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LIST OF PROCEDURES DONE
PROJECT REPORT

TITLE OF THE PROJECT: CORRELATION OF ELECTRICAL POTENTIALS WITH TWO DIFFERENT TYPES OF ELECTROCARDIOGRAPHY MACHINES

NAME.....DEBANU GHOSH RAY.....

PROGRAMME : DM CARDIOLOGY.....

MONTH & YEAR
OF SUBMISSION : NOVEMBER 1991.....

CERTIFICATE

I, Dr. DEBANU GHOSH RAY .....hereby declare that I have actually performed all the procedures listed / carried out the project under report.

Signature..... Debanu Ghosh Ray

Place Trivandrum

Name in DEBANU GHOSH RAY.....
capital letters

Date: 8.11.91

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

Signature

Head of the department

CONTENTS

LIST OF CARDIAC CATHETERISATION AND ANGIOCARDIOGRAPHY

Name	Age	Sex	Cath No.	Hospital No.	Rt. Heart	Lt. Heart	Angio	Diagnosis
2	3	4	5	6	7	8	9	10
Indu R Pillai	8	F	8020	8601328	+		+	ASD
Kousalya KM	26	F	8035	33051	+		+	RVEMF
Shally Joseph	22	F	8061	8809219	+			PDA PAH
Bindu S Pillai	13	F	8071	8600754	+			ASD
Devraj S	19	M	8072	8901801	+		+	Post op ASD
Sunimol P	65	F	8075	32475	+		+	Post op TOF
Biju G	12	M	8076	8604548	+		+	ASD,PAH, MS,MR
Ahmedkutty	40	M	8077	8901318	+	+	+	CAD
Sreekala S	14	F	8079	8805846	+	+	+	RVEMF
Sooraj R	35	M	8083	8601484	+	+	+	DORV,VSD, PS
Omana C	25	F	8090	8807393	+	+	+	Coarctation
Ajesh VJ	9	M	8093	8902964	+			ASD
P.Leela	50	F	8096	8900267	+			PDA
Sojan Thomas	18	M	8098	8800153	+		+	TOF
Abdul Khader	50	M	8105	8808575	+	+	+	DORV,VSD, PS
Lenin NC	8	M	8107	8709264	+		+	VSD,PS
Anitha CM	11.5	F	8116	8704470	+		+	Val PS
Sreeja	15	F	8153	8702784	+			ASD
Biju	21	M	8154	05129	+			ASD
Aleyamma Varghese	38	F	8159	8805737	+	+	+	AP Window
Alekutty Devassia	36	F	8168	8901384	+	+	+	HOCM,MR
Mohana CV	36	M	8182	8801107	+			ASD
Manju S	65	F	8186	30389	+			ASD
PV Susmitha	14	F	8187	8706900	+		+	ASD
Nimmy Joseph	6	F	8198	28090	+			ASD
Latha S	13	F	8200	8603202	+	+	+	VSD
Pushapa M	24	F	8202	8900535	+			ASD

2	3	4	5	6	7	8	9	10
. Safeer EM	11	M	8207	8703299	+		*	ASD
. Regi Varghese	26	M	8215	8806481	+		+	TOF
. Shoba Kumari	33	F	8219	8600952	+	+	+	VSD
. Divakaran S	46	M	8223	34717	+	+	+	HOCM
. Babu Rajan V	24	M	8227	8801134	+		+	RSOV
. K.J.Paul	52	M	8230	8809529	+			ASD,PAH
. Krishnan V	45	M	8860	8908169	+			ASD,PAPVC
. Swapna Sebastian	10	F	8902	21736	+	+	+	Post op residual VSD
. Zeenath	17	F	8904	8903734	+	+	+	Primum ASD
. Sameer K	3	M	8912	8908518	+	+	+	Pulmonary atresia.
. Karunakaran	51	M	8914	8900172	+	+	+	CAD
. Sindhu AV	19	F	8919	8906054	+			ASD
. Bindu R	8	F	8958	8602872	+	+	+	TOF
. Sajeev PK	18	M	8967	13153	+	+	+	Single Atrium,MR
. Omana VG	16	F	8971	8803425	+		+	DORV,VSD, PS
. Sreeja	14	F	8972	26021	+	+	+	ASD,MR
. Seema S	10	F	10053	8605552	+		+	TOF
. Sandhya	7.5	F	10054	8800916	+		+	TOF
. Rubina M	8	F	10059	8708229	+		+	DCRV
. Prakash Kumar	21	M	10060	8905994	+	+	+	ASD
. Rohini P	39	F	10061	9001306	+	+	+	ASD
. Sobha N	11.5	F	10063	8605649	+		+	ASD
. Parthibhan	8	M	10066	8804748	+		+	TOF
. Sameera KP	12	F	10067	89001641	+		+	VSD,ASD
. Anish T	4	M	10074	8606346	+		+	VSD
. Mohanan V	22	M	10075	8702663	+		+	TOF
. Ramesh TV	17	M	10080	8702430	+	+	+	ASD,MR
. Ponamma VR	23	F	10088	9006855	+	+	+	MS,AR,TS, TR

2	3	4	5	6	7	8	9	10
V.P.Natarajan	43	M	10089	9006887	+		+	BVEMF
Prem Churchill	26	M	10090	8606530	+	+	+	ASD
Mymoona	22	F	10091	8705371	+	+	+	Situs Inversus, Dextrocardia, VSD,PS
Balan C	39	M	10093	9001666	+			Normal Heart
Venkateswari	3	F	10094	8904037	+	+	+	Infundibular PS
Remarati	6	F	10095	9006663	+	+	+	TOF
Asokan KT	23	M	10096	9001662	+			ASD, WPW
Ansarkhan	3.5	M	10098	9006097	+			Pulmonary atresia
Suhair	18	M	10100	8906705	+		+	Single Atrium
Gokila LS	18	F	10101	8909457	+		+	Post balloon PS, ASD
Jayakumari	16	F	10104	8600762	+		+	TOF,PR
Padmarajan M	28	M	10110	9006257	+	+	+	ASD,MR
Sheela S	20	F	10111	8707305	+			PPH
Sajitha	6	F	10115	8704744	+	+	+	ASD,MR
Sreena R	11	F	10120	22867	+		+	Single Atrium
Beena M	14	F	10127	9002106	+	+	+	VSD,MAR ASD
Sibichan Joseph	17	M	10127	8901918	+	+	+	Primum ASD
Kamalam C	27	F	10129	9007187	+	+	+	LTGA,PAH,CHB
Joly Antony	9	M	10132	8701157	+	+	+	VSD,PAH
Jancy	12	F	10133	33738	+		+	TOF
Bindu Kumari	18	F	10140	8701207	+		+	TOF
Thomas M	29	M	10141	8701025	+			ASD
Sadanandan Nair	40	M	10142	9006691	+		+	ASD
Girijam N	28	F	10143	9001891	+			ASD
Binu Pappanju	11	M	10144	9007057	+		+	TAPVC
Bijoy Yohannan	11	M	10148	12289	+		+	TOF
Manikantan K	13	M	10698	9004813	+	+	+	DCRV,VSD,ASD
Sujatha K	8	F	10700	8803477	+	+	+	PS,VSD,PDA
Vijayan	11	M	10704	9004206	+		+	ASD,PAPVC
Sankari	22	F	10705	10577	+		+	Valvar and peripheral PS

	3	4	5	6	7	8	9	10
amila A	4.5	F	10795	8708557	+	+	+	Single Ventricle Pulmonary atresia, BT Shunt.
eepa S	12	F	10799	3312	+			VSD
ukumaran Nair	38	M	10800	9100734	+	+	+	RSOV to RV
amasivayam	35	M	10828	37708	+	+	+	HOCM
ohammed Ibrahim	19	M	10836	9005300	+		+	TOF
aveedran Pillai	44	M	10839	9003686	+	+	+	EMF
athew PK	27	M	10842	8901760	+	+	+	Residual VSD, MVR
radeep Kumar	24	M	10865	8606610	+		+	VSD
alithamma	57	F	10866	587	+	+	+	BVEMF
oosuf KM	20	M	10869	8807079	+			ASD
unjunni Menon	43	M	10870	9004478	+			ASD
ijoy KP	6	M	10890	8806664	+		+	Post balloon PS
itus TT	60	M	10891	9102348		+	+	CAD
ditya Bhalla	7	M	10900	9007907	+		+	Post balloon PS
nil kumar MS	22	M	10901	18318	+	+	+	AR

	3	4	5	6	7	8	9	10
geswaran A	6	M	10709	9009563	+	+	+	TOF
ish V.G	4.5	M	10710	8800502	+		+	TOF
raswthy M	38	F	10711	9001984	+		+	Valvar, Subvalvar and supravalvar PS
varama Pillai	42	M	10712	9007269		+	+	CAD
machandran K	48	M	10713	8805414		+	+	CAD
apna	8	F	10718	19294		+	+	TOF
inthu Das	4	M	10719	8707	+	+	+	TOF
jeshwari	35	F	10721	9100259	+	+	+	Primum ASD
ureet Kaur	3	F	10722	9101919	+	+	+	Submitral aneurysm.
itto George	3.5	M	10725	8806051	+	+	+	DCRV
eeba PC	14	F	10733	16360	+		+	Post balloon PS
jimol CV	20	F	10734	8806697	+			MVP, MR
ahida	7	F	10735	8902428	+		+	TOF
nn Biitto	4	M	10740	9001092	+		+	ASD, PS
i Krishnan	16	M	10741	9099	+			PS
bhaschandra	35	M	10758	9010290	+		+	ASD
etha K	30	F	10759	9101319	+		+	ASD
itha kumari	22	F	10760	17902	+		+	Post balloon PS
shpa PK	28	F	10761	9007402	+	+	+	ASD, MS
dhakrishnan Nair Nair	38	M	10765	9009192	+	+	+	CAD
ja	19	F	10770	8607419	+	+	+	Pulmonary atresia, VSD, Rt.BT Shunt
ana M	17	F	10775	9007185	+	+	+	TOF, absent LPA
akkivalli J	4	F	10777	8805685		+	+	TOF, BT shunt
silin T	5	F	10784	8701400	+		+	TOF
dul Rehiman	53	M	10787	9010362		+	+	CAD
ria Goretti	25	F	10788	9005908	+	+	+	ASD.

LIST OF ELECTROPHYSIOLOGICAL STUDIES

Name	Age	Sex	Hospital No.	Cath No.	Diagnosis
Thankappan Nair	52	M	8900567	8140	SSS
Karuna K	55	F	8902366	8193	SSS
Rev.Jacob John	56	M	8902477	8204	Bifascicular Block
Jaya P.G	16	F	21623	8209	CHB
Kuttapan	57	M	8908319	8875	SSS
Babu	20	M	8902269	8920	SSS
Chandrasekharan Nair	68	M	12718	10055	CAD-bifascicular block
Gracy Thomas	22	F	8808685	10071	CHB
Siddique K	17	M	9006907	10092	CHB
Asokan KT	23	M	9001662	10096	ASD,WPW
Periaswamy P	60	M	9007214	10128	SSS
Komalam C	27	F	9007187	10129	LTGA,PAH,CHB
Uttaman KG	36	M	8905452	10697	VT
Ambrose clarence	25	M	9100062	10751	WPW Syndrome
Rajan	30	M	9102912	10835	CHB

LIST OF TEMPORARY TRANSVENOUS PACING

Name	Age	Sex	Hospital No.	Diagnosis
Saraswathi Amma	53	F	29342	CHB
A.K. Sarojini	43	F	8903573	CHB
P.J. Antony	65	M	8903367	CHB
K.P. Sulochana	50	F	8903529	CHB
Narayan Pillai K	68	M	8908514	Bifascicular Block
Mathew Thekkumkattil	76	M	9006650	CHB
Vijay kumar KC	36	M	8901358	Recurrent VT
Zeenath C	9	F	8903255	CHB
Joseph P	70	M	9102406	Ac.Ant.MI,CHB
Mandokini	63	F	8604965	Ac.Inf.MI,CHB
Surendranathan	47	M	9009062	CAD-asystolic cardiac arrest.

LIST OF TEMPORARY TRANSVENOUS PACING

Name	Age	Sex	Hospital No.	Diagnosis
Saraswathi Amma	53	F	29342	CHB
A.K. Sarojini	43	F	8903573	CHB
P.J. Antony	65	M	8903367	CHB
K.P. Sulochana	50	F	8903529	CHB
Narayan Pillai K	68	M	8908514	Bifascicular Block
Mathew Thekkumkattil	76	M	9006650	CHB
Vijay kumar KC	36	M	8901358	Recurrent VT
Zeenath C	9	F	8903255	CHB
Joseph P	70	M	9102406	Ac.Ant.MI,CHB
Mandokini	63	F	8604965	Ac.Inf.MI,CHB
Surendranathan	47	M	9009062	CAD-asystolic cardiac arrest.

LIST OF ECHOCARDIOGRAPHIC EXAMINATIONS

INCLUDING DOPPLER

Name	Age	Sex	Hospital No.	Diagnosis
Saneesh George	8	M	8600126	DCM
Balakrishnan Nair	60	M	9108373	CAD
Saddisan AB	51	M	9108372	CAD
Pradeepan VV	18	M	9108375	RHD -MS,PAH,TR
Haleema Sulaiman	36	F	9008550	RHD -MS,PAH
Krishnankutty	64	M	9108378	CAD
Vasudevan Nair	52	M	9108381	CAD
Mohammed Kunju	48	M	9108407	CAD
Ramaswamy	65	M	9108411	RHD - CAD
Shanmugham K	27	M	9108374	RHD . MS,AR
Kumari	40	F	9108385	RHD -MS,PAH
Savithri	32	F	9108386	RHD -MS,MR
Minimol	22	F	8602729	AR
Unnikrishnan P	11	M	9108423	RHD -MS,PAH
P.K. Narayanan	45	M	9104463	CAD
Ramachandran Assari	45	M	9108342	CAD
Maya KJ	12	F	9108428	RHD -MR

	Name	Age	Sex	Hospital No.	Diagnosis
.	Kamalakshy PA	46	F	9006625	Recurrent VT
.	Annama Chacko	50	F	9001148	RHD -MS,MR
.	Biji MP	18	M	9104910	Viral Myocarditis
.	Paul SC	55	M	9008503	Post op CABG
.	Sivankutty K	24	M	8907945	AR
.	Nazeema N	45	F	9108458	CAD
.	M.Hamsa	25	M	8708715	RHD . AS,AR,MS
.	Kanchana AN	21	F	01026	Atrial Paralysis
.	Sabu John K	22	M	9002305	RHD -AR
.	Sasikala	22	F	9108484	RHD-MS,PAH
.	V.V. Paul	61	M	9108492	CAD
.	Pratap Singh	50	M	9108494	CAD
.	Saranya S	4	F	9008447	MVP - MR

CORRELATION OF ELECTRICAL POTENTIALS WITH TWO DIFFERENT TYPES OF ELECTROCARDIOGRAPHY MACHINES.

INTRODUCTION

In 1975 the electrocardiogram (ECG) recording devices in this Institute recorded with the help of a heated stylus. In 1985 the newer devices which were bought recorded by spraying an ink jet. Many patients who were on followup for a long time had their electrocardiogram recorded by both the devices. However, differences were often ignored as it was felt that the electrocardiogram of the two devices were not strictly comparable. This study was undertaken to compare and contrast the ECG recorded by the two devices.

MATERIALS AND METHODS

25 consequetive patients each above the age of 4 with right ventricular hypertrophy (RVH) and left ventricular hypertrophy (LVH) who were admitted to the ward had their ECG recorded by both the devices. RVH was diagnosed if R/S ratio was greater than 1, in Lead V_1. LVH was diagnosed if S in V1 + R in V5 or V6 was greater than 35.

12 lead ECG was first recorded in Hewlett Packard 4700 A Cardiograph and henceforth will be referred as page writer. The lead positions in the chest were marked. Then using the same marks 12 lead ECG was recorded in Hewlett Packard 1504 A Electrocardiograph Sanborn Series and henceforth

will be referred as heat stylus. Standardisation for records was 1mv = 10 mm. However, if the voltages were very high and ECG could not be recorded in the strip of paper then standardisation was 1 mv = 5mm. Amplitude of R, S, and T waves in the two records were compared. Also the configuration of QRS, ST and T waves were noted to look for any differences.

Statistical Methods: Students t test was used to compare paired data.

RESULTS

RVH : There were 15 males and 10 females and their age ranged from 4 to 37 years (mean 11.6 ± 9 years). As shown in Table I & II amplitude of R and S waves were higher in the heat stylus records. There was no significant difference in T wave amplitude (Table III). The configuration of QRS, ST and T waves were same.

LVH : There were 16 males and 9 females and their age ranged from 9 to 60 years (mean 23.7 ± 15 years) As shown in Table IV and V amplitude of R and S waves were higher in the heat stylus records. There was no significant difference in the T wave amplitude (Table VI). The configuration of QRS, ST and T waves were same.

DISCUSSION

The configuration of the waves in the two records were comparable. At lower voltages the amplitude of the waves were

also same in two records. However, at higher voltages the heat stylus recorded higher voltages. The most likely cause for this is overshoot.

The differences were most noticeble when the amplitude of the waves were 15 to 20 mm. At higher voltages the graph could not be recorded in the strip of paper and half standardisation had to be used. This reduced the amplitude of the waves and the overshoot. This is the likely explanation for reduced significant difference at higher voltages.

There was a trend for higher amplitude waves recorded by heat stylus in almost all leads but in many leads it did not reach significance. There are two reasons for this. First is the marked variation in the amplitude of waves in different patients. Second is the marked variation in voltage with slight change in position of the precordial leads.

In conclusion, the heat stylus recorded higher amplitude waves at higher voltages because of overshoot. In all other respect the two tracings were comparable.

Table - I

R wave amplitude in millimeter in 25 patients with right Ventricular hypertrophy

Lead	Page Writer	Heat stylus	P Value (less than)
I	4.72 ± 3.55	4.84 ± 3.60	NS
II	9.08 ± 4.41	9.24 ± 4.85	NS
III	16.52 ± 5.95	17.2 ± 6.34	0.05
aVR	4.08 ± 3.64	4.28 ± 3.68	NS
aVL	3.56 ± 4.65	3.68 ± 4.71	NS
aVF	12.44 ± 4.98	12.84 ± 5.73	NS
V1	17.88 ± 10.12	18.56 ± 10.37	0.05
V2	20.52 ± 15.08	21.48 ± 14.89	0.05
V3	20.08 ± 14.19	21.24± 14.88	NS
V4	19.2 ± 13.54	20 ± 13	0.05
V5	16.64 ± 14.53	17.6 ± 14.4	0.05
V6	11.04 ± 8.11	11.96± 7.86	NS

NS = not significant.

Table - II

S Wave amplitude in millimeters in 25 patients with right Ventricular hypertrophy

Lead	Page writer	Heat stylus	P Value (less than)
I	10.56 ± 3.28	10.96 ± 3.23	0.05
II	2 ± 3.75	2.2 ± 4.26	NS
III	1.28 ± 5.99	1.32 ± 5.99	NS
aVR	0	0	NS
aVL	12.84 ± 6.67	13.32 ± 6.45	NS
aVF	1.28 ± 4.38	1.32 ± 4.39	NS
V1	2.96 ± 3.21	3.16 ± 3.25	NS
V2	18.28 ± 10.59	19.48 ± 11.38	0.05
V3	20.76 ± 9.74	21.48 ± 8.68	NS
V4	16.44 ± 8.42	18.16 ± 8.3	0.001
V5	10.76 ± 6.61	12.12 ± 6.8	0.05
V6	6.08 ± 4.4	7.12 ± 4.65	NS

NS = not significant

Table - III

T Wave amplitude in millimeters in 25 patients with right ventricular hypertrophy

Lead	Page Writer	Heat stylus	P value
I	2.08 ± 1.26	2.08 ± 1.19	NS
II	2.84 ± 1.37	2.8 ± 1.41	NS
III	0.6 ± 1.8	0.64 ± 1.75	NS
aVR	2.6 ± 0.91	2.52 ± 0.87	NS
aVL	0.84 ± 1.51	0.76 ± 1.45	NS
aVF	1.76 ± 1.51	1.8 ± 1.44	NS
V1	-0.56 ± 2.96	-0.36 ± 2.91	NS
V2	3.08 ± 5.02	3.08 ± 5.12	NS
V3	2.9 ± 4.86	2.96 ± 5.02	NS
V4	2.72 ± 4.23	2.88 ± 4.07	NS
V5	2.76 ± 2.3	2.84 ± 2.53	NS
V6	2.72 ± 2.09	2.88 ± 2.03	NS

NS = not significant

Table - IV

R Wave amplitude in millimeters in 25 patients with left ventricular hypertrophy

Lead	Page Writer	Heat stylus	p value (less than)
I	12.24 ± 5.5	12.6 ± 5.83	NS
II	18.92 ± 7.84	19.52± 8.1	0.05
III	13.12 ± 9.09	13.48 ± 9.59	NS
aVR	2.4 ± 3.66	2.4 ± 3.63	NS
aVL	6.84 ± 4.36	6.92 ± 4.44	NS
aVF	15.6 ± 8.09	16.16 ± 8.53	0.05
V1	9.36 ± 11.04	9.12 ± 10.25	NS
V2	14.24 ± 12.06	14.96 ± 12.52	NS
V3	18.04 ± 12.43	17.64 ± 11.99	NS
V4	26.56 ± 14.84	29.68 ± 17.01	0.01
V5	40.24 ± 13.32	40.6 ± 13.33	NS
V6	35.52 ± 13.58	37.52 ± 13.21	NS

NS = not significant

Table - V

S Wave amplitude in millimeters in 25 patients with left ventricular hypertrophy

Lead	Page writer	Heat stylus	p Value (less than)
I	2.48 ± 4.51	2.56 ± 4.67	NS
II	2.96 ± 3.61	3.12 ± 3.72	NS
III	4.08 ± 5.37	3.96 ± 5.15	NS
aVR	2-6 ± 6.14	2-64 ± 6.16	NS
aVL	4.68 ± 5-7	5 ± 6.23	NS
aVF	3.04 ± 3.23	3.16 ± 3.29	NS
V1	25.36± 13.56	26.56 ± 14.6	NS
V2	40 ± 17.08	41-84 ± 17.08	0.05
V3	30-96± 16.27	34.8 ± 15.65	0.001
V4	17.4 ± 14.27	20.08 ± 15.31	0.001
V5	5.52± 8.27	6.52 ± 8.68	0.01
V6	2.2 ± 5.77	2.44 ± 5.87	NS

NS = not significant

Table - VI

T Wave amplitude in millimters in 25 patients with left ventricular hypertrophy

Lead	Page writer	Heat stylus	p Value
I	1 ± 2-29	0.96 ± 2.35	NS
II	0.64 ± 2.67	0.64 ± 3.49	NS
III	-0.24 ± 2.67	-0.24 ± 2.68	NS
aVR	-0.76 ± 2-68	-0.76 ± 2.68	NS
aVL	0.48 ± 2.84	0.48 ± 1.69	NS
aVF	0.56 ± 2.84	0.6 ± 2.81	NS
V1	2.24 ± 4.83	2.28 ± 4.77	NS
V2	6.28 ± 7-23	6.36 ± 7.32	NS
V3	4.88 ± 7.57	5.2 ± 7.16	NS
V4	3.44 ± 6.37	3.16 ± 6.5	NS
V5	1.64 ± 7.25	1.4 ± 7.47	NS
V6	0.6 ± 5	0.68 ± 4.87	NS

NS = not significant

BALLOON ANGIOPLASTY FOR NATIVE COARCTATION OF AORTA AFTER INFANCY : FACTORS DETERMINING THE OUTCOME.

BALLOON ANGIOPLASTY FOR NATIVE COARCTATION OF AORTA AFTER INFANCY : FACTORS DETERMINING THE OUTCOME.

S U M M A R Y

Balloon angioplasty was performed in 46 patients (age 2-40 years) with discrete native coarctation of aorta. Patients with associated patent ductus arteriosus, aberrant subclavian artery and aneurysms were excluded. The peak systolic gradient across the coarcted segment decreased from 52.1± 18.5 mm Hg to 18.6 ± 14.8 mm Hg (P<0.001), and the diameter of the coarcted segment increased from 3.6± 1.7 mm/m^2 to 9.1± 3.2 mm/m^2 (p<0.001). Follow-up haemodynamic and angiographic study, performed in 21 patients at 13.1± 6.9 months after angioplasty, showed good result in 15 patients. Three patients developed aneurysms. The remaining 3 patients, and 5 other patients on noninvasive evaluation were graded as having poor result at follow-up. In 5 of these patients the poor result was due to primary failure of angioplasty in relieving the gradient, and three developed recoarctation after initial fall in the transcoarctation gradient. Three risk factors were identified among 22 variables, which were associated with poor result at follow-up: 1) Size of balloon/size of coarcted segment ratio <3.0; 2) size of isthmus/size of coarcted segment ratio<3.0; and 3) size of descending thoracic aorta distal to the coarctation/size of isthmus ratio>1.75. Presence of one or more risk factors was associated with larger residual gradients. Patients with aneurysm formation were found to have relatively smaller

isthmic diameters, and the balloon diameter exceeded the isthmus size in all 3 patients. One patient needed early surgery but the other two have remained stable with small and non-progressive aneurysms. We conclude that balloon angioplasty is an effective and safe procedure in patients with discrete native coarctation, and the early improvement is sustained at follow-up in the majority of patients. With the identification of risk factors it should be possible to improve the result further and minimise the risk of aneurysm formation.

Key words:

Coarctation, Aorta, Angioplasty, Balloon dilatation.

INTRODUCTION

Balloon angioplasty of coarctation of aorta was first reported in 1982(1). Since then a large number of patients with coarctation of aorta have been subjected to balloon dilatation (2-7). It is generally accepted as a safe alternative to operation in patients with recoarctation after prior surgery. Many studies have also documented its efficacy in native coarctation and the short-term results have been impressive(3-10). The long-term results, however, have been less uniformly positive(5, 8-10). While many patients continue to show good haemodynamic results on late follow-up, some studies have reported development of recoarctation, especially

in infants (8-9). Others have documented aneurysm formation on follow-up (9-10). Few studies, however, have analysed the factors determining the late results of balloon angioplasty in native coarctation(8,10).

The purpose of this study is to investigate the factors which can predict the late outcome of balloon angioplasty of native coarctation, performed in patients beyond one year of age.

METHODS

During the 52 months period ending in October 1990, balloon dilatation of native aortic coarctation was planned in 53 consecutive patients. Only patients with discrete coarctation were included. All patients with vascular anomalies around the coarctation site such as patent ductus arteriosus, aberrant subclavian artery, presubclavian coarctation and aneurysms were excluded. In 7 patients the coarcted segment could not be crossed by guidewire and/or catheter. The remaining 46 patients underwent balloon dilatation and form the basis of this report. There were 35 males and 11 females, and their ages ranged from 2 to 40 years (mean 17.3 $\pm$ 9.7 years).

Balloon angioplasty was performed by standard percutaneous method described earlier (4,5). The size of the

balloon chosen for angioplasty was equal to or just less than the diameter of the isthmus, according to availability. The aortic isthmus was measured midway between the origin of the left subclavian artery and the coarcted segment. This measurement was obtained by suprasternal echocardiography before catheterisation, and confirmed on the angiogram after correcting for magnification. If satisfactory reduction of gradient was not obtained, the next bigger balloon was used. At no time was a guidewire or catheter manipulated across a freshly dilated coarcted segment. The data listed in Tables 1 to 5 were obtained. Measurements of the aorta were made in the left anterior oblique and/or anteroposterior views.

Thirty three patients were available for follow-up by clinical and doppler echocardiographic examinations for 15.8$\pm$ 12 months (range 2 - 48 months) after the angioplasty. Follow-up catheterisation and angiography was performed in 21 patients, 4 to 32 months (mean 13.1 $\pm$ 6.9 months)after the angioplasty.

The late results of balloon angioplasty were classified into 3 groups. Good result (Group A) was defined as peak residual systolic gradient across the coarcted segment of less than 20 mm Hg or a gradient of 20 to 29 mm Hg with at least 50% reduction from the pre-dilatation gradient. Poor result (Group B) was defined as a peak systolic residual gradient of

30mm Hg or more, or residual systolic gradient of 20 to 29 mm Hg but with less than 50% reduction in the gradient. When the angiogram at follow-up showed an aneurysm, the result was classified as Group C,irrespective of the gradient.

STATISTICAL METHODS Data are presented as mean values ± S.D. Student's t test was used to compare data before and after angioplasty, and for comparison of different groups. Fishers Exact test was used to compare frequency distributions. Multivariate analysis was performed by stepwise logistic regression to identify significant factors related to the result of balloon angioplasty. A p value of less than 0.05 was considered "significant", and a p value of 0.05 to 0.1 was considered to be "Borderline significant".

R E S U L T S

The haemodynamic and angiographic findings before and after angioplasty in the total group of 46 patients are shown in Table 1. Thirty eight patients had a good result soon after angioplasty. There were significant reductions in the peak systolic gradient across the coarcted segment ($p<0.001$) and in the proximal systolic arterial pressure ($P<0.001$) for the whole group. There was also a significant increase in the diameter of the coarcted segment ($P<0.001$). There were no deaths related to the procedure. One patient developed aortic tear and false aneurysm with left haemothorax, needing

surgical intervention. This patient was a 40 year old male, on oral anticoagulant therapy for prosthetic aortic valve. Another patient had a large haematoma at the site of arterial puncture, which was treated conservatively. Two patients had loss of distal pulses. All other patients were discharged home within 48 hours after the procedure.

Follow-up results The data at follow-up in comparison with the basal and immediate post-dilatation data are shown in Table 2. Figure 1 shows the peak systolic pressure gradient across the coarcted segment for each patient before, immediately after and at follow-up. Despite improvement as a group, the gradient did not fall to satisfactory levels immediately after dilatation in all patients. In 13 patients the result could be graded as good in both the immediate as well as late follow-up study. In 2 patients the immediate postdilatation fall in gradient was not satisfactory but a further fall in the gradient was noted at follow-up, and there was a small but definite increase in the diameter of the coarcted segment for the whole group at follow-up(Table 2). In 3 patients a satisfactory reduction in the pressure gradient was obtained by angioplasty, but an increase in the trans-coarctation gradient (recoarctation) was found at follow-up (Figure 1).

Among 21 patients who underwent follow-up catheterisation fifteen patients had good result (group A). Three patients had aneurysm formation (group C), while the remaining three patients showed poor result (group B). In addition, 5 of the 12 patients, who were followed clinically and by doppler echocardiography had poor results, as judged by the doppler gradient and the blood pressure difference between the upper and lower extremities. These five patients were also included in group B (poor results) for purposes of further analysis, as they were not willing to have a follow-up catheterisation study. No patient was considered to have a good late result in the absence of follow-up catheterisation. Three patients underwent repeat balloon dilatation at the follow-up study.

Factors related to the late outcome after angioplasty

Twenty two factors listed in Table 3 to 5 were examined to determine their usefulness in predicting a successful outcome of balloon angioplasty at follow-up. On univariate analysis three anatomical factors were significantly different between group A and group B. The absolute size of the coarcted segment was smaller (p 0.04) and the ratio of the isthmic size to the size of coarcted segment was larger (p 0.01) in group A as compared to group B. In addition the ratio of the size of descending thoracic aorta immediately distal to the

coarcted segment to the size of the isthmus was smaller in group A(p 0.01). No physiological variable appeared to be significantly different between the two groups. Among the technical factors examined (Table 5) the ratio of the balloon diameter to the size of the coarcted segment was significantly higher in group A than in group B (4.19 $\pm$ 1.34 versus 2.54 $\pm$ 0.65, p 0.001).

Table 6 shows the effect of these four factors on the residual gradient at follow-up. The coarcted segment measured more than 4.0 mm/m^2 of body surface area in 8 patients. The residual gradient in these 8 patients was not significantly different from the remaining 15 patients. The residual gradient was significantly higher in the presence of the following risk factors: 1) ratio of isthmic size to size of coarcted segment, less than 3.0 (p 0.04); 2) ratio of descending aortic diameter just distal to the coarcted segment to the isthmic diameter, more than 1.75 (p 0.1) and 3) balloon size to coarcted segment size ratio of less than 3.0 (p 0.02). The effects of multiple risk factors on the results of angioplasty are examined in Table 7. The probability of a poor result in a given patient increased with the presence of a larger number of risk factors. Multivariate stepwise logistic regression analysis identified only two risk factors, which were related to the outcome after angioplasty 1) size of the

balloon in relation to the size of the coarcted segment (p 0.02), and 2) ratio of descending aorta immediately distal to the coarctation to the size of the isthmus (p 0.09, Borderline significant) (Tables 3 and 5). A larger balloon diameter in relation to the coarcted segment, and a larger isthmic diameter in relation to the post-coarctation aortic diameter indicated a higher probability of good result after balloon angioplasty.

Factors affecting aneurysmal complication following angioplasty

One patient needed early surgical intervention for aortic tear leading to left haemothorax and a false aneurysm. Two other patients aged 7 and 14 years respectively, were noted to have small "aneurysms" at the site of dilatation at follow-up. The aneurysms were located on the inner curvature of the aorta and measured less than the diameter of the descending thoracic aorta. No intervention was considered necessary. On further follow-up for 2 years with serial x-rays and angiogram (1 patient),the patients have remained well with no increase in aneurysm size. Table 8 shows a comparison of some anatomical, physiological and technical factors in patients with good result after balloon angioplasty (group A) and in patients who developed aneurysms (group C). All three patients in group C had excellent reduction in the transcoarctation pressure gradient, the mean residual gradient

at follow-up being 5 mm Hg. These patients were found to have a smaller isthmus in relation to the size of ascending aorta and in relation to the size of the coarcted segment. The size of post-coarctation descending aorta to isthmus size ratio was also found to be larger in this group. Further it was noted that the ratio of the balloon size to the size of the coarcted segment was larger in group C, and the balloon diameter exceeded the size of the isthmus in all three patients in this group. Statistical significance could not be shown for these differences, due to the small number of observations. But the trend suggested that a satisfactory reduction in the gradient was achieved at the cost of overstretching the aorta, with balloon diameters exceeding the size of the isthmus.

D I S C U S S I O N

This study documents the results and the factors affecting the results, in a fairly large number of patients past infancy, in whom cardiac catheterisation and angiographic follow-up data were available after balloon angioplasty of native coarctation. It was a selected group of patients with coarctation considered suitable for balloon angioplasty, patients with long-segment coarctation and coarctation with any adjacent vascular anomaly being excluded. Previous studies (10) have shown that patients with discrete

coarctation have lower residual gradients than patients with other types of coarctation. The presence of associated patent ductus arteriosus has been implicated in causing death during balloon angioplasty in infancy (7,11). Balloon dilatation relieves obstruction by causing intimal and inner medial tear (12), while the aim is to make a controlled tear, it may extend through the media into the adventitia, resulting in extravasation or late aneurysm formation. The tear could also extend into the side branches or an aneurysm,if located at the site of coarctation.

Persistent high residual gradient at follow-up could be due to failure of dilatation or restenosis at the site of dilatation (5,6,8,9). Eight patients in this study had poor results at follow-up. In 5 patients the balloon angioplasty failed to relieve the gradient, and the gradient persisted at follow-up. Three patients had recurrence of stenosis at the site of coarctation. It was, however, interesting to note that the immediate post-dilatation residual gradient, decreased further in 2 patients at follow-up. Even among patients who has successful dilatation there was a further decrease in gradient by more than 10 mm Hg in 3 patients and there was a definite further increase in the size of the coarcted segment for the whole group. Similar findings have been reported by Suarez de Lezo et al (10). These authors have found

significant remodelling of the aorta on follow-up after balloon angioplasty (10). These findings would indicate that the definitive residual gradient and the late results cannot be evaluated early after dilatation. Moreover none of the factors listed in Tables 3 to 5 were useful in predicting the result, when the immediate post-dilatation gradient was considered. When the late results were analysed, some anatomical and technical risk factors could be identified which were associated with poor results of balloon angioplasty. These included 1) size of the balloon in relation to the size of the coarcted segment; a ratio of less than 3.0 was associated with poor result; 2) size of the isthmus in relation to the size of post-coarctation descending aorta; a relatively smaller isthmus predisposed to poor outcome; 3) size of the isthmus in relation to the size of the coarcted segment; smaller the isthmus (ratio<3.0) greater the probability of failed angioplasty and larger residual gradients at follow-up.

On multivariate analysis, however, only the first two of these three factors were found to be significantly related to the outcome. Patients with good results at follow-up had significantly smaller coarcted segment diameters before dilation (Table 3, p 0.04), as compared to patients who had poor result. But this did not appear to be an independent

risk factor affecting the outcome, and the predilatation coarctation diameter did not affect the residual gradients at follow up (Table 6). Few other studies have reported on the determinants of the outcome after balloon angioplasty (5,8,10). Rao et al (8) found that a small aortic isthmus was useful in predicting recurrence of coarctation. Other risk factors identified in that study (8) included age less than one year and size of coarcted segment before angioplasty (<3·5mm) and after angioplasty (<6.0mm) respectively. Unlike our findings they found a smaller coarcted segment (<3·5mm) was associated with a higher chance of recoarctation. A systolic pressure gradient across the coarctation of greater than 50 mm Hg has been reported to be associated with recurrence of coarctation (5). Our findings do not support this observation. Like Rao et al (8) we found that presence of multiple risk factors was associated with a higher probability of poor result.

This study also attempts to look at the factors associated with aneurysm formation after balloon angioplasty for coarctation of aorta. Patients who developed aneurysmal complication were compared with patients who did not develop aneurysms despite a satisfactory reduction in the gradient. Patients with aneurysm formation had relatively smaller isthmic diameters, and the diameter of the balloon used

exceeded the size of the isthmus in all 3 cases. The average balloon size to coarcted segment size ratio, was larger in them. We believe that overstretching of the aorta by a relatively large balloon in patients with relatively small isthmus, can cause complete rupture of the media and progression to aneurysm, while relieving the obstruction effectively. Cystic medial necrosis of the aorta is a common finding in aortic coarctation and may also contribute to the development of an aneurysm in some patients (13).

The present study showed that nearly two-thirds of patients beyond 1 year of age and having discrete native coarctation, can undergo successful and safe balloon dilatation, the good result being sustained at follow-up. With the recognition of risk factors, it should be possible to improve the results further by proper selection of patients and by using balloons appropriate for the patient's aorta. Based on our findings, we would recommend balloon angioplasty for any patient with discrete coarctation, in whom the isthmus is large enough to permit use of a balloon, measuring at least three times the diameter of the coarcted segment. Use of smaller balloons is likely to leave significant residual gradients. A larger balloon size in relation to the size of coarcted segment may be necessary in some patients to produce effective dilatation, but it would be advisable not to exceed

the diameter of the aortic isthmus.

A C K N O W L E D G E M E N T S

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Table 1. Haemodynamic and angiographic findings before and after angioplasty in 46 patients

	Basal	Postdilatation
SAP (mm Hg)	159.2 ± 26.6	146.1 ± 25.9*
PSG (mm Hg)	52.1 ± 18.5	18.6 ± 14.8*
Size of coarcted segment/BSA (mm/m^2)	3.6 ± 1.7	9.1 ± 3.2*
Increment of size of coarcted segment (mm/m^2)		5.49 ± 2.53

* $P < 0.001$ Comparing postdilatation with basal values.

SAP = systolic arterial pressure in ascending aorta,

PSG = peak systolic gradient across coarcted segment,

BSA = body surface area.

Table 2. Haemodynamic and Angiographic findings in patients before and after angioplasty and at follow-up.

	Basal	Postdilatation	Follow-up
SAP (mm Hg)	160.3 ± 30.8	146.6 ± 27.4*	140.1 ± 24.5^{+}
PSG (mm Hg)	56.1 ± 20.9	20.6 ± 15.9**	20.8 ± 15.8^{++}
Size of coarcted segment/BSA (mm/m^2)	3.4 ± 1.3	8.9 ± 3.0**	10.1 ± 2.6^{++}‡
Increment of Size coarcted segment (mm/m^2)		5.5 ± 2.4	6.95 ± 2.62

* $P < 0.01$ and ** $p < 0.001$ comparing postdilatation with basal values; + $p < 0.01$ and ++ $p < 0.001$ comparing basal with follow-up values; ‡ $p < 0.01$ comparing values at followup with postdilatation values.

SAP = systolic arterial pressure in ascending aorta;

PSG = peak systolic gradient across coarctation;

BSA = body surface area.

Table 3. Anatomical and Physiological factors before angioplasty in 23 patients.

	Group A (Mean ± SD or number)	Group B (Mean ± SD or number)	p Value	
			t	Logistic
No. of patients	15	8		
Age	16.9 ± 8.0	14.3 ± 8.8	NS	NS
Sex M:F	12:3	7:1	NS	NS
Size of Isthmus/ BSA (mm/m^2)	13.6 ± 4.18	12.96 ± 5.69	NS	NS
Size of Coarcted segment/BSA (mm/m^2)	3.03± 1.13	4.43± 1.42	0.04	NS
Size of Isthmus/ size of aorta proximal to right innominate artery	0.59 ± 0.13	0.58 ± 0.19	NS	NS
Size of Isthmus/ size of coarcted segment	4.76 ± 1.35	2.88 ± 1.03	0.01	NS
Size of DAO immediately distal to coarcted segment/ size of coarcted segment	7.29 ± 2.63	5.59 ± 1.56	NS	NS
Size of DAo at diaphram/size of coarcted segment	5.92 ± 2.96	4.08 ± 0.84	NS	NS
Size of DAo immediately distal to coarcted segment/ size of isthmus	1.52 ± 0.27	1.98 ± 0.47	0.01	0.09
Systolic pressure proximal to coarctation (mm Hg)	164.6 ± 31.6	155 ± 29.6	NS	NS
Peak systolic gradient across coarctation (mm Hg)	57 ± 24.8	51.5 ± 13.8	NS	NS

NS = Not significant; BSA = body surface area;

DAo = descending aorta.

Table 4. Anatomical and Physiological factors after angioplasty in 23 patients

	Group A (mean±SD) (n = 15)	Group B (mean±SD) (n = 8)	p Value	
			t	Logistic
Size of coarcted segment (mm/m^2)	8.68 ± 2.38	9.07± 4.70	NS	NS
Size of coarcted segment after angioplasty/size of coarcted segment before angioplasty	3.17±0.92	1.8±0.59	0.01	NS
Size of isthmus/ size of coarcted segment after angioplasty	1.57±0.36	1.46±0.18	NS	NS
Size of DAo immediately distal to coarcted segment/ size of coarcted segment after angioplasty	2.45±0.77	3.12±1.09	NS	NS
Size of DAo at diaphram/size of coarcted segment after angioplasty	1.93±0.92	2.47±1.13	NS	NS
Systolic pressure proximal to coarctation(mm Hg)	146.7±26.7	149.1±34.3	NS	NS

DAo = descending aorta, NS= not significant.

Table 5. Technical factors during balloon angioplasty in 23 patients

	Group A (mean± SD) (n= 15)	Group B (mean±SD) (n=8)	p Value	
			t	Logistic
Balloon size/size of coarcted segment	4.19± 1.34	2.54± 0.65	0.001	0.02
Balloon size/size of isthmus	0.88± 0.18	0.91±0.32	NS	NS
Balloon size/size of DAo immediately distal to coarcted segment	0.6 ± 0.17	0.47±0.22	NS	NS
Balloon size/BSA (mm/m^2)	11.6±2.76	10.75±3.65	NS	NS
Balloon size/size of DAo at diaphragm	0.80±0.25	0.64±0.25	NS	NS

NS = not significant; DAo = descending aorta;

BSA = body surface area.

Table 6. Factors affecting residual gradient at followup after angioplasty

	Residual gradient (mm of Hg)	p Value
Size of coarcted segment/BSA (mm/m^2)		
⩽4.0 (n=15)	21.5± 16.6	NS
>4 (n=8)	22.9±14.5	
Size of isthmus/ size of coarcted segment		
⩾ 3.0 (n=17)	17.8± 11.9	0.04
< 3 (n=6)	33.7± 19.9	
Size of DAo just distal to coarcted segment/size of isthmus		
< 1.75 (n=16)	18.4± 14.1	0.1
⩾ 1.75 (n=7)	30.0± 16.7	
Size of balloon/ size of coarcted segment		
< 3.0 (n=10)	31.0 ± 18.3	0.02
⩾ 3.0 (n= 13)	15.0 ± 8.6	

BSA = body surface area. NS= Not significant

Table 7. Influence of multiple risk factors on the result of balloon angioplasty

Number of risk factors	Number of cases	Poor result	p Value*
≥1	12	7	< 0.02
0	11	1	
≥2	8	7	< 0.001
<2	15	1	
3	3	3	< 0.03
<3	20	5	

* Fisher's exact test.

Risk factors : 1) Balloon size/size of coarcted segment, ratio < 3.0; 2) Isthmus size/size of coarcted segment, ratio < 3.0; 3) size of post coarctation descending aorta /size of isthmus, ratio≥1.75.

Table 8. Comparison of patients with good result of angioplasty and patients with aneurysm formation

	Group A (mean± SD) n = 15	Group C (mean±SD) n = 3
Peak systolic gradient at follow-up	16.5± 10.1	5.0 ± 5.0
Size of isthmus/size of aorta proximal to right innominate artery	0.6 ± 0.13	0.43 ± 0.10
Size of isthmus/size of coarcted segment	4.76 ± 1.35	4.09 ± 1.44
Size of DAo immediately distal to coarcted segment /size of isthmus	1.52 ± 0.27	2.0 ± 0.86
Size of balloon/size of coarcted segment	4.2 ± 1.34	5.2 ± 2.85
Size of balloon/size of isthmus	0.88 ± 0.18	1.22 ± 0.31

Group A : Patients with good result of balloon angioplasty.

Group C : Patients with aneurysm formation.

DAo : descending aorta.

BALLOON PULMONARY VALVULOPLASTY: FACTORS DETERMINING SHORT AND LONG-TERM RESULTS.

SUMMARY

Balloon pulmonary valvuloplasty (BPV) was performed in 139 patients (age 2-44 years) with pulmonary valve stenosis. The right ventricular peak systolic pressure (RVSP) decreased from 137.1 ± 46.8mm of Hg to 76± 51.3mm of Hg ($p < 0.001$) and the right ventricle - to - pulmonary artery peak systolic gradient (RV-PA gradient) decreased from 116.3± 49 mm of Hg to 54.4 ± 51.9mm of Hg ($p < 0.001$). There was no significant change in systemic artery systolic pressure (SASP). The RVSP/SASP ratio decreased from 1.13 ± 0.41 to 0.63± 0.42 ($p<0.001$). Patients with incomplete immediate relief of obstruction (RV-PA gradient > 35 mm of Hg) had higher RVSP (161.1± 45.3mm of Hg vs 93.9 ± 38.8 mm of Hg, $p<0.001$) and higher RVSP/SASP ratio (1.31 ± 0.42 vs 0.98± 0.33, $p<0.001$) and were older (17.2 ± 8.6 years vs 12.8±9.7 years, $p<0.01$). The residual Rv-PA gradients in majority of patients were infundibular, which regressed at followup even in patients who did not receive long term oral beta blockers. Followup catheterisation done in 79 patients after 13 ± 8.7 months showed a further fall in RVSP ($p<0.001$) and RV-PA gradient ($p<0.001$). As assessed by followup catheterisation data, 81% had no significant residual RV-PA gradient while 4 patients showed significant increase in gradients compared to

immediate post BPV. A higher postdilatation RVSP/SASP ratio was predictive of an unsatisfactory late result. Among patients with dysplastic pulmonary valve only those with mild degree of dysplasia improved. In conclusion, BPV is safe and provides long term relief of obstruction in majority of patients with pulmonary valve stenosis. Older patients with more severe stenosis are more likely to develop infundibular gradients. Infundibular gradients regress at followup with or without beta blockers.

Key words: Pulmonary valve stenosis, interventional catheterisation.

INTRODUCTION

Balloon pulmonary valvuloplasty (BPV) has replaced surgery as the initial treatment of choice for congenital pulmonary valve stenosis since it was first reported by Kan et al in 1982 (1). Many studies have documented it's short term & long term efficacy (2-18). Few studies, however, have analysed the factors determining the results of BPV (3,4,7,9,10,13,18). The purpose of this study is to investigate the factors which can predict the immediate and late results of BPV.

METHODS

During the 62 month period ending July 1991, BPV was attempted in 139 patients with pulmonary valve stenosis. There were 77 males and 62 females and their ages ranged from 2 to 44 years (mean 14.9± 9.4 years). Right ventricular peak systolic pressure (RVSP) was suprasystemic in 62 patients, systemic in 32 patients and subsystemic in 45 patients. Associated anomalies are listed in Table 1 and valve characteristics as noted in two dimensional echocardiography and angiocardiography are listed in Table 2.

BPV was performed by standard percutaneous methods described earlier (1,2). The size of balloon used was 118.4 ± 16.1% of the measured pulmonary valve annulus. The annulus was measured by parasternal echocardiography before catheterisation. In 8 patients with poor echocardiographic window, annulus was measured from the lateral projection of angiocardiogram after correcting for magnification as described by Sievers et al (19). Predictive annulus diameters were derived from normative angiocardiographic data related to body surface area (19). A single balloon was used in 102 patients, two balloons in 33 patients and trifoil balloons in 4 patients. In 1 patient with inferior vena cava

interruption, pulmonary artery was entered from the femoral vein via azygos vein and superior vena cava with the help of a balloon flotation catheter. It was then replaced with an 0.038" Teflon coated exchange wire over which the dilatation balloon catheter was introduced. Haemodynamic assessment as listed in Table 3 were obtained before and after valve dilatation.

Ninety two patients were available for followup for 15.5± 11.8 months. Electrocardiogram and Doppler echocardiographic examinations were repeated at every followup visit. Followup catheterisation and angiocardiography was performed in 79 patients 3 to 42 months (mean 13 ± 8.7 months) after BPV. Five patients underwent repeat BPV.

Good result was defined as a right ventricle - to - pulmonary artery peak systolic gradient (RV-PA gradient) of less than 36 mm of Hg. This cut off point was chosen because it represented a reasonable gradient above which intervention might be considered. The immediate postdilatation or acute results were classified into 2 groups, Group A with good result and Group B with incomplete relief of obstruction or poor result. Thirty five patients in Group B were given long term oral beta blockers. Thirty nine patients of Group A and

40 patients of Group B underwent repeat catheterisation. The results at followup catheterisation were again divided into two groups, Group C with good results and Group D with incomplete relief of obstruction or poor result.

Statistical Methods:

Data are presented as mean value ± SD. Pooled data were compared using student's t test and chi-square test. A p value of less than 0.05 was considered significant.

R E S U L T S

The haemodynamic findings before and after BPV in the total group of 139 patients are shown in Table 3. There was a significant reduction in RVSP ($p<0.001$) and in RV-PA gradient ($p<0.001$). There was no significant change in systemic artery systolic pressure (SASP). There was also significant reduction in RVSP/SASP ratio ($p<0.001$). As shown in Table 4, 74 patients had good results and 65 patients had incomplete relief of obstruction as assessed by immediate postdilatation peak to peak RV-PA gradient of more than 35mm of Hg. Patients in group B were older ($p<0.01$) and had higher RVSP ($p<0.001$). higher RV-PA gradient ($p<0.001$) and higher RVSP/SASP ratio ($p<0.001$). Amplitude of R wave (1mV=10mm) in VI lead was lower in patients with good results ($p<0.001$).

At repeat cardiac catheterisation after 13 ± 8.7 months there was a further significant reduction in RVSP ($p<0.001$) and RV-PA gradient ($p<0.001$) (Table 5.). 35 patients of Group A and 29 patients of Group B had good result at followup (p = NS). As shown in Table 6 patients with incomplete relief of obstruction at followup had higher immediate post dilatation RVSP ($p<0.01$), higher postdilatation RV-PA gradient ($p<0.01$) and higher postdilatation RVSP/SASP ratio ($p<0.01$).

Immediately after dilatation 73 patients (53%) had both valvar (13 ± 12.6mm of Hg) and infundibular (49.3± 52.7mm of Hg) gradients. Fortyone of these 73 patients underwent repeat catheterisation after 11.6 ± 7.7 months. While there was no significant change in valvar gradient (11.6 ± 10mm of Hg vs 16 ± 10.1 mm of Hg), there was a significant reduction in the subvalvar gradient (53.5 ± 53.5 mm of Hg vs 8.2 ± 15mm of Hg, $p<0.001$) at repeat study in all but one patient.

Twentyseven patients did not have typical doming pulmonary valve stenosis and had varying degrees of dysplasia. As shown in Table 6 no particular characteristic of dysplastic valve was associated with uniformly poor results. Three patients had 4 or more characteristics of

dysplastic valve and in them only one patient had good result immediately after dilatation. At restudy he had significant increase in RV-PA gradient. Of the 2 patients with incomplete relief of obstruction one was restudied and he had persistent obstruction. The only patient with calcific valve had infundibular gradient only.

Of the 40 patients in Group B who underwent restudy 25 had received long term oral beta blockers. 15 patients did not receive any drug. In the group of patients who received beta blockers the RVSP before dilatation was 184.8 $\pm$ 40.3 mm of Hg. At followup catheterisation after 13 $\pm$ 9.2 months the RVSP was 52.3 $\pm$ 20mm of Hg ($p<0.001$). In the group of patients who did not receive any drug, the predilatation RVSP was 138.3 $\pm$ 41.9 mm of Hg. At followup catheterisation after 14.9 $\pm$ 11.7 months the RVSP was 59 $\pm$ 41.7 mm of Hg ($p<0.001$). Patients who received beta blocker had significantly higher predilatation RVSP ($p<0.01$). Seventeen patients in the group which received beta blockers and 12 patients in the group which did not receive any drug improved (p = NS).

Acute results were good in 56% of patients with significant TR, 43% of patients with systemic desaturation and 40% of patients with Noonan syndrome. At followup 75% patients with significant TR (3/4), 66% patients with

systemic desaturation (6/9) and 33% patients with Noonan syndrome (1/3) had good results. BPV failed in both patients with heart failure and they were sent for surgery. There was no significant increase in left to right shunt by oxymetry in any of the patients with secundum atrial septal defect or small ventricular septal defect, who underwent BPV. In all the patients with right to left shunt at the atrial level the shunt decreased.

Complications: Most patients developed transient hypotension and bradycardia during balloon inflation. However, 10 patients had significant bradycardia and hypotension persisting after balloon deflation and required atropine and intravenous fluids. Patients with interatrial communication tolerated the procedure better and showed no drop in SASP during balloon inflation. Four patients had significant bleeding from puncture site requiring blood transfusion. One patient developed atrial fibrillation which later reverted to sinus rhythm. One patient developed complete right bundle branch block which persisted at followup. Pulmonary regurgitation developed in 86% of patients but in only 3 patients it was more than mild in severity. The only mortality was in a 6 year old girl with typical doming pulmonary valve stenosis. She also had persistent left superior vena cava draining into coronary sinus. Measured

pulmonary valve annulus was 14 mm and a single 18mm balloon (3 cm long) was used to dilate the valve. The procedure was uneventful with good result and she was stable till she had sudden cardiac arrest, 6 hours after the procedure. The exact cause of death could not be ascertained as autopsy could not be done. Unfortunately, she was the only patient who was not monitored in the ICU following the procedure.

DISCUSSION

This study documents the results and factors predicting the results in a large number of patients. BPV was successful in 54% of patients immediately after the procedure and 81% of patients at long term followup.

Patients with incomplete relief of obstruction immediately after dilatation were older and had more severe pulmonary valve stenosis as characterised by higher RVSP, higher RV-PA gradient, higher RVSP/SASP ratio and higher amplitude of R wave in VI lead of electrocardiogram. The incomplete relief of obstruction in most of these patients were due to residual gradients at infundibular level. Data from the VACA Registry study (4) has also shown that most of the residual gradients are at the infundibular level and the higher the degree of stenosis before BPV the higher the

infundibular gradient immediately after BPV. Infundibular gradient have been recorded after surgical valvulotomy in older patients with severe pulmonary valve stenosis and some surgeons have advocated resecting the hypertrophied musculature at the level of the infundibulum thus completing the pulmonary valvar commisurotomy (20,21). Brock et al (22) recorded the appearance of a systolic gradient at infundibular level after pulmonary valvotomy in cases of severe pulmonary valve stenosis, postulating 2 possible mechanisms to explain it. The first was a mechanical source in the region of the hypertrophied supraventricular crest and the second was an exaggeration of the tonus of the infundibular musculature during ventricular systole.

Regression of infundibular gradient as noted in our study has been reported earlier (12,18). Regression of infundibular hypertrophy has been noted after surgical pulmonary valvulotomy (23). Age and severity of stenosis could not predict long term result as these factors were mainly predictive of a residual infundibular gradient which tended to regress at followup. An intravenous propranolol test has been used as a method of determining whether muscular or fibrous structures are the cause of persistent residual stenosis after surgical valvulotomy (24). However, the infundibular gradient did not decrease in the

catheterisation laboratory in any of the 8 patients who received propranolol (2 to 6mg) intravenously. Rey et al (15) had similar experience in 4 patients. The infundibular gradient regressed in patients who received beta blockers as well as in patients who did not receive the drug. Although the two groups are not strictly comparable, it appears that beta blockers are not necessary.

Persistent high residual gradients at followup could be due to failure of dilatation or incomplete regression of infundibular gradient or rarely restenosis. Only 4 of the 79 patients restudied showed significant increase in RV-PA gradient at followup as compared to immediate post BPV. High post dilatation RVSP, high postdilatation RV-PA gradient and high postdilatation RVSP/SASP ratio were predictive of persistently high RV-PA gradient at followup.

Results of BPV in patients with dysplastic pulmonary valve are variable depending upon criteria used to diagnose dysplastic valves (4,7,14,15,25-28). As apparent in Table 6 no particular characteristic of dysplastic valve was associated with uniformly poor results. Patients with 4 or more characteristics of dysplastic valves had incomplete relief of obstruction.

Balloon size was not predictive of immediate postdilatation or followup results as adequate size balloons were used in almost all patients. Mc Crindle et al (18) have found significantly less, ratio of the measured pulmonary annulus to predicted pulmonary annulus diameter, in patients with poor long term results. Our findings do not support this observation.

The present study showed that BPV is safe and effective for relief of obstruction due to pulmonary valve stenosis in majority of patients. In older patients with severe stenosis the immediate postdilatation results may not be satisfactory because of residual infundibular gradients. Infundibular gradients regress over time. Beta blockers are not necessary for the infundibular gradients to regress.

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TABLE 1

ASSOCIATED ANOMALIES IN 139 PATIENTS WITH PULMONARY VALVE STENOSIS

	No. of patients
Secundum Atrial Septal Defect	21
Right to left shunt at atrial level	14
Significant Tricuspid regurgitation	9
Noonan syndrome	5
Small Ventricular Septal Defect	4
Congestive heart failure	2
Peripheral pulmonary stenosis	2
Left Superior vena cava to coronary sinus	2
Inferior vena cava interruption	1
Partial anomalous pulmonary venous drainage	1
Ebstein anomaly	1
Wolf Parkinson White Syndrome	1

TABLE 2

IMMEDIATE POSTDILATATION AND FOLLOWUP RESULTS IN PATIENTS NOT HAVING TYPICAL DOMING PULMONARY VALVE STENOSIS

	Acute result (No. of patients)		Late result (No. of patients)	
	Good	Poor	Good	Poor
Markedly thickened valve	4	10	7	3
Absent or minimal mobility of valve cusps	2	3	2	2
Hypoplastic annulus	5	9	9	2
No post-stenotic dilatation	1	2	0	2
Poorly formed sinuses	3	5	2	2
Supravalvar tethering	3	5	3	1
Calcific valve	0	1	0	1

TABLE 3

HAEMODYNAMIC FINDINGS BEFORE AND IMMEDIATELY AFTER BALLOON PULMONARY VALVULOPLASTY IN 139 PATIENTS

	Basal	Postdilatation
RVSP (mm of Hg)	137.1 ± 46.8	76 ± 51.3*
RV-PA gradient (mm of Hg)	116.3 ± 49	54.4 ± 51.9*
SASP (mm of Hg)	123.1 ± 19.9	123.4 ± 19.4
RVSP/SASP	1.13 ± 0.41	0.63 ± 0.42*

* P <0.001 comparing postdilatation with basal values.

RVSP = right ventricular peak systolic pressure.

RV - PA gradient = right ventricle - to - pulmonary artery peak systolic gradient, SASP = Systemic artery systolic pressure.

TABLE 4

FACTORS PREDICTING RESULTS IMMEDIATELY AFTER BALLOON VALVULOPLASTY IN 139 PATIENTS.

	Group A (Mean ± SD or number)	Group B (Mean ± SD or number)
No. of patients	74	65
Age (in years)	12.8 ± 9.7	17.2 ± 8.6+
Sex M:F	43:31	34:31
RVSP (mm of Hg)	116.1 ± 37	161.1 ± 45.3*
RV-PA gradient (mm of Hg)	93.9 ± 38.8	141.3 ± 46.8*
RVSP/SASP	0.98 ± 0.33	1.31 ± 0.42*
Balloon/measured annulus	116.8 ± 14.4%	120.2 ± 17.8%
R wave in V_1 (mm)	16.3 ± 8.7	23.3 ± 13.7*
Measured annulus/ predicted annulus	0.99 ± 0.14	0.97 ± 0.12

* P <0.001 comparing Group A and Group B. + P<0.01 comparing Group A and Group B. RVSP = right ventricular peak systolic pressure, RV-PA gradient = right ventricle -to-pulmonary artery peak systolic gradient, SASP = systemic artery systolic pressure.

TABLE 5

HAEMODYNAMIC FINDINGS BEFORE AND IMMEDIATELY AFTER BALLOON PULMONARY VALVULOPLASTY AND AT FOLLOWUP IN 79 PATIENTS

	Basal	Postdilatation	Followup
RVSP (mm of Hg)	143.3 ± 49.6	79.6 ± 50.1*	51.1 ± 24.5+
RV-PA gradient (mm of Hg)	122 ± 52.3	57.7 ± 51*	28.2 ± 24.4+
SASP (mm of Hg)	128.1 ± 20.1	127.2 ± 19.2	
RVSP/SASP	1.15 ± 0.45	0.64 ± 0.42*	

* P<0.001 comparing postdilatation with basal values;

+ P<0.001 comparing followup with postdilatation values.

RVSP = right ventricular peak systolic pressure.

RV-PA gradient = right ventricule-to- pulmonary artery peak systolic gradient, SASP = systemic artery systolic pressure.

TABLE 6

FACTORS PREDICTING RESULTS AT FOLLOWUP AFTER BALLOON VALVULOPLASTY IN 79 PATIENTS.

	Group C (Mean ± SD or number)	Group D (Mean ± SD or number)
No. of patients	64	15
Age (in years)	16.6 ± 10.4	16.7 ± 8.1
Sex M:F	31:33	10:5
RVSP before dilatation (mm of Hg)	138.1 ± 44.5	165.5 ± 64.5
RV-PA gradient before dilatation (mm of Hg)	116.8 ± 47.3	144.2 ± 67.1
RVSP/SASP before dilatation	1.13 ± 0.44	1.24 ± 0.47
R wave in V_1 (mm)	18.2 ± 10.3	21.4 ± 16.8
Balloon/Measured annulus	118.6 ± 16.7%	123.2 ± 19.8%
Measured annulus/ predicted annulus	0.96 ± 0.14	0.96 ± 0.12
RVSP after dilatation (mm of Hg)	72.7 ± 41.4	111.5 ± 70.5*
RV-PA gradient after dilatation (mm of Hg)	49 ± 38.3	89.5 ± 71.2*
RVSP/SASP after dilatation	0.59 ± 0.35	0.9 ± 0.59*

* $P<0.01$ comparing Group C and Group D. RVSP = right ventricular peak systolic pressure, RV-PA gradient = right ventricle -to- pulmonary artery peak systolic gradient, SASP = systemic artery systolic pressure.