Cardioaortic Fistula

Temporary Circulatory Occlusion as an Aid in Diagnosis

By Bernard L. Brofman, M.D., and John C. Elder, M.D.

Rupture of a sinus of Valsalva aneurysm into the right ventricle produces a dramatic clinical syndrome characterized by a rapid and relentlessly unfavorable course. Early diagnosis is essential for surgical repair before an irreversible stage is reached. Cardiac catheterization and retrograde aortography confirmed the diagnosis in the case reported. With the aid of temporary circulatory occlusion, the fistula was demonstrated by contrast material injected via an aortic catheter. Although the fistula and the accompanying ventricular septal defect were closed at open-heart surgery, the markedly enlarged heart could not resume its function.

Sudden rupture of an aneurysm of a sinus of Valsalva into a low pressure chamber of the heart has always been considered a medical curiosity and of only academic interest. Only rarely has a definitive diagnosis been made prior to exploratory operation or autopsy.\textsuperscript{1-4} Unruptured congenital aneurysms of the sinuses of Valsalva are probably not too rare\textsuperscript{6} and have been demonstrated in life by angiography;\textsuperscript{7-8} they are presumably of little hemodynamic significance. However, once rupture occurs, a medical emergency exists. This catastrophe always results in rapid circulatory deterioration, with death supervening within a period ranging from a few days to several years. Recent advances in intracardiac surgery render such a condition amenable to surgical correction. The diagnosis must be established soon after rupture occurs, otherwise the relentless deteriorating course may well preclude later diagnostic and therapeutic measures.

The clinical picture appears to be so typical that the diagnosis should be suspected without special studies. However, an absolute diagnosis can be established only by demonstrating the shunt, with the aid of suitable technics. In the case presented here the clinical picture was correctly interpreted as that of rupture of a sinus of Valsalva into the right ventricle. The special studies were, of course, confirmatory, and demonstrated the exact localization of the lesion, thus determining the surgical approach.

The actual demonstration of the fistula was accomplished with the aid of a new diagnostic technic devised to meet the immediate needs of this case. Temporary circulatory occlusion reduced circulation to the degree that the retrograde injection of contrast material into the aorta demonstrated the fistula from the right sinus of Valsalva into the right ventricle.

Operation was carried out with the aid of an artificial heart-lung apparatus. Unfortunately, the markedly dilated and hypertrophied heart fibrillated even before the machine had taken over the circulation. The lesion was repaired as planned. However, even with successful repair, irreversible failure precluded recovery.

Case Report

A 26-year-old white man was known to had have a heart murmur since childhood. There was no history of rheumatic fever or other serious childhood disease. He had been completely asymptomatic with no limitation of activity until June 1955, at which time he suddenly developed easy fatigability and dyspnea on slight exertion. There was no undue exertion or chest pain associated with the onset of symptoms. It was reported that there was a marked change in the heart murmur at that time. Orthopnea and pedal edema appeared within a few days of the onset of symptoms and became rapidly worse. He was placed on strict bed rest, digitalis, and frequent mercurial diuretics with rather striking improvement, and he was able to return to a sedentary job within a few weeks. However, in December 1955, his symptoms had progressed to the point where he was completely incapacitated, the slightest activity causing severe dyspnea.
He was first hospitalized in February, 1956. At this time he did not appear to be in acute distress, but was obviously chronically ill. The skin appeared to have a slightly cyanotic tinge. The peripheral arterial pulses were bounding; the brachial artery pressure (by sphygmomanometer) was 140 systolic; the diastolic pressure was usually read as 50 to 60 although muffled sounds could be heard to 0. The heart was markedly enlarged; the apex beat was at the anterior axillary line in the sixth intercostal space.

The heart sounds were of normal intensity except for accentuation of the pulmonic second sound. There was a very loud harsh continuous machinery-like murmur heard best in the third left intercostal space 5 cm. from the midsternum. The murmur was widely transmitted over the precordium (fig. 1). A readily palpable continuous thrill was also present. The lungs showed occasional basilar rales. The liver was palpable 2 cm. below the costal margin. Urine and blood studies were essentially normal.

Cardiac fluoroscopy revealed a markedly enlarged heart, with generalized chamber enlargement (fig. 2). The pulmonary artery was prominent with increased pulsations; the aortic knob was small. The electrocardiogram showed combined ventricular hypertrophy.

The abrupt onset together with the clinical findings suggested the diagnosis of rupture of a sinus of Valsalva into the heart. Cardiac catheterization was carried out, followed by angiocardiography.

The patient was then discharged, to return for definitive surgery. During this period he remained at home, on digitalis, receiving weekly mercurial injections with no progression of his symptoms. He was readmitted in August 1956, at which time the final angiographic studies were carried out and open-heart surgery was eventually performed.
HEMODYNAMIC STUDIES

Cardiac catheterization was performed using a special triple-lumen catheter with an inflatable cuff. This type of catheter has been used in this laboratory to produce unilateral pulmonary artery occlusion and in determination of the size of intracardiac defects. Simultaneous samplings in adjacent chambers were obtained via the different lumens. The catheter readily entered a markedly dilated right atrium, but there was considerable difficulty in manipulating it into the right ventricle and then into the pulmonary artery. Pressures were recorded via electromanometers and a Sanborn Polyviso. Duplicate blood samples for determinations of oxygen content were obtained from the various chambers.

Percutaneous insertion of a plastic catheter into the left femoral artery permitted arterial blood sampling and pressure recording. Oxygen consumption was determined by means of a Collins respirometer.

The pulmonary "capillary" (wedge) pressure could not be obtained in the usual manner. In order to do so, the right pulmonary artery was occluded temporarily by inflating the cuff with 10 ml. of contrast material. Distal to occlusion, the pressure and blood oxygen content were obtained. These are considered equivalent to "pulmonary capillary" determinations.

Table 1 summarizes the hemodynamic data obtained at right heart catheterization and during subsequent retrograde arterial studies. The abrupt increase in blood oxygen content in the right ventricle indicates a left-to-right shunt at the level of the outflow tract of the right ventricle. The slight degree of desaturation of arterial and "pulmonary capillary" blood is consistent with congestive heart failure. There is no evidence of a right-to-left shunt. The calculated flows and shunt are, of course, merely approximations, but do reveal a considerable left-to-right shunt with significantly reduced systemic flow.

In figure 3 are demonstrated the various pressure tracings obtained during right heart catheterization. Although the "pulmonary capillary" pressure is only moderately elevated

<table>
<thead>
<tr>
<th>Table 1.—Hemodynamic Data</th>
<th>Pressure (mm. Hg)</th>
<th>O₂ content (vol.%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior vena cava</td>
<td>18/10</td>
<td>9.7</td>
</tr>
<tr>
<td>High right atrium</td>
<td>18/10</td>
<td>10.6</td>
</tr>
<tr>
<td>Low right atrium</td>
<td>18/10</td>
<td>11.5</td>
</tr>
<tr>
<td>Right ventricular inflow</td>
<td>65/14/18 (mean 28)</td>
<td>13.9</td>
</tr>
<tr>
<td>Right ventricular outflow</td>
<td>65/14/18 (mean 28)</td>
<td>16.4</td>
</tr>
<tr>
<td>Main pulmonary artery</td>
<td>60/28 (mean 38)</td>
<td>15.1</td>
</tr>
<tr>
<td>Left pulmonary artery</td>
<td>60/28 (mean 38)</td>
<td>15.0</td>
</tr>
<tr>
<td>Right pulmonary artery</td>
<td>60/28 (mean 38)</td>
<td>15.0</td>
</tr>
<tr>
<td>&quot;Pulmonary capillary&quot;</td>
<td>20/10</td>
<td>17.0 (91%)</td>
</tr>
<tr>
<td>Femoral artery</td>
<td>150/50 (mean 75)</td>
<td>17.0 (91%)</td>
</tr>
<tr>
<td>Brachial artery</td>
<td>150/70</td>
<td></td>
</tr>
<tr>
<td>Ascending aorta</td>
<td>140/80</td>
<td></td>
</tr>
<tr>
<td>Left ventricle</td>
<td>140/4/20</td>
<td></td>
</tr>
</tbody>
</table>

Blood O₂ capacity: 18.5 vol. %.
O₂ Consumption: 260 ml./min.

Systemic flow: \[
\frac{260}{17.0 - 10.0} = 3.7 \text{ L./min.}
\]

Pulmonary flow: \[
\frac{260}{17.0 - 15.0} = 13.0 \text{ L./min.}
\]

Left-to-right shunt: 9.3 L./min.

Fig. 3. Pressure curves obtained at right heart catheterization. Pressures in mm. Hg. Pulmonary artery (P.A.) and "pulmonary capillary" (P.C.) curves are superimposed, demonstrating considerable pulmonary artery hypertension with only a moderately elevated "capillary" pressure. The right ventricular (R.V.) diastolic pressure and right atrial (R.A.) pressure are high, indicating right ventricular failure. The normal pressure gradient across the tricuspid valve indicates normal function of that valve.
there is considerable pulmonary hypertension, indicating an increase in pulmonary arteriolar resistance. The right ventricular curve shows an elevated end-diastolic pressure preceded by a shallow diastolic "dip." The elevated right atrial pressure is consistent with the ventricular diastolic pressure, with the tricuspid valve functioning normally.

Following cardiac catheterization, a specially constructed balloon-tipped catheter was inserted via the same vein into the right atrium. Approximately 40 ml. of 70 per cent Diodrast were instantaneously liberated in this chamber, while x-ray exposures at 1/2-second intervals were made. However, satisfactory visualization of the defect could not be obtained despite the large bolus of contrast material.

Retrograde aortic catheterization was carried out by means of a no. 7 cardiac catheter inserted via the left ulnar artery. Under fluoroscopic and manometric control, the catheter was advanced to the root of the aorta, and then through the aortic valve into the left ventricle. Repeated attempts were made to enter the suspected fistula, without success. However, during exploration of the left ventricle, the right ventricle was entered. The pressure transition from right ventricle to left ventricle to aorta established the presence of a ventricular septal defect (fig. 4).

In figure 5 are shown pressure curves from the left ventricle, ascending aorta, and the brachial and femoral arteries. The left ventricular curve exhibits a small diastolic dip and an anacrotic notch. The elevated end-diastolic pressure and "dip" are evidence of failure, with a relatively hypodynamic left ventricle. The aortic curve demonstrates a good incisura with a well maintained diastolic pressure. Even the brachial artery pulse pressure is somewhat less than would be expected from the cuff measurements. The femoral artery curve exhibits a significantly elevated pulse pressure.

Retrograde aortography was performed with the injection of 70 per cent Diodrast via the no. 7 catheter at the root of the aorta. However, it was impossible to obtain visualization of the ascending aorta by this method. The marked cardiac enlargement made x-ray penetration particularly unsatisfactory.

Subsequently, a thin-walled no. 8 catheter was inserted via the left brachial artery to the root of the aorta. During bilateral carotid artery compression, 20 ml. of 70 per cent Diodrast were rapidly injected 3 times, again with complete lack of visualization. Up to this point the patient had received a total of at least 110 ml. of 70 per cent Diodrast within a 6-month period, without untoward reaction.

**Angiographic Demonstration of Fistula**

Although immediate open-heart surgery was recommended in view of the probable diagnosis of cardioaortic fistula, there was considerable resistance to such an undertaking. It was argued that such possibilities as atypical patent ductus and aortic septal defect had not been adequately ruled out. As a matter of fact, in a patient ultimately proved to have a congenital aneurysm of a coronary artery we had heard a murmur very similar to that in the present case.

Exploratory thoracotomy was strongly considered. In order to obviate such a move, a new diagnostic technic was developed and successfully employed:

Since rapid manual injection of 70 per cent Diodrast is particularly difficult via a long catheter (and against aortic pressure), an automatic injection apparatus was constructed. This consisted of a metal syringe, the tip of which was connected to the compressed air line via a simple stop-cock. The plunger of the syringe was fitted with a metal cap shaped to adapt to the barrel of a standard 50 ml. glass syringe. The 2 syringes were firmly mounted in series on a wood block, so that as compressed air forced the plunger back up the metal syringe, the barrel of the

![Fig. 4. Pressure curves during withdrawal of the catheter from right ventricle (R.V.) to left ventricle (L.V.) to aorta (A.O.), demonstrating the presence of a ventricular septal defect.](image-url)
Fig. 5. Pressure curves from left ventricle (L.V.), ascending aorta (A.), brachial artery (B.A.), and femoral artery (F.A.).

glass syringe was forced in, resulting in rapid ejection of the contained contrast material, with no danger of air embolism. With such an apparatus, 25 ml. of 70 per cent Diodrast could be injected via a no. 9 catheter in 1½ seconds.

However, even with rapid injection of large volumes of contrast material, we still failed to demonstrate the defect. It was then suggested that temporary reduction of ventricular ejection might encourage retrograde perfusion of the lesion with an adequate concentration of contrast material. Experimentally, it has been demonstrated that circulation can be reduced by means of increased intrathoracic pressure.12

In our extensive studies with unilateral pulmonary artery occlusion9 and with intracardiac angiography10 in unanesthetized patients, temporary circulatory occlusion had been produced inadvertently on a number of occasions. On 1 occasion in a patient with emphysema, the balloon-tipped catheter had been positioned in the right pulmonary artery. During inflation with 20 ml. of contrast material, the balloon had slipped into the main pulmonary artery producing complete occlusion for at least 15 seconds before it could be deflated. Although the patient complained of discomfort and shortness of breath, there were no alarming symptoms, and, within 1 minute of deflation, no residual effects.

In another patient suspected of having a patent ductus arteriosus, right pulmonary artery occlusion was carried out as part of a hemodynamic study. In this instance, unilateral occlusion produced a marked rise in main pulmonary artery pressure, with paroxysmal coughing and respiratory distress. The balloon was then deflated after about 20 seconds of occlusion of the right pulmonary artery and the patient was immediately comfortable, with no sequelae. Subsequent angiographic studies revealed that this patient actually had congenital absence of the left pulmonary artery, so that temporary occlusion of his right pulmonary artery actually produced more or less total obstruction of pulmonary flow for a short period.

In a third patient, suspected of having an atypical patent ductus arteriosus, it was planned to inflate the Diodrast-filled balloon in the right pulmonary artery for an intracardiac angiogram. Accordingly, the tip of the special catheter was positioned in the right pulmonary artery. Fifty milliliters of 70 per cent Diodrast were then rapidly injected into the balloon and serial x-ray exposures were made as the contrast material was released. During this period the unanesthetized patient exhibited no particular distress. However, examination of the serial x-rays subsequently showed that the balloon had slipped back into the main pulmonary artery during inflation, so that the main pulmonary artery had been occluded for at least 10 seconds, without ill-effects.

These 3 experiences (and other lesser ones) suggested to the authors that temporary obstruction of circulation, even in the unanesthetized patient, was feasible, and could be performed if indicated. Accordingly, it was decided that circumstances justified its application in this patient. Of course, occlusion had to be planned so that the dynamic consequences would aid in demonstrating the suspected lesion; that is, if a left-to-right shunt were present, the contrast material would be encouraged to go from the site of injection at the root of the aorta into a lower pressure area. Technically, the easiest area for temporary occlusion is the main pulmonary artery. This would be indicated in a suspected patent
ductus, since distal pulmonary artery pressure would fall, facilitating shunting of contrast material from aorta to pulmonary artery. However, such a maneuver would tend to raise right ventricular pressure, so that if an aorta-right ventricular shunt were present, visualization would be impeded.

It appeared that the most likely site for obstruction would be at the inflow tract of the right atrium. A balloon catheter was constructed for simultaneous occlusion of both venae cavae. However, it was decided subsequently that inferior vena cava occlusion would suffice since, during retrograde aortic injection, circulation of the head and neck was manually occluded. Application of tourniquets to both arms could then provide almost complete obstruction of venous return to the heart.

One hour prior to the procedure the patient received secobarbital, 0.1 Gm. and meperidine hydrochloride, 50 mg. intramuscularly, which resulted in only mild sedation. A no. 9 thin-walled arterial catheter was inserted via the right brachial artery and advanced to the root of the aorta. During manipulation at this point a spot x-ray was taken, which subsequently was interpreted as showing the catheter tip traversing the fistula from the aorta into the right ventricle (fig. 6). (Comparison of this spot film with the final retrograde angiogram (fig. 7B) shows the catheter in the same approximate position as the fistula.) The catheter tip was then positioned just above the aortic valve (fig. 7A).

A balloon-tipped catheter was then inserted via the right brachial vein and advanced just into the

![Fig. 6. Spot x-ray film during retrograde aortic catheterization showing aortic catheter traversing fistula from right sinus of Valsalva into right ventricle.](image-url)
inferior vena cava (fig. 7A). Heparin, 50 mg., was given intravenously to prevent fibrin deposition on the catheters. The patient was then transferred via cart to the x-ray department, where he was placed in the anteroposterior position on the x-ray table. Suitable scout films were obtained.

In accordance with a well-rehearsed plan, the inferior vena cava was occluded by inflation of the balloon with 30 ml. of 5 per cent Diodrast, bilateral manual compression of the neck was applied, and 25 ml. of 70 per cent Diodrast were rapidly injected via the aortic catheter. A single x-ray exposure was made just at the end of injection. Immediately after exposure the inferior vena cava balloon was deflated; neck compression was released a few seconds later. More or less complete circulatory occlusion had been maintained for approximately 15 seconds. The patient exhibited remarkably little discomfort during the entire procedure. As a matter of fact, examination of the film revealed that the catheter tip had been displaced into the descending aorta so that excellent visualization of the arch and descending aorta was obtained, but not of the base. Accordingly, the catheter tip was repositioned and the entire procedure repeated. Again, there was little patient reaction despite another intra-aortic dose of 25 ml. of 70 per cent Diodrast. A few minutes later the patient was able to walk unsupported back to his bed. There were no further symptoms following the slight distress during the procedure. (The unintentional angiogram of the descending aorta revealed no evidence of a patent ductus arteriosus.)

Thus, after months of careful consideration and repeated failures, our efforts were finally crowned with success in the form of a single x-ray exposure (fig. 7B). Excellent visualization of the base of the aorta was obtained; a competent aortic valve is demonstrated. The small fistula is well shown (arrow). By virtue of its position, it was presumed to originate from the right sinus of Valsalva and to enter the right ventricle. The right ventricular chamber is not demonstrated. This x-ray was considered by all concerned as final proof of presence of a cardioaortic fistula, and there was then unanimous approval of the decision to proceed with open-heart surgery.

**Operation**

Operation was performed on August 28, 1956, by Dr. Melvyn Reydmian. An artificial heart-lung machine with a bubble-type oxygenator was used. This machine is capable of maintaining a flow of at least 4 L. per minute. Induction of anesthesia was uneventful. An arterial catheter was inserted via the left femoral artery for continuous blood pressure.
registration via a strain gage-oscilloscopic assembly. A trans-sternal incision provided adequate exposure. The heart was tremendously enlarged and dilated, so that manipulation was particularly difficult. As the various cannulations were being carried out in preparation for heart-lung by-pass, atrial tachycardia developed with a rate of 150 per minute. Within a few minutes ventricular fibrillation supervened. Manual cardiac massage was instituted, maintaining a satisfactory aortic pressure while final connections were made to the heart-lung apparatus. When the heart-lung machine had taken over, 25 ml. of approximately 5 per cent potassium citrate in blood were injected proximal to a clamp across the ascending aorta, thus perfusing the coronary bed and producing complete standstill of the fibrillating heart. A mean aortic pressure of 75 mm. Hg was maintained by the machine throughout.

The pulmonary artery was huge, as were the right ventricle and right atrium. Through a long right ventriculotomy incision excellent exposure of the outflow tract of the right ventricle was obtained. Just below the pulmonic valve, at the septum, could be seen the opening of the fistula, with a small crescent-shaped septal defect just below. An irregular gray-white area of thickened endocardium extended along the interventricular septum below the perforation. With the aid of a teflon plug, the combined ventricular openings were sutured closed. Perfusion of blood into the base of the aorta caused only a slight leakage into the right ventricle. As the ventriculotomy was closed gradual release of the aortic clamp allowed the heart to be perfused with blood. The potassium was flushed from the coronary arteries. At this point the huge heart almost filled the thoracic cavity. Despite vigorous and prolonged cardiac massage and other resuscitative procedures, the heart beat could not be restored. An autopsy was performed.

**Postmortem Examination of the Heart**  
(Figs. 8A and B)

The heart weighed 820 Gm. The aorta was normal in size. The aortic valves were not deformed and there was no aortic incompetence. The coronary arteries were normal. The pulmonary artery, right ventricle, and right atrium were markedly enlarged. The right ventricle measured 1.1 cm. in thickness. At the base of the right sinus of Valsalva was an aneurysm that protruded into the outflow tract of the right ventricle. At the tip of this aneurysm was a round perforation 4 mm. in diameter. Adjacent to the perforation there was a somewhat larger crescent-shaped ventricular septal defect. An irregular endocardial "jet lesion" extended below the perforation. Grossly and microscopically the combined defects were quite similar to those described by Edwards and co-workers, and Lin and co-workers. As a matter of fact, the physiologic findings were also remarkably similar in most respects.

**Discussion**

At this time, fewer than 50 cases of rupture of a sinus of Valsalva have been reported. Within the past year, however, isolated cases
have appeared with increased frequency. On occasion, only after exploratory operation has the correct diagnosis been established. Following a definitive diagnosis, operation has been carried out in 3 reported cases,2, 3, 16 all as open-heart procedures. In 2 patients an artificial heart-lung was used,2, 16 and in the other, hypothermia. Unfortunately, in both patients operated with the artificial heart-lung, the outcome was the same as in our case. The operation under hypothermia was successful, but the patient died 14 days postoperatively of overwhelming infection. At this writing, there have been no reported cases of long-term survival after operation.

Technically, the lesion is amenable to surgery. However, operation must be performed as soon as possible after rupture has occurred. The duration of life after rupture has been reported as 7 days to 7 years.2 As in the case herein reported, a remarkable degree of hypertrophy and dilatation may occur in a short time, even though the fistula is relatively small. Presumably, a stage of irreversible failure is soon reached, so that even with successful closure of the defect, there may be no reversal of the deteriorating course.

This case demonstrates the remarkable difference in hemodynamic consequences of congenital shunts as compared to those that are suddenly acquired. Even large congenital shunts are usually well tolerated for many years, with lesser degrees of hypertrophy and dilatation. In this patient, the calculated shunt flow from left-to-right was approximately 9 L. per minute. By modifying the patent ductus formula of Gorlin and Gorlin,17 it can be calculated that a fistula of the size produced by the perforation in this case would give a shunt flow of less than 3 L. per minute, with 6 L. per minute shunting through the ventricular defect. Thus, although the interventricular shunt presumably had been well tolerated, a sudden 50 per cent increase in the shunt produced dire consequences associated with marked hypertrophy and dilatation. Although some degree of cardiac enlargement may have preceded the rupture in this case, there was no clinical evidence to that effect.

The clinical and pathologic aspects of unruptured and ruptured aneurysms of the sinuses of Valsalva have been reviewed by others.2, 3, 5, 6, 14, 15 Edwards and Burchell14 have demonstrated that the basic lesion in such cases is always the same: A lack of continuity between the aortic media and the ring of the aortic valve. The right coronary sinus, from which the right coronary artery originates, is most frequently involved, and anatomic relationships are such that it most frequently ruptures into the right ventricle. In the case reported, the defect in the ventricular membranous septum is, presumably, merely an extension of this congenital lack of continuity.

The ruptured sinus of Valsalva appears to be much more common in males, with ages varying from 20 to 67 (average: 42 years). Although there is usually severe pain at the onset, our patient exhibited only sudden severe dyspnea but not pain.

The murmur associated with the fistula is usually very loud and continuous throughout the cardiac cycle. As in our case, rupture into the right ventricle produces a murmur of maximum intensity in the third left intercostal space that seems to radiate toward the apex. The pulmonic second sound is accentuated and is associated with increased pulmonary artery pressure and flow.

The collapsing pulse and wide pulse pressure indicate a rapid aortic run-off. It is interesting to note that the sphygmanometric pulse pressure was much greater than the actual direct brachial artery determination. Furthermore, the lowest diastolic recording was in the femoral artery, whereas the pressure in the ascending aorta (near the site of the fistula) was essentially normal.

X-ray changes are not specific. Generally, there is marked cardiac enlargement, with increased pulmonary blood flow. The electrocardiogram usually shows the pattern of combined hypertrophy.

Cardiac catheterization has been carried out in a number of reported cases1-4, 18, 15 with demonstration of a left-to-right shunt. Of course, right heart catheterization does not demonstrate the origin of the shunt. However, given evidence of an interventricular septal defect, with a wide pulse pressure, rupture of a
sinus of Valsalva into the right ventricle should be considered.

Although unruptured aneurysms have been demonstrated by angiocardiography, we know of only 2 cases presumed to have ruptured aneurysms in which there was angiocardiographic confirmation. However, in only 1 of these was there retrograde demonstration of a defect, (neither with autopsy confirmation). We believe the case reported to be the first proved cardioaortic fistula demonstrated by retrograde visualization of the fistula.

The technic of temporary circulatory occlusion was particularly effective in this case in enabling us to visualize the defect. Although there is an obvious real risk in its use, it has been demonstrated that the technic can be applied effectively under special circumstances. As a matter of fact, variations of this technic are now being applied in this laboratory in a number of diagnostic problems.

SUMMARY

Sudden rupture of an aneurysm of a sinus of Valsalva results in a dramatic clinical picture, with a rapidly progressive, deteriorating course. Most frequently, the right sinus of Valsalva ruptures into the right ventricle, as in the case presented. A loud continuous murmur and thrill, wide pulse pressure, and hemodynamic evidence of a left-to-right interventricular shunt characterize this cardioaortic fistula.

Definitive diagnosis was finally accomplished in this case only with the aid of a new technic: Temporary circulatory occlusion (inferior vena cava occlusion by a balloon and bilateral manual compression of the neck) adequately reduced circulation to enable visualization of the cardioaortic fistula by means of contrast material injected via an aortic catheter. The markedly hypertrophied heart failed during open-heart surgery; function could not be restored following repair. Early recognition and repair, before a stage of irreversibility is reached, is mandatory for a condition that is appearing with increased frequency.

SUMMARIO IN INTERLINGUA

Le rupture subitanee de un aneurysma del sinus de Valsalva resulta in un frappante situacion clinic con rapide e progressive deterioration. In le majoritate del casos, le sinus dextere de Valsalva es le sito del ruptura, con apertura versu, le ventriculo dextere. Isto esseva le situation in le caso hic presentate. Iste typo de fistula cardioaortic es characterisate per un forte e continue murmure, un late pression pulsatile, e signos hemodynamic de un derivation interventricular sinistro-dextere.

In nostre caso le diagnose definitiv esseva finalmente effectuate per medio de un nove technic. Un occlusion temporari del circulazione esseva inducite per medio de un ballon in le vena cave inferior insimul con le compression manual de ambe lateres del cervice. Le resultante reduction circulatori sufficeva pro render possibile le visualisation del fistula cardioaortic per medio de un substantia de contrasto que esseva injicite via un catheter aortic. Le corde esseva marcamente hypertrophiate. Illo disfalleva durante le operation a corde aperte, e su function non poteva esser restabilite post que le reparo esseva completate. Precoce recognition del presentia de un fistula cardioaortic e prompte intervention chirurgicante ante le adventimento del stato de irreversibilitate es indispensabile in le tractamento de iste condition que occurre con crescente frequentias.

REFERENCES

8 Falholt, W., and Thomsen, G.: Congenital an-


---


The author discusses the measurement of over-all coagulability during Dicumarol therapy as indicated by measurement of the clotting time at both room temperature and at 37 C., the 4-gamma and 1-gamma heparin clotting time (HCT) and the 1-stage prothrombin time (PT). Serial coagulation studies were performed on 21 patients receiving Dicumarol and an index of over-all coagulability was obtained from the ratio of the HCT to the PT. The PT showed a rough correlation with other clotting tests, particularly with the heparin clotting time. The author attributed this to the limitations of the PT, which measured only 1 phase of the clotting mechanism and did not reflect changes in the thromboplastic factors (platelets, antihemophilic globulin, plasma thromboplastin component, plasma thromboplastin antecedent, etc.). These factors may be affected by Dicumarol (particularly PTC), or the patient's disease. The studies on patients with acute myocardial infarction revealed hypercoagulability fluctuating with hypocoagulability, superimposed on the Dicumarol effects. Hemorrhagic manifestations seemed more closely related to over-all coagulability than to prothrombin time alone.

The significance of the over-all coagulability in the control of anticoagulant therapy with Dicumarol and related drugs, particularly in relation to acute myocardial infarction was discussed. These studies suggested that the prothrombin time may be inadequate as the basis of a therapeutic range and in control of therapy with Dicumarol and related compounds.

Maxwell
Cardioaortic Fistula: Temporary Circulatory Occlusion as an Aid in Diagnosis

BERNARD L. BROFMAN and JOHN C. ELDER

Circulation. 1957;16:77-87
doi: 10.1161/01.CIR.16.1.77

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1957 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/16/1/77

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/