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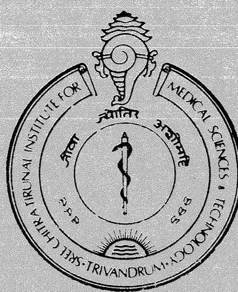

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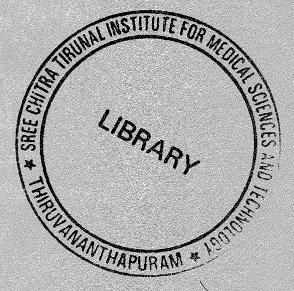
PROJECT REPORT



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NAME : Dr. J. S. Bhuvaneswaran

PROGRAMME : D.M. Cardiology

MONTH AND YEAR OF SUBMISSION : November 1993

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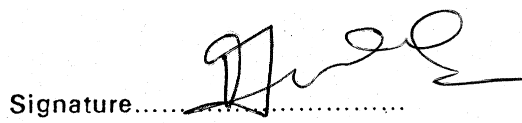
CERTIFICATE

I, Dr. J. S. BHUVANESWARAN hereby declare that I have actually performed all the procedures listed/carried out the project under report.



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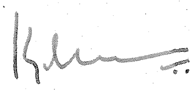
Date : 11-11-1993.

Name in capital letters DR J.S. BHUVANESWARAN

Forwarded. He has carried out the minimum requirement of procedures / etc.



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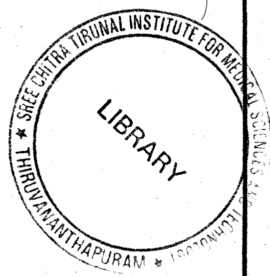

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PROJECT REPORT

TITLE OF THE PROJECT: PREDICTION OF MEAN PULMONARY ARTERY PRESSURE IN PATIENTS WITH PULMONARY ARTERIAL HYPERTENSION USING PULMONARY REGURGITATION FLOW SIGNAL BY DOPPLER.

NAME........DR. J.S.BHUVANESWARAN........

PROGRAMME : D.M. CARDIOLOGY........

MONTH & YEAR OF SUBMISSION : DECEMBER 1993.

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM 695 011

Name	Bhuvaneswaran
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Date	

Note:— (i) In the case compilation of procedures done, the contents and the subsequent pages should be made into different sections (a) Procedures done (b) Procedures assisted (c) Procedures participated (d) Procedures attended / participated etc. in Other Centres. Each section should be preceded by a leaf carrying the name of the section that is succeeding.

(ii) The Contents page will carry information as per model given under

PROCEDURES DONE

Closed Mitral valvotomy ------------------------------------- 124 (say)

Patent ductus arteriosus-ligation 10

Atrial septal defects.....................................20

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....

PROCEDURES ASSISTED

Closed Mitral valvotomy ------------------------------------- 100 (say)

....

(iii) In the subsequent pages details of each procedure done/assisted should be given in the format given below :—

Heading : Closed mitral valvotomy

Date	Name of the patient	Age	Sex	Patient No.

(iv) In the case of Project Report in the page immediately following the Certificate page the under-mentioned details should be given :—

(a) Title

(b) Duration

(c) Aim and scope

(d) 50 word summary of work done

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2. INTRODUCTION
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PREDICTION OF MEAN PULMONARY ARTERY PRESSURE IN PATIENTS WITH PULMONARY ARTERIAL HYPERTENSION USING PULMONARY REGURGITATION BY DOPPLER

SUMMARY

Doppler echocardiography provides an impressive noninvasive method to predict the right sided pressures using various parameters like peak TR jet, pulmonary acceleration time with high correlation to data obtained by haemodynamic studies. We hypothesised that peak pulmonary regurgitation (PR) velocity assessed by Doppler would correlate with instantaneous pulmonary artery pressure at dicrotic notch obtained at haemodynamic study. To assess this, we studied 30 patients of pulmonary arterial hypertension with lesions like ventricular septal defect (10), atrial septal defect (12), patent ductus(5) and primary pulmonary hypertension(3). Their age ranged from 4 to 40 years with mean of 21.6 $\pm$ 10.8 yrs. All patients had undergone Doppler evaluation of PR jet (ATL.UM8) for assessment, the previous evening or the morning of the cardiac catheterisation, to assess PA pressures. The results were analysed using Pearson formula for coefficient of correlation using computer and a linear regression formula was derived. As expected PA dicrotic pressures and PA mean pressure were highly correlated ($r = 0.93$ $p < 0.001$). The peak PR jet velocity correlated well with PA dicrotic pressure ($r = 0.8$ $p < 0.001$) and PA mean pressure ($r = 0.75$ $p < 0.001$). A linear regresssion equation formula with peak PR as predictive variable and PA mean as dependent variable was derived.

PA mean pressure . = 0.39 + (1.15 x peak PR jet).
(mm of Hg) **(mm of Hg)**

PREDICTION OF MEAN PULMONARY ARTERY PRESSURE IN PATIENTS WITH PULMONARY ARTERIAL HYPERTENSION USING PULMONARY REGURGITATION FLOW SIGNAL BY DOPPLER

INTRODUCTION

Pulmonary Arterial Hypertension (PAH) can be assessed non invasively by various parameters obtained by M-mode, two dimensional and Doppler echocardiography[1] although the gold standard for quantitiative estimation is cardiac catheterisation. Investigators have used various parameters like right ventricular systolic time intervals[2], flow velocity pattern[3], and tricuspid regurgitation[4] for prediction of right sided pressures. Masuyama et al[5] have reported correlation of pulmonary regurgitation (PR) velocities to pulmonary arterial pressure in normals and in small group of patients with PAH. We have observed that the PA dicrotic pressures obtained at haemodynamic studies correlated well with PA mean pressures. Hence we hypothesized that Doppler predicted peak PR gradient would correlate with instantaneous PA pressure at the time of pulmonary valve closure (dicrotic notch) which in turn would correlate with PA mean pressure. The purpose of this study was to assess the predictive accuracy of estimate of PA mean pressure and PA dicrotic pressure from peak PR gradient derived from Doppler PR signal.

MATERIALS AND METHODS

The study was conducted from March 1992 to Feb 1993 in our Institute. 30 patients were enrolled in the study. There were 15 males and 15 females. The age ranged from 4 to 40 years. All the patients had PAH as assessed by clinical examination. 12 patients had atrial septal defect, 10 had ventricular septal defect 5 had patent ductus and 3 had primary pulmonary hypertension. All the patients were in sinus rhythm. All patients had undergone Doppler study the previous evening or the morning, within 24 hours of the cardiac catheterisation. All Doppler studies were done by a single examiner who was blinded to the haemodynamic study results.

Only those patients who had a pandiastolic PR signal were included in the study. The Doppler examination was performed using duplex echocardiograph (ATL UM8) equipped with 3.5 MHZ mechanical transducer. The signal was obtained in left lateral decubitus position using pulsed mode, high pulse repetition frequency mode and continuous wave mode. A minimum of 5 signals of optimal quality representing maximal flow velocity spectrum during entire diastole were taken for analysis. The peak PR gradient was calculated using simplified Bernoulli equation developed and validated by Holen[6], Hattle[7] and colleagues.

Pressure gradient (mmHg) = 4 v^2 where v is the peak PR velolcity in metres/sec.

The following method was used for the assessment of Peak PR velocity. The deceleration slope of PR Doppler flow signal was obtained by joining the point of peak PR velocity (PPR-V) and point of endiastolic PR velocity coinciding with the onset of next QRS of ECG. This slope was extended backwards to meet a vertical line drawn coinciding with pulmonary valve closure artifact to obtain peak PR velocity at the time of pulmonary valve closure (PPR-PVC). The peak PR velocity (PPR-V) and the peak PR velocity obtained by extending the slope backwards to pulmonary valve closure artifact point (PPR-PVC) were obtained in all patients.(as shown in the figure 2).

Cardiac catheterisation was carried out by the percutaneous femoral venous route and all pressures were recorded using standard fluid filled catheters. An average of five beats were taken to measure the PA systolic, diastolic, dicrotic, and mean pressures (as shown in the figure 1).

ANALYSIS

All values were tabulated amd statistical analysis was done using Pearson formula for coefficient of correlation

with computer software backup. (SPSS/PC+version 4). A linear regression equation formula was derived to predict the PA mean pressure.

RESULTS

A representative figure to show RV and PA pressure tracing (Figure 1) and Doppler derived PR jet envelope (Figure 2) are shown.

The dicrotic pressure obtained at cardiac catheterisation was compared to mean PA pressure, which showed a very high correlation ($r = 0.95$ $p < 0.001$--Figure 3).

The Doppler derived PPR-V was correlated to both PA dicrotic and PA mean pressures. The Correlation coefficient for PPR-V to PA dicrotic pressure was 0.73 ($r=0.73$ $p < 0.001$ -- Figure 4) and PPR-V to PA mean pressure was 0.62 ($r = 0.62$ $p < 0.001$ -- Figure 5).

In comparison, the correlation coefficient for PPR-PVC to PA dicrotic pressure was 0.79 ($r=0.79$ $p < 0.001$ -- Figure 6) and PPR-PVC to PA mean pressure was 0.76 ($r = 0.76$ $p < 0.001$ -- Figure 7) which were distinctly superior to the correlations obtained using PPR-V. A linear regression equation was used to derive the formula.

Mean PA pressure (mmHg) = 0.39 + (1.15 x PPR-PVC)mmHg.

DISCUSSION

In this study we have analysed the correlation of peak PR flow gradient to PA dicrotic and mean pressures obtained at cardiac catheterisation. The PR jet obtained by Doppler shows a characteristic contour as reported earlier[8]. The PR jet is obtained in high percentage of normal as well as in patients with all grades of PAH[9]. This PR is not necessarily attributed to organic changes in the pulmonary valve and can occur normally or due to PA dialatation[10]. The PR Doppler flow velocity signal depicts the instantaneous pressure gradient between PA and RV in diastole from the onset of pulmonary valve closure (dicrotic notch) to the onset of next systole. The instantneous PR gradient is expected to peak at the end of isovolumetric relaxation period of RV which occurs 30 to 80 ms after pulmonary valve closure[11]. Hence it was postulated that if the deceleration slope of PR jet was extended backwards to the onset of pulmonary valve closure on Doppler spectral envelope of PR the velocity thus obtained will reflect PA pressure at the time of pulmonary valve closure ie. dicrotic pressure.

As observed earlier there was a very high correlation of mean PA pressure and to PA dicrotic pressure ($r = 0.9$ $p = <0.001$) obtained at cardiac catheterisation. Both (PPR-V) and (PPR-PVC) had a good correlation to PA dicrotic as well

as PA mean pressure although the (PPR-PVC) had better correlation to mean PA pressure. (0.76 P = <0.001). A linear regression equation formulae was derived using (PPR-PVC) as predictive variable and mean PA pressure as a dependant variable.

PA mean pressure (mmHg) = 0.39 + (1.15 x (PPR-PVC) in mmHg

CONCLUSION

It is concluded that Doppler derived peak PR gradient is predictive of mean PA pressure in patients in PAH. This new noninvasive Doppler method using the pulmonary regurgitation Dopper flow signal may be useful in patients of PAH to predict the PA mean pressure.

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FIGURE 1. PA AND RV PRESSURES

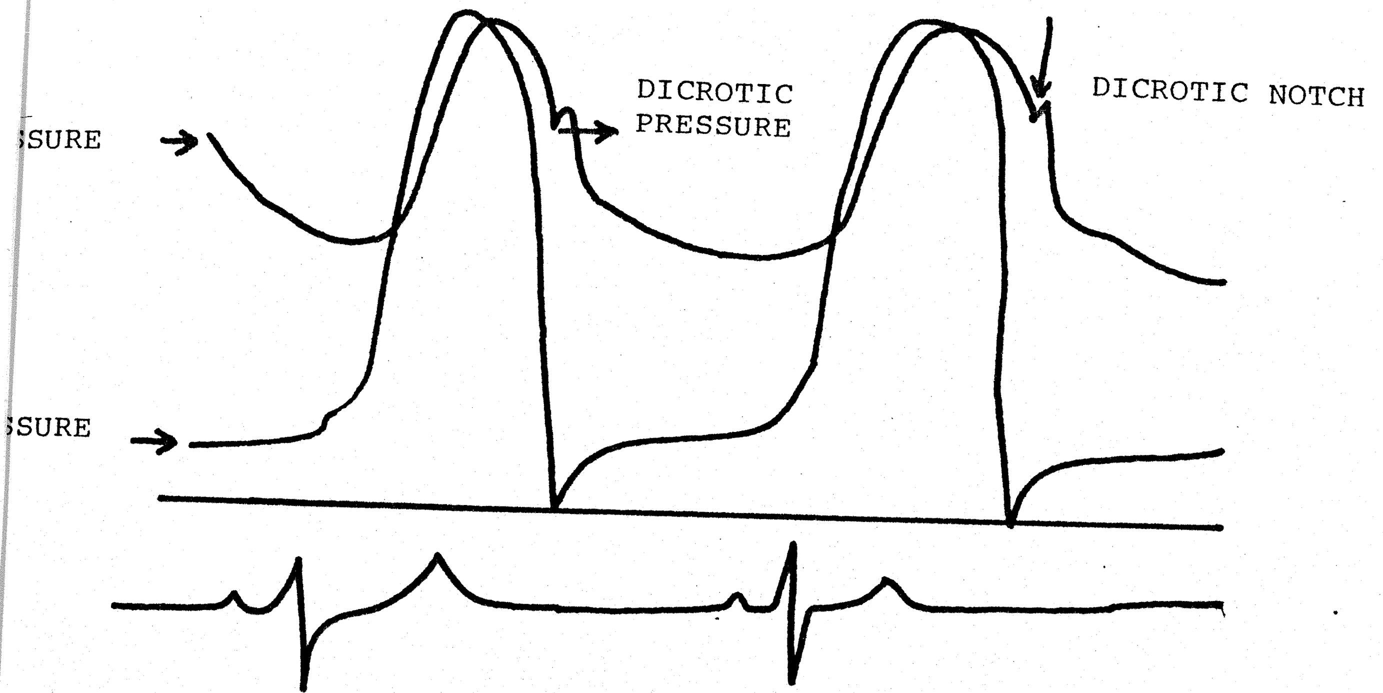


FIGURE 2 . PR SIGNAL ANALYSIS

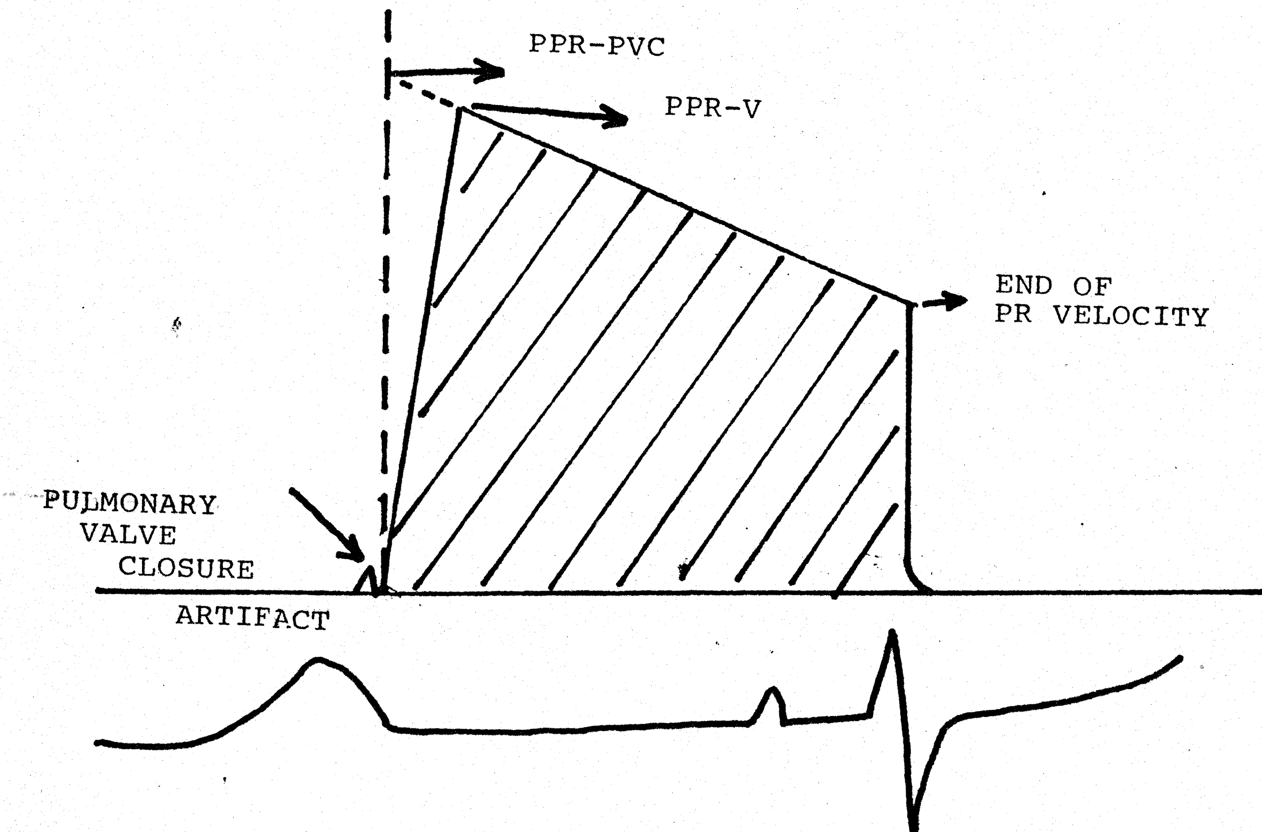
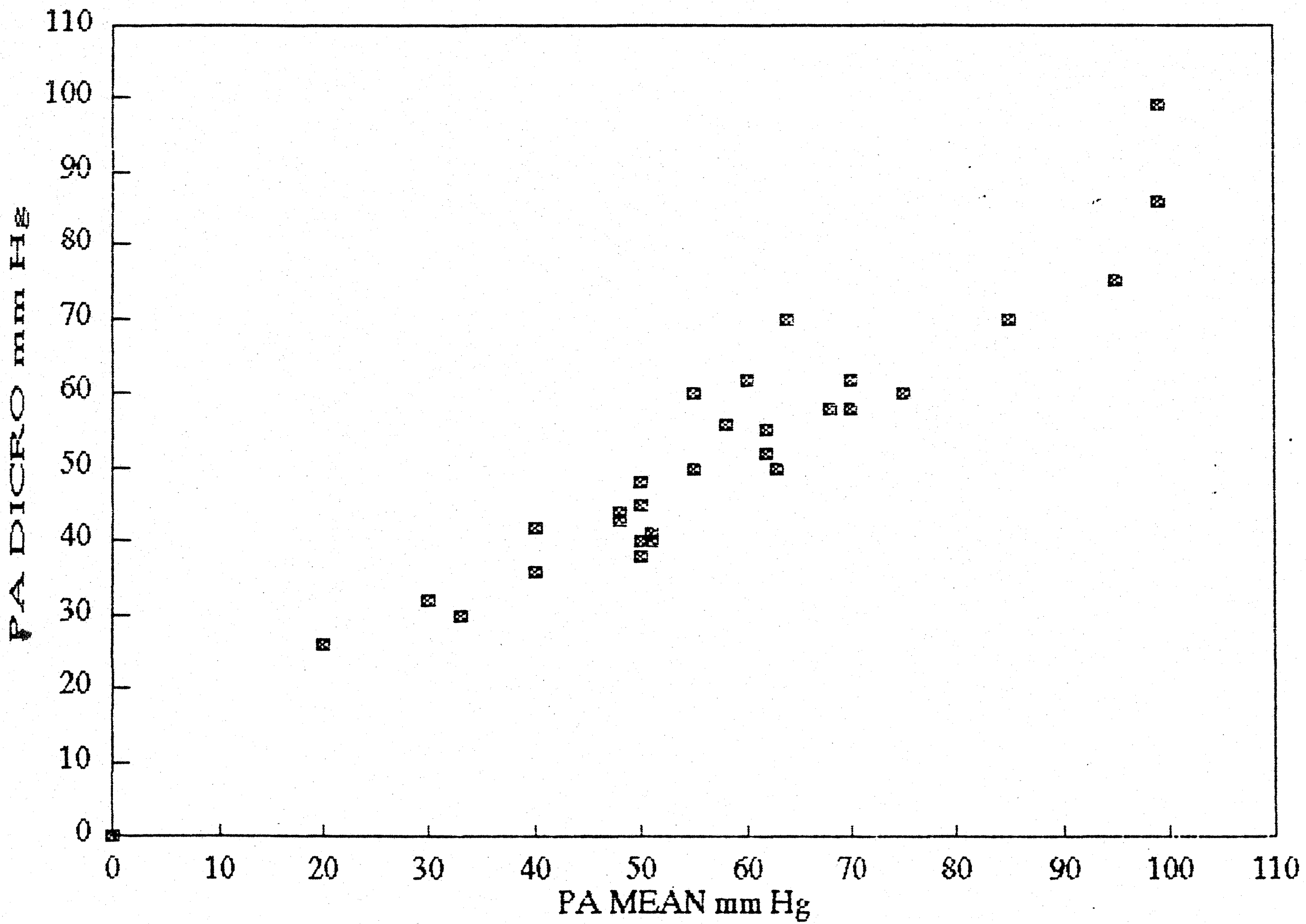


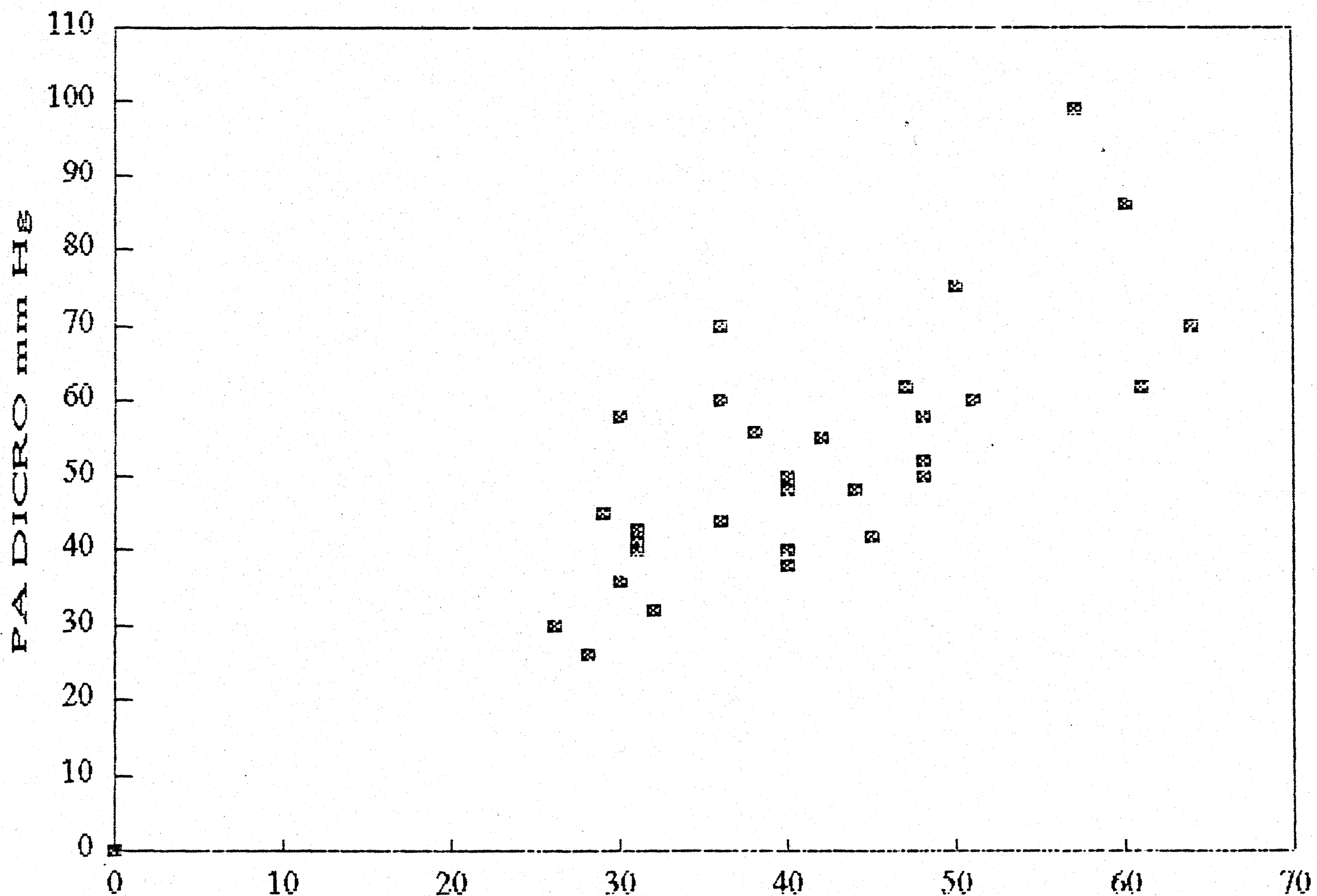
FIGURE 3

Correlations



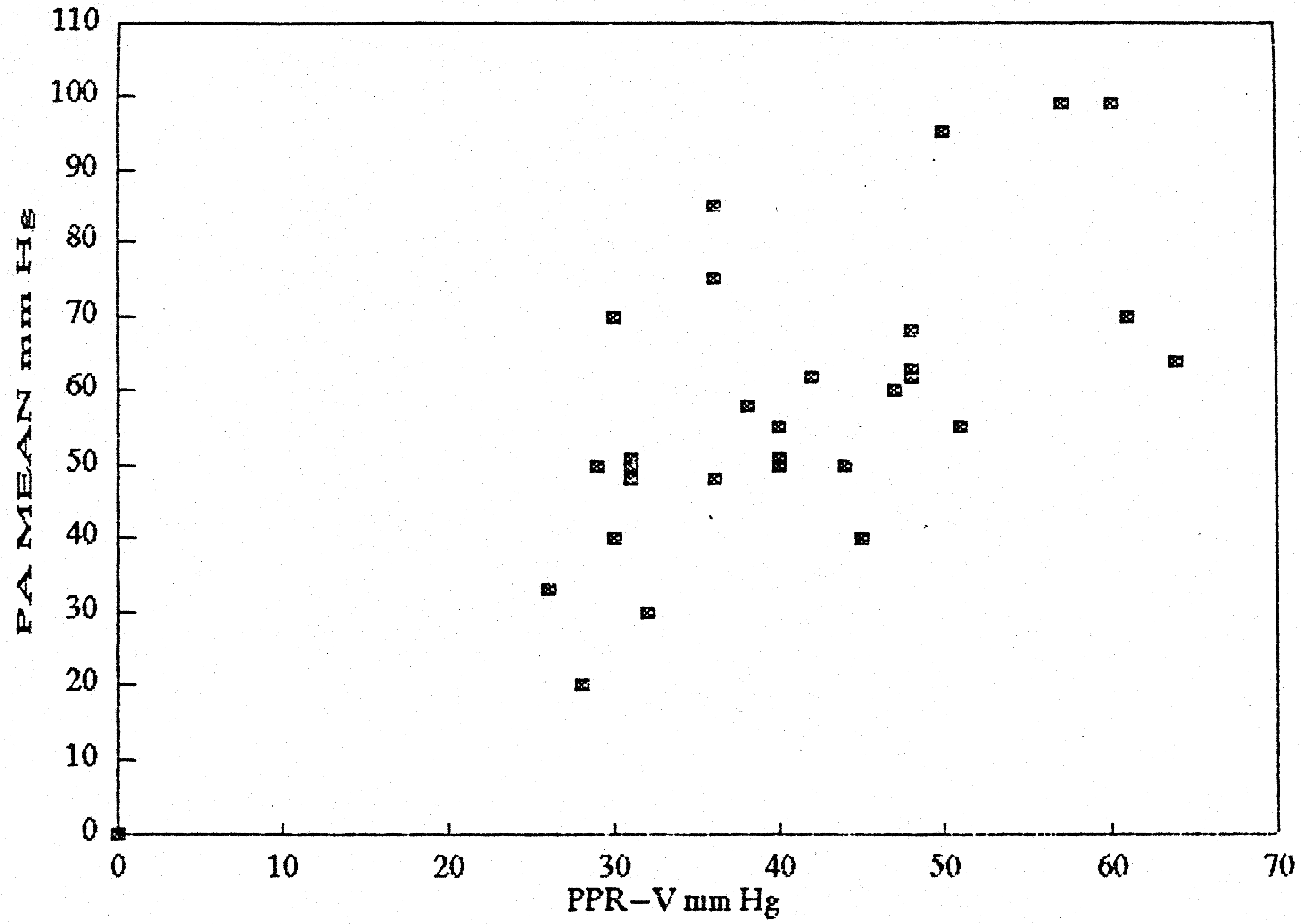
r = .94 p less than .001

FIGURE 4



r = .72 p less than .001

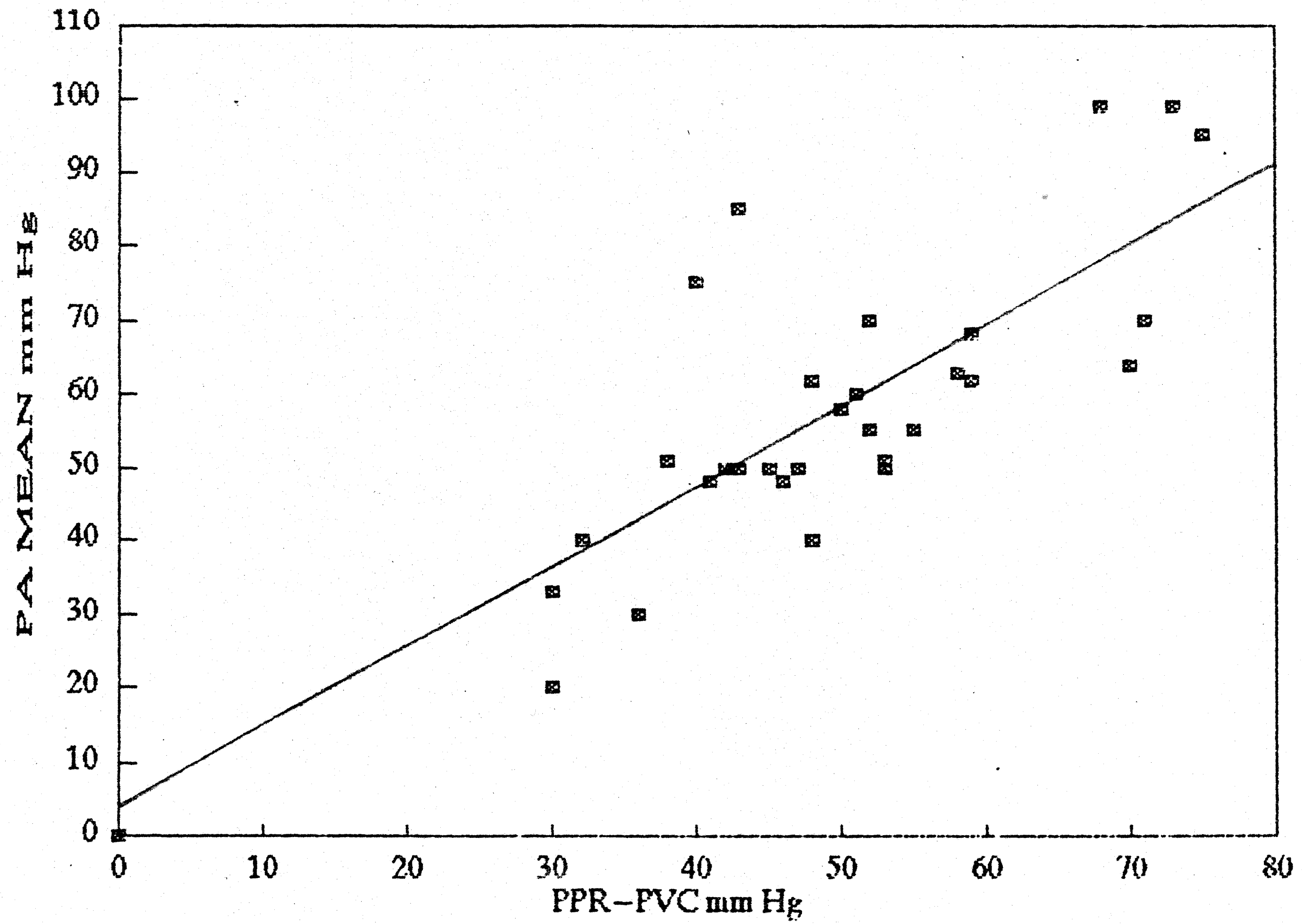
FIGURE 5



r=.62 p less than .001

FIGURE 7

Correlations



r = .76 p less than .001

~~LIST OF PROCEDURES DONE~~

PROJECT REPORT

TITLE OF THE PROJECT: AN ANALYSIS OF TOTAL ANOMOLOUS PULMONARY VENOUS CONNECTION : A REPORT WITH 15 YEARS FOLLOW UP.

NAME DR. J.S.BHUVANESWARAN

PROGRAMME : D.M. CARDIOLOGY PROGRAMME

MONTH & YEAR OF SUBMISSION : DECEMBER 1993.

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM 695 011

Name	Bhuvaneswaran
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AN ANALYSIS OF TAPVC

(TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION)

A REPORT WITH 15 YEARS FOLLOW UP

SUMMARY

Total anomalous pulmonary venous connection (TAPVC) is an uncommon congenital anomaly with an incidence of 0.02 to 2% with a high mortality in infancy. From December 1976 to March 1993 we have encountered 71 cases, of which 37 were males and 34 were females. 20/71 were over 12 years of age and 20/71 were infants. 43 (60%) had supracardiac type, 13 intracradiac type and 15 of mixed type. 12/71 (17%) had obstruction with resultant pulmonary hypertension. The mean pulmonary arterial (PA) pressure ranged from 15 to 86 mmHg with 59/71 having more than 21 mmHg. 39/71 had L-R shunt greater than 2:1. 10 had associated lesions like ventricular septal defect, patent ductus, pulmonary stenosis and two patients had significant extracardiac anomalies. A subgroup of 4 patients were identified to have a right sided vertical vein draining near SVC-RA junction with PA mean pressure of 37$\pm$22mmHg. 8 patients died awaiting surgery in which 7 were infants with mean PA pressure of 41 $\pm$ 15.1 mmHg. 42 patients have been operated so far. 10 were lost for followup. In the right sided vertical vein group with pulmonary hypertension one is alive, one died awaiting surgery and the other two have been operated. The remaining 42 survivors were followed up till March 1993 are doing well.

AN ANALYSIS OF TOTAL ANOMOLOUS PULMONARY VENOUS CONNECTION

A REPORT WITH 15 YEARS FOLLOW UP

Introduction

Total Anomolous Pulmonary Venous Connection is an uncommon congenital anomaly with reported incidence of 0.02 to 2% with a very high mortality in infancy if untreated[1,2]. Uncommonly children or even adults may present for the first time with this leision as, those who Survive infancy have relatively a stable course in the absence of obstruction, pulmonary arterial hypertension and associated anomalies[3]. In this report we present our analysis of anatomic, haemodynamic and angiocardiographic features of 71 cases of Total Anomolous Pulmonary Venous Connection who were seen and followed up in our institute over 15 years.

Materials and Methods:

Between December 1976 to March 1993, we encountered 71 cases of Total Anomolous Pulmonary Venous Connection [TAPVC] who were diagnosed on the basis of clinical, ECG, X-ray chest, echocardiography [Two dimensional and Doppler] and haemodynamic studies. 61/71 cases had undergone cardiac catheterisation and angiography to determine the type of TAPVC, haemodynamic data, and associated cardiac anomalies.

42 patients had undergone surgery till March 1993 and all surviving patients were periodically followed up till date for the assessment of symptomatic status and complications.

Results

There were 37 males and 34 females in the age range of 1 month to 30 years, mean age being 7.67 $\pm$ 7.74 years. Table 1 shows the age and sex distribution of the groups. One third of the patients were infants, and another third in the age group of above 12 years.

The patients were sub divided on the basis of mode of connection [modified from Darling et al[4]] as Group I [supra-cardiac], Group II [Cardiac], Group III [Mixed] and Group IV Sub diaphragmatic type and patients who were evaluated only with echocardiography but expired before confirmatory evaluation. These groups were further subdivided on the basis of exact site of connection, sex, left to right shunt, pulmonary artery mean pressure and pulmonary venous obstruction as shown in table 2. Ten patients of supra cardiac, four patients of cardiac, and two patients of mixed group had severe pulmonary arterial hypertenison (above 50 mmHg),

Presence of atrial septal defect or persistant foramen ovale was considered as a part of the anomaly and any other

associated anomaly was considered for analysis. Four patients had valvar pulmonary stenosis, three had patent ductus arteriosus and one each ventricular septal defect and bilateral peripheral pulmonary stenosis. Three patients had complex cardiac anomalies like corrected transposition with ventricular septal defect, isolated levocardia single ventricle with pulmonary stenosis and inferior venacaval obstruction single ventricle with pulmonary stenosis. 2 Patients had significant extra cardiac anomalies; one had Ellis Van crevald syndrome (Single atrium and polydactyly]with pulmonary venous chamber joining right superior vena cava right atrial junction and other with Craniovertebral anomaly [Basillar invagination, C2 C3 vertebral fusion and hypoplasia of atlas] with left sided vertical vein joining innominate vein.

8 patients died awaiting surgery of whom seven were infants, four with left vertical vein obstruction and two with complex cardiac anomalies complicated by high pulmonary arterial mean pressure [Mean 41$\pm$14 mmHg]. 11 patients died either during surgery or in the post operative period before discharge. 42 patients were operated till March 1993 and 10 were lost for follow up. The 42 survivors [including 31 post operative survivors and 11 cases awaiting surgery] were

followed up till March 1993. 3 patients had developed atrial fibrillation and are now in New York Association Functional[NYHA] Class III. The rest are all in NYHA Class I and II.

Discussion:

Although described by Wilson[5] almost two centuries ago TAPVC still poses problems for both the cardiologist and the cardiac surgeon. Although echocardiography has high specificity and sensitivity[6,7], cases of mixed type, significant pulmonary hypertension and associated anomalies would still require a confirmatory haemodynamic study before subjecting the patients for surgery. The term itself is a misnomer as they are developmentally neither anomolous nor pulmonary but abnormal persistance of embryonic anastomoses between pulmonary and systemic veins in the postnatal period[8]

There was no sex preponderence in our group. An unusual feature is the large sub group of patients (28%) in the age group of 12 years and above. Majority of the patients succumb during infancy due to associated anomalies and presence of venous obstructions. The patients who survive infancy have a relatively stable course and present later as atrial septal defect with increased

pulmonary blood flow[9,10]. Our series too highlights the point that patients with large shunts wihtout any obstruction survive beyond infancy more commonly. Although majority of them have mild to moderate pulmonary hypertension, most of them are flow related and a few were protected by association of mild or moderate valvar pulmonary stenosis.

In comparison with other Western series and recently published Indian series our group differed in certain aspects as shown in table 3.[6,11,12,14] The commonest type of TAPVC was supracardiac as in other series but there were more cases of mixed type and paucity of sub diaphragmatic type in our series. This can be explained on the basis of very high neonatal mortality of subdiaphragmatic type who has virtually 100% incidence of obstruction and may fail to reach the hospital[11,12]. The commonest type of connection in mixed group was in the form of left sided pulmonary veins joining left vertical vein and right pulmonary veins to coronary sinus. Obstruction to the pulmoanry venous flow was present only in 16% of supracardiac group, in contrast to 40-50% obstruction as reported in other series[11,12,14]. In four patients the final confirmatory diagnosis could not be decided although echocardiogram was suggestive of mixed or intracardiac TAPVC. These sick infants died before the

haemodynamic studies either in the hospital or outside and in none autopsy could be obtained.

Pulmonary arterial hypertension is invariable in TAPVC with obstruction and is an important prognostic factor for survival beyond infancy.

Contrary to earlier reports[12] TAPVC directly to right superior vena cava is an uncommon subgroup in supra cardiac type of TAPVC. Although reported with very high incidence of associated complex anomalies and obstruction in other series, only one out of four of our patients had associated Ellis Van Crevald Syndrome. This sub group was found to have higher pulmonary artery pressures and larger shunts.

Associated cardiac and extracardiac anomalies are well known in TAPVC more so with complex anomalies like aspleenia polyspleenia syndromes[12], Two of our patients had extracardiac anomalies and three had complex anomalies like singleventricle, corrected transposition and and isolated levocardia.

Even after surgery TAPVC is not totally free from complications. Atrial arrythmias are known to develop on follow up as in isolated atrial septal defect[15]. Three of

our patients on follow up were found to be in atrial fibrillation, and were well controlled on oral medications.

Conclusions:

TAPVC is not an uncommon cardiac anomaly. It has a high mortality in infancy in the presence of associated obstruction to pulmonary venous flow, pulmonary arterial hypertension and complex cardiac anomalies. Supra cardiac type is the commonest although the obstruction is not as high as reported in other series. Sub diaphragmatic type is very rare, so also TAPVC draining directly to right Superior venacava. Although the mortality is high in infancy, patients with large shunts with only flow related pulmonary hypetension or valvar pulmonary stenosis survive even to adulthood. Long term follow up is necessary after surgery as they have a propensity to develop atrial arrythmias.

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Table 1

Age and Sex Distribution

Age group	Males	Females	Number
< 1 yr	9	11	20
> 1- < 12 yrs	19	12	31
≥ 12 yrs	9	11	20
TOTAL	37	34	71

Table 2
Distribution of anatomic types and haemodynamic factors

Groups	Sub groups	Number	Total	Sex		Pulmonary Artery mean pressure mmHg		(L-R) Shunt		Pulmonary Venous Obstruction	
				M	F	<21	≥21	<2:1	≥2:1	(+)	(-)
Supra Cardiac	A To left sided vertical vein	40	44# [62%]	24	20	14 [39%]	22 [61%]	10 [26%]	28 [74%]	7 [16%]	36 [84%]
	B To right superior vena cava	4									
Cardiac	A To Right atrium	7	13# [18%]	7	6	3	6	3	8	1	8
	B To coronary sinus	6									
Mixed Cardiac	A To left vertical vein& Coronary sinus.										
	B To left ventrical vein& right atrium.	1	10# [14%]	4	6	2	7	2	3	1	5
	C To right superior vena cava & Coronary sinus	1									
	D To right atrium & coronary sinus	1									
Others	A Sub diaphragmatic	0	4 [6%]	2	2	-	-	-	-	-	-
	B Uncertain*	4									

* Diagnosis by echo only.
Site of connection uncertain.

\# Including cases were PA entry and
Shunt assessment not quantified

Table 3

Comparison with other series

TAPVC Group	Burrough & Edwards	Lucas & Lock	Delisle et al	Goswamy et al	Bhuvaneswaran et al
	113 cases (%)	44 cases (%)	93 cases (%)	110 cases (%)	71 cases (%)
Supra cardiac	47	49	41	63	62
Intra cardiac	31	18	26	27	18
Mixed	7	10	5	5	14
Sub Diaphragmatic	13	23	24	5	0
Others & Uncertain	2	0	4	0	6