Aneurysm of the Sinus of Valsalva

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There are three localized bulgings in the aortic root opposite the 3 cusps of the aortic valve, they are commonly known as the sinuses of Valsalva. Untill recently they were mainly of interest to the anatomist and physiologist because of the origin of Coronary arteries from these sinuses. Since the introduction of Coronary Angiography the exact localization of these sinsuses for purpose of selective catheterization of the coronary artery ostia, proper orientation of these sinuses is of great importance for this purpose. However, before the present era only rarely was this name mentioned in clinical meetings in association with aneurysm arising from these sinuses and subsequent rupture into one of the cardiac chamber. This disease remained mostly an autopsy diagnosis till surgical correction and newer diagnostic techniques were available. While it remains a rare condition in the western countries, report of Sakakibara et al from Japan stressed their increased prevalence in 1962(1). Since 1976 we have studied 8 cases of this anomaly and in the present paper their clinical, haemodynamic angiographic and results of surgical correction will be discussed.

Materials and Methods

8 cases were diagnosed in the cardiac cath. lab. of NICVD since February 1976.

Table I gives their age, sex and the clinical diagnosis for which they were referred for study. The average age for ruptured aneurysm is 31 years. 7 patients were males and one female. 4 cases had clinical diagnosis of A.S., A.I. and M.S. M.I. Three cases had diagnosis of P.D.A. and one of V.S.D. was found to have unruptured aneurysm of right sinus of Valsalva. All these patients had history, physical examination, X-ray chest P.A. lateral and R.A.O. with Ba swallow and E.C.G. in addition to routine lab work.

Table No. I

	Name	*Age*	*Sex*	*Clinical Diagnosis*
1.	A.R.	35 Yrs.	M	A.S.; A.I.; M.I.; T.I.
2.	M.R.	37 Yrs.	M	PDA (?) Aorto Pulmonary window.
3.	Mrs F.	34 Yrs.	F	PDA? P.H.
4.	D.L.	24 Yrs.	M	A.S.; A.I.
5.	M.A.	31 Yrs.	M	A.S.; A.I.; M.S.; M.I.
6.	M.H.	12 Yrs.	M	V.S.D.
7.	M.Y.	38 Yrs.	M	A.S.; A.I.
8.	A.L.	29 Yrs.	M	P.D.A.

Results

Table II & III show their clinical profile. The presenting complaint of our patients was dyspnea on exertion and palpitation, only one had history of paroxymal nocturnal dyspnea. In six out of 8 cases the onset of symptoms was insidious and 4 patients were aware of buzzing sensation in the chest. Only one patient Mrs. F. could exactly tell the acute onset of symptoms associated with lifting of her baby, followed by palpitation, sweating and weakness and something running in the chest. She was taken to hospital where she was told to have a P.D.A. and heart failure.

Table II Clinical Profile

	NYHA Class	*P.N.D.*	*Chest Pain*	*Palpitation*	*Leg Swelling*	*Buzzing Sensantion in Chest*	*Onset*
A.R.	III	Nil	+	+++	—	—	Insidious
M.R.	III	+	+	+++	++	—	Sudden
Mrs. F.	II	—	++	+++	—	+	Sudden
D.L.	III	—	++	+++	—	+	Insidious
M.A.	III	+	+	+++	—	—	Insidious
M.H.	II	—	—	+++	—	—	Insidious
M.Y.	III	—	++	+++	—	+	Insidious
R.L.	II	+	++	+++	—	—	Insidious

Table III Clinical Profile

	J.V.P.	*Liver*	*Pedal-edema*	*MURMUR Type/Intensity*	*Location*	*E.C.G.*	*X-RAY Heart Size*	*CHEST P.A. Segments*
1. A.R.	+	+	—	Continous V/VI	L.S.B. 3 & 4	N.S.R. Biven Hypert	Gross Cardio Megaly	
2. M.R.	+	++	++	Continous IV/VI	L.S.B. 3 & 4	,,	—do—	Enlarged
3. M.F.	—	—	—	Continous V/VI	,,	L.V.H.	Normal	Normal
4. D.L.	—	—	—	Continous 5/6	,,	L.V.H.	Cardio-megaly+	Normal
5. M.A.	—	—	—	Continous 4/6	,,	L.V.H.	++	Normal
6. M.H.	—	—	—	Holosystilic 4/6	L.B.S. 3	Bivent Hypeit.	+	Enlarged
7. M.Y.	—	—	—	Continous 4/6	L.S.B.	L.V.H.	+	Normal
8. A.L.	+	+	—	Continous 4/6	L.S.B.	Bivent Hyp.	+	Normal

Table IV Haemodynamic and Angiographic Findings

No.	Name	O2 SATURATION %								PRESSURE mmHg										Angio, Final Dx. Aortic Root/LV
										R.A.						WEDGE				
		SVC	IVC	RA	RVIF	RVOF	MPA	W	AORTA	a	v	m	RVIF	RVOF	MPA	a	v	m	AORTA	L.V.	
1.	A.R.	74	75	82	84	86	86	95	92	10	15	10	55/0-10	55/0-10	55/35	12	14	12	140/60	140/0-12	(1) Large finger like projection from RCS opening into RVOF (2) 2+A.I.
2.	M.R.	72	74	80	84	86	86	97	94	15	25	12	80/0-15	80/0-15	70/50	20	22	18	140/40	140/0-20	(1) RCS Large aneurysm rupture into RV (2) L.V.F., R.V.F., T.I.
3.	M.F.	75	76	76	82	83	82	98	95	5	3	2	40/0-5	40/0-5	40/20	10	12	8	150/50	140/0-10	Ruptured R.C.S. aneurysm into R.V.O.F. No A.I., No V.S.D.
4.	D.L.	75	77	75	86	86	85	98	95	5	4	3	55/0-5	40/0-5	40/30	12	14	10	150/60	150/0-12	(1) Ruptured R.C.S. aneurysm in R.V.O.F. (2) V.S.D.
5.	M.A.	74	76	86	86	86	86	98	95	10	12	10	60/0-10	60/0-10	60/40	15	16	12	140/40	140/0-15	Ruptured R.C.S.A. into R.A.; A.I.—nil. Aortic catheter into R.A.
6.	M.H.	80	80	80	84	84	85	98	96	5	2	2	45/0-5	40/0-5	40/25	8	5	6	120/70	120/0-8	(1) Unruptured R.C.S. aneurysm (2) V.S.D.+.
7.	M.Y.H.	78	80	80	85	86	86	97	95	4	3	3	50/0-4	50/0-5	40/30	12	10	10	160/50	160/0-12	(1) Large R.C.S. aneurysm (2) Coronary Arteries Normal. (3) A.I.—nil.
8.	A.L.	78	78	79	82	84	84	98	96	5	4	4	35/0-5	35/0-5	30/20	12	12	10	140/50	140/0-12	Ruptured R.C.S. aneurysm into R.V.O.F.

Seven patients had characteristic 4-5/6 continuous murmur with maximal intensity at left sternal border in the 3rd and 4th inter-costal space, one patient with unruptured aneurysm had grade 4/6 pan systolic murmur at LSB3.

All patients were in N.S.R. with slight sinus tachycardia at times. P wave amplitude was high in 2 cases, isolated L.V.H. in 4 cases and biventricular hypertrophy in the rest. Although pulmonary plethora was present in all cases, the main pulmonary artery and its segments were dilated in only 2 cases. Gross cardiomegaly was present in 4 cases, in the other ruptured aneurysms there was moderate enlargement of the cardiac silhouette. Left atrium was enlarged in 2 cases.

All patients underwent Right and Left heart catheterization and aortic root injection.

Table IV shows pressure and O_2 saturation in these cases. In two cases, there was 8-10%, step-up in right atrium and in the rest in right ventricle. There was mild P.H. in 6 cases; in one case P.H. was severe and the wedge pressure was also high.

Aortic root injections confirmed the presence of finger like projection from the right coronary sinus with fistulous communication in R.A. in one case and R.V. in 6 cases. In one case the aneurysm was unruptured. V.S.D. was present in 2 cases only.

Table V shows follow-up of these cases. 4 cases underwent surgery; one case has residual A.I. other 3 cases are asymptomatic and no residual defect. One case died while waiting for surgery. 2 cases refused surgery and were still living 2 years following cardiac catheterization.

Table V

	Type	*Sinus Affected*	*Part of Sinus Occurs*	*Chamber into which perforation*	*Associated V.S.D.*
I. Congenital	I	R.C.S.	ANT (Left)	R.V. Just below the pulmonary valve.	—
	I VSC	R.C.S.	ANT	,, ,, ,,	Present
	II	R.C.S.	Middle	R.V. The outflow tract at C.S.V.	—
	II VSD	R.C.S.	Middle	,, ,, ,,	Present
	III	R.C.S.	Posterior	R.V. below the medial leaflet of T.V.	—
	III (a)	R.C.S.	Posterior	Just above the septal leaflet of T.V.	—
	IV	N.S.C.		R.A.	—
II. Acquired	(1) Multiple Coronary Sinuses Affected. R & Non Coronary Commonly and Left also Affected. Secondary to: (*a*) Syphilis. (*b*) S.B.E. (*c*) Marfan's Syndrome (*d*) Trauma (*e*) Rheumatoid Arthritis. (*d*) Dissecting Aneurysm of the Aorta.				

R.C.S.—Right Coronary Sinus.
C.S.V.—Crista Supraventricularis.
N.C.S.—Non Coronary Sinus.
T.V.—Tricuspid Valve.

Discussion

There are 3 sinuses at the root of the aorta just above the aortic valve; opposite the 3 aortic cusps. Two are anterior and one is posterior. They have been variously named however since 1929 this terminology has been widely adopted. The two anterior sinuses are named after their respective coronary ostia. That is right coronary sinus and left coronary sinus and the posterior coronary sinus is called the non coronary sinus. The right coronary sinus (R.C.S.) is the most anterior of these. It should be rememebered that the words "coronary sinus" apply to the venous drainage of the heart and thus right, left and posterior sinus of valsalva may be preferable to avoid confusion.

The right coronary sinus is the most anterior in 60° LAO while the left coronary sinus is anterior in 30° LAO; however it is not the most anterior in this projection and in left lateral projection it is towards the spine.

The non coronary sinus (posterior coronary sinus) is at a lower level than these two. The term aortic sinus should be reserved for the part of ascending aorta joining the arch of aorta.

Figure No. 1(a) shows the relationship of these sinuses to the adjacent structures. The right coronary sinus has three main relationships. In its most anterior 3rd (left 3rd) it is related to the commissure between the right and left posterior pulmonary cusps. Thus an aneurysm arising from this part will protrude in to the right ventricular outflow just below the pulmonary valve. The second part or middle 3rd of the R.C.S. is related with outflow tract at the crista supraventriculares. The posterior 3rd of the R.C.S. is related with the inflow tract of the right ventricle, and an aneurysm arising from this part of the coronary sinus will protrude just below the septal leaflet of the tricuspid valve. If the aneurysm takes a superior and posterior course it will protrude into the right atrium just above the medial leaflet of the tricuspid valve. The noncoronary sinus is related to right atrium in its right 3rd. Interatrial septum middle 3rd and left atrium in the left 3rd. below the plane of the aortic valve it is related with the interventricular septum and L.V. The medial leaflet of the mitral valve is also continuous with this, Figure 3.

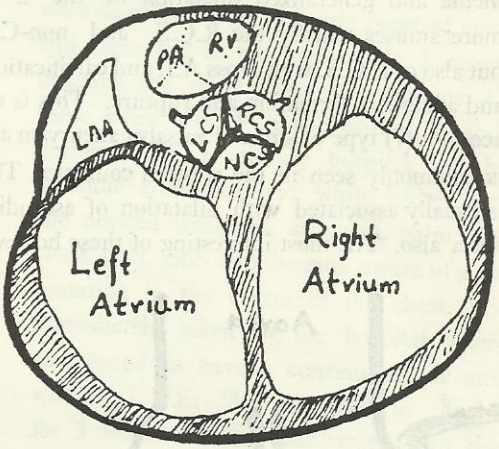


Fig. No. 1(*a*)

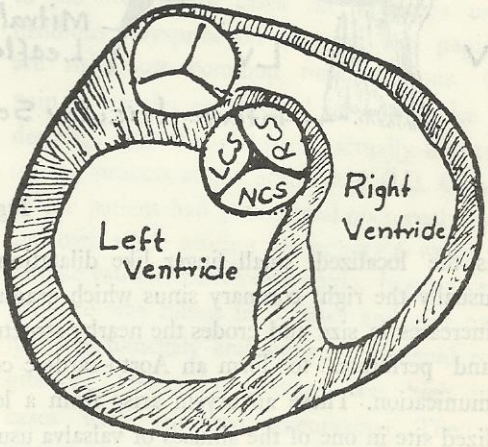
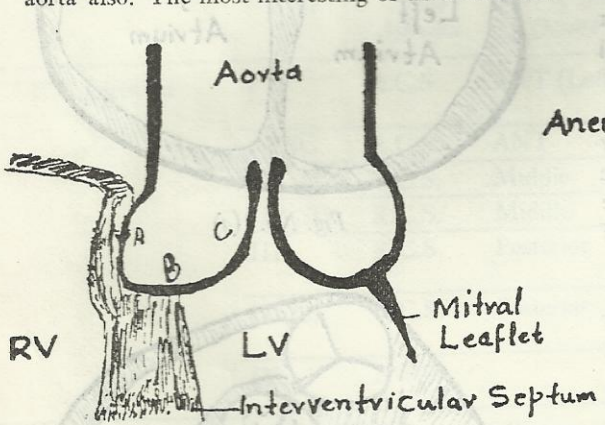


Fig. No. 1(*b*)

Figure No. 1(a) and (b) shows the relationship of these sinuses to the ventricle and atria respectively. The left coronary sinus is related above to the left atrium below to L.V. and anteriorly to pericardium.

These sinuses may be affected by conditions like syphilitic aortitis(2), Marfan's syndrome(3), Rheumatoid arthritis(4), ankylozing spondylitis, S.B.E.(5,6) or trauma and lead to weakness of the media and generalized dilatation of the 2 or more sinuses usually the R.C.S. and non-C.S. but also of L.C.S. with gross A.I. and calcifications and aneurysm formation and rupture. This is the acquired(7) type of sinus of valsalva aneurysm and is commonly seen in the western countries. This is usually associated with dilatation of ascending aorta also. The most interesting of these however



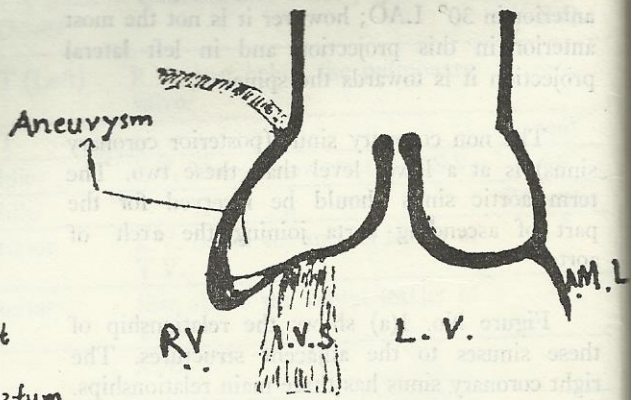


Fig. No. 3

is the localized small finger like dilatation of usually the right coronary sinus which gradually increases in size and erodes the nearby structures and perforates to form an Aorto-cardiac communication. These aneurysm arise from a localized site in one of the sinuses of valsalva usually site "A" shown in figure 3. Then it gradually increases in size to involve B&C also and later perforates at its tip. The pathological anatomy of congenital aneurysm of sinus of valsalva has been reveiwed by Edward and Burchall in 1957(8). They attribute the formation of an aneurysm and this site to weakness as a result of lack of fusion of aortic media with the annulus fibrosus of the aortic valve. This may be due to failure of fusion of the bulbar septum or of the conus or truncus ridges.

The congenital aneurysm commonly arise from the R.C.S. in our cases all of them arose from R.C.S. in Japanese series nearly 97% from the R.C.S. while in six European series 84% R.C.S., 13% N.S.C. and 3% L.C.S. As mentioned earlier multiple sinus involvement and left sinus involvement is usually due to acquired cases but at times it is quite difficult to differentiate between these two groups esp. cases following S.B.E.(9). Sakikabara and others have classified these aneurysms into six groups and 16 subgroups according to the site of origin; presence or absence of V.S.D. and the site of perforation(1). Table V summarizes the com-

monly occuring groups. 89% of our cases belonged to group I & I VSD and 11% IV while in Japanese series type I 60%. Type II 11% Type III 11%. Type IV 2%. Type V nil. Type VI (R.C.S. in Bicuspid aortic valve with coarctation of the aorta) 5% (10) (Figure 2).

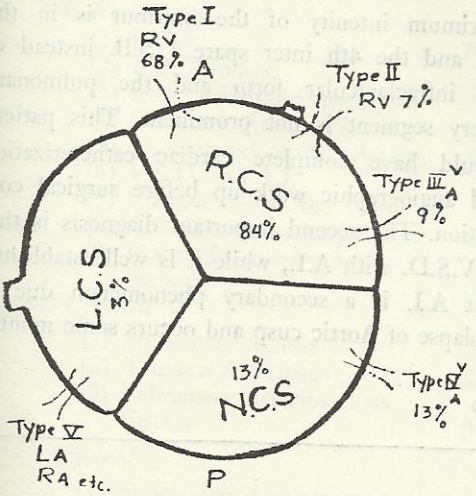


Fig. 2

Seven of our patients presented after rupture of the aneurysm and one patient had signs and symptoms due to a V.S.D. and on investigation was found to have an unruptured aneurysm of the sinus of Valsalva.

While the weakness due to separation of aortic media and annulus fibrossus is congenital, these aneurysm usually rupture in the late 3rd decade or early 4th decade. Early cases were described with dramatic onset of symptoms and signs but in our series only 2 cases could describe the exact onset of symptoms. One of these cases Mrs. F. was followed clinically for pregnancy in a military hospital and had no murmur after delivery; while lifting her baby she noticed weakness, sweating, palpitation and chest pain. She also became aware of a running sensation in the centre of the chest. She was immediately taken to the hospital where she was found to have a continuous murmur and was in L.V.F. She remained in the hospital for 3 weeks and was discharged with the final diagnosis of P.D.A. This history is quite characteristic and is found only in few cases and in the majority of cases the onset is usually insidious. Dyspnea on exertion and papitation are the most common manifestations. Chest pain is usually retrosternal but otherwise nondescript. It is thought to be actually due to the tearing process at the aortic root (11). Only one of our patient had paroxysmal nocturnal dyspnea and died while waiting for surgery 6 weeks after cardiac catheterization and angio. Jugular venous pressure was raised in 3 cases and one patient had hepatomegaly and signs of tricuspid regurgitation. The pulse pressure was high in all cases with ruptured aneurysms. The most important clinical finding is the presence of continuous murmur at the left sternal border

increasing with expiration. The most important differential point is the location of the murmur at the left sternal border with maximum intensity in 3rd and 4th inter costal spaces(12). Our patient A.R. also had significant A.I. and the diastolic component was more prominent than the systolic component, in intensity at the 4th and 5th spaces along the left sternal border, auscultation limited to this area may have lead to diagnosis of Aortic insufficiency only.

Table No. VII gives other causes for a continuous murmur. The most important differential diagnosis however in these cases is aortic valve disease with AS, AI, P.D.A. Aortopulmonary window and V.S.D. with A.I.

rupture requires a median sternotomy incision and cardiopulmonary bypass.

Table No. III shows the important points of differentiation. However generally speaking if the sex is male, age is above 20 years and the maximum intenity of the murmur is in the 3rd and the 4th inter spare L.S.B. instead of left infraclavicular form and the pulmonary artery segment is not prominent. This patient should have complete cardiac catheterization and angiographic work up before surgical correction. The second important diagnosis is that of V.S.D. with A.I., while it is well established that A.I. is a secondary phenomenon due to prolapse of Aortic cusp and occurs some months

Table VI Follow Up

No.	*Name*	*From onset of Symptoms to Cath. duration in Mths.*	*Since Cath Months*	*Since Surgery Months*	*Living/Dead*
I	A.R.	24	40	38	Living
II.	M.R.	8	3	—	Dead
III.	Mrs. F.	12	18	—	Lost to follow up after 18 months.
IV.	D.L.	16	14	13	Living
V.	M.A.	28	7	6	Living
VI.	M.Y.	24	2	—	Living.
VII.	A.L.	12	42	39	Living.

In A.S., A.I. the murmur is not continuous while in ruptured aneurysm of Valsalva it is continuous. Thus the most important differential diagnosis to be made clinically is P.D.A. This is also very important in view of the operative procedure involved in the two conditions. While P.D.A. closure is done from the left lateral approach, sinus of valsalva aneurysm

even years after birth in patients born with VSD, it is not settled whether these cases of A.I. are associated with some degree of aneurysmal dilatation of the sinuses of Valsalva.

Sakakibara and S. Konno(13) (1968) have divided cases of V.S.D. and A.I. in 4 groups on the basis of 55 operated cases and 15 autopsied

Table VII Continuous Murmurs

	No of cases
(A) INNOCENT	
(1) Venous Hum	3
(2) Mammary Souffle	2
(B) ORGANIC	
(*a*) *Congenital*	
I. Non Cyanotic	
(1) P.D.A.	70
(2) A.P. Window	1
(3) Rup. S. Valsalva Aneurysm	7
(4) Coronary Artery Fistula	0
(5) Anomalous Coronary Artery	0
(6) V.S.D. and A.I.	3
(7) Coarctation of the Aorta	2
(8) Abberant Pulmonary Artery	0
(9) Branch Pulmonary Artery Stenosis	0
II. Cyanotic	
(1) Pulmonary Atresia	2
(2) Truncus Arteriosus	2
(3) Pulmonary Arteriovenous Fistula	0
(*b*) *Acquired*	
(1) Subclavian Ateriovenous Fistula	2
(2) Internal Mammary A.V. Fistula	0
(3) Blalock Taussig Shunt	8
(4) Renal Dialysis A.V. Shunt	0
(5) Secondary A.S.D.	1
Total No. of cases	103

(*c*) To and Fro murmur confused with continous murmur: A.S.+A.I.

specimens. In Type 1 V.S.D. the aneurysm is just below the pulomnary valve, 3 stages are described. In stage I the aneurysm is small and does not bulge in to R.V. and A.I. is the main manifestation, in stage 2 the aneurysm bulges into the R.V. in hemispherical fashion.

In stage 3 spherical aneurysm occludes the V.S.D. and is ready to burst into R.V. While stage 2 and 3 are definitely examples of Aneurysms of valsalva associated with V.S.D.; stage I is doubtful. In type II V.S.D. the aneurysm arises in the middle 3rd of R.C.S. and V.S.D. is subscristal. In type III V.S.D. the V.S.D. is subcristal, the aneurysm is in the posterior part of the R.C.S. In Type IV V.S.D. aneurysm arises from the N.C.S.

Tagushi et al have also described cases of type V and type VI. Type VI V.S.D. is the one when there is bicuspid aortic valve and aneurysm arises from it. Van Praagh R.(14) has given pathological classification of 11 autopsied cases having VSD and A.I. while he has used the words "ballooned" for the sinuses, it appears that specimens were not examined for aneurysms of sinuses.

We have studied 3 cases of V.S.D. with A.I.; In these 3 cases distinct aneurysm of the sinuses could not be seen in the aortic root angiograms and these were not included in this series. However they would correspond to stage I of Type I V.S.D. of Sakakibara et al.

In our own opinion the formation of aneurysm or prolapse of cusps depend upon the anatomy of the case. If the weakness due to lack of support begins at site A in figure 3 than aneurysm formations occurs 1st and A.I. may be later, but if the lack of support leads to weaknes at B or C then A.I. and prolapse of the cusps occurs 1st. If both occur then there is aneurysm as well as A.I.

It is extremely important to determine the presence of significant A.I. associated with sinus of valsalva aneurysms because aortic valve replacement is needed in these cases having significant A.I.

The unruptured aneurysms are only diagnosed when aortic root angio is done for some other conditions e.g. V.S.D. However these aneurysms may produce signs and symptoms of pulmonary outflow obstruction(15); severe tricuspid regurgitation, complete heart block, angina pectoris, myocardial ischaemia and even sudden death (16).

Bulkley and Ross reported 5 cases of Acquired unruptured sinus of valsalva aneurysms; 4 of them died suddenly and one in intractable C.H.F. All of them had clinical diagnosis of pulmonary stenosis and 2 of them tricuspid regurgitation. Aneurysms were responsible for outflow obstruction in these cases.

Lateral chest X-rays in acquired cases may show calcifications however in congenital cases the heart is usually enlarged but the pulmonary artery segments are not dilated. Cardiac cath and aortic root injection is necessary to confirm the diagnosis. If the aneurysm ruptures into R.A. there is usually 10-12% step up of O_2 saturation, however, this may be due to tricuspid incompetance as in our case 2. If the aneurysm ruptures into R.V. there is usually step up in R.V. outflow. The aortic catheter may be manipulated across the defect. Echocardiography(16, 17) may be helpful. However, angiography is necessary prior to surgery for confirmation and actual localization. Cardiac cath and aortic root injection is necessary to confirm the diagnosis(18, 19). Selective injection of Urographin 76 per cent into aortic root will delineate the aneurysm as well as the communication.

It is interesting to note that the 1st case was described by James Hop in 1831(20, 21) and the 1st successful surgical correction was done by Lillehei in 1957(22). Since then Sakakibara, Tagushi, Bahey(22) and others(23) have published reports of surgical correction in their cases. All agree that surgery should be done as soon as ruptured sinus of valsalva aneurysm is diagnosed because without surgery most cases would die in intractable C.H.F. In cases where there is no associated V.S.D. the onset and course is more dramatic and severe while in cases with V.S.D. the survival may be longer. Leaving a few exceptions the mean survival is 1-2 years after diagnsois. In our cases 4 had surgery with no mortality. One case has residual A.I., the others are asymptomatic. Patients requiring Aortic valve replacement and repair of aneurysm will have higher mortality than these requiring repair alone. These patients should be observed for S.B.E. and recurrence after surgery.

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