 10.2 mm in diameter. In our study small defects with QP/QS ratio $\prec$1.5 were few. Therefore the V.S.D. size relationships to pulmonary arterial pressure and QP/QS ratio need to be elucidated further for Smaller defects.

Since ventricular septal defects show great variations in shape and location on the ventricular septum, (15) Echocardiographic imaging of the defect should be obtained from all possible locations in order to determin the largest diameter. Restriction of V.S.D. imaging to only one location, in our experience, does not accurately define the limits of the defect. Further more axial angiography allows for defining the size limits of V.S.D. with greater accurracy than before (16, 17). Therefore echo and angiographic correlations can be he varified with greater degree of certainty.

Our study population is small nonetheless it demonstrates that echocardiographic deter-

mination of V.S.D. diameter allows for the seperation of hemodynamically small, moderate and large ventricular septal defects in patients older than 2 years age. Quantitative M-mode-echocardiographic predictors of PAP such as PEP/RVET (4) ratio and right vantricular wall thickness Combined with V.S.D. diameter and SSE should help evaluate the hemodynamic consequences in each case. Great advantage of this non invasive echocardiographic evaluation is that longitudinal follow up of V.S.D. size and its hemodynamic carrelates can be undertaken with greater surity and frequency.

In our study incidence of supracristal or subpulmonary V.S.D. was higher than reported for European races (18, 19, 20). This may not be all due to greater detection rate of supracristal V.S.D. by echocardiography or due to a greater bias toward undertaking catheterization studies in these patients. In Oriental races the incidence of supracristal VSD is greater than reported for westren population (18 20). In Asian's the incidence of supracristal V.S.D. is reported to be 10%; an incidence closer to the orientals (21) and strongly suggests a genetic basis. For predilection of supracristal defects in Asians and Oriental races. We plan to further elucidate this with larger series of patients.

Management of Ventricular Septal Defect:

Our present policy regarding management of ventricular septal defect is based on the following guidelines. It is extreamly uncommon for the Eisenmenger reaction to develop under two years age (22). Therefore we tend to manage babies with medical means till 2 years age, provided growth is not severely Compromised. The babies in whom congestive heart failure is unremitting may have to be treated surgically at an early age. our present surgical and nursing Capbilities do not allow us to close the V.S.D. during infancy, which requires the use of candiopulmonary by pass technique, however pulmonary arterial Banding can be undertaken in our present set up. This policy of course would change with aquisition of greater expertise in handling small infants. This change in policy necessitates a major reordering of priorities in a general Hospital setting quite appart from the need to train specialised nurses and para amedical personnel. We have undertaken primary closure of V.S.D. at 13—15 Kg weight without Surgical death. Pulmonary arterial banding operation has been undertaken for anatomic constraints and age.

It has been pointed out that closure of large V.S.D, with pulmonary artry pressure near systemic, should he undertaken within 2 year of age in order to avoid the development of obstructive pulmonary vacular disease (PVD) (19, 22). The reported experience suggest that the surgical mortality is greatly increased with increasing degree of P.V.D. (9) Our data shows that by 4-5 year of age significant number of our patients with large V.S.D. showed elevated level of pulmonary vascular resistence. We are expected to encounter large number of such patients over the ensuing years since many of the infant survivors with large V.S.D. would be expected to have developed a degree of Pulmonary vascular obstructive disease It is greatly hoped that as the services for managing children with Congenital cardiac defects increase in our part of the world

development of Eisenmneger reaction would became a rarity. This would require careful evaluation and follow up of infants through infancy and surgical closure and palliation at apporpriate time.

Large V.S.D. in a child over the age of 2 years can be safely closed at our institution and therefore surgical closure should not be undualy delayed otherwise obstructive pulmonary vascular disease would develop and make the surgery hazzardous. Or may lead to inoperability. In summary our study shows that management of V.S.D. can be based on 2D-echocardiography in concert with other traditional means such as Radiology and electrocardiography. That surgery for Large V.S.D. can be undertaken with relative safety after 2 years of age. It is appreciated that earlier closure of V.S.D. may be desirable in some instances and it is expected that with further development of medical, nursing, paramedical and surgical services at the N.I.C.V.D. This would become feasible in the near future.

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