Cineangiocardiographic Studies of the Origin of Cardiovascular Physical Signs

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The contraction of the cardiac muscle is responsible for the movement of blood within the heart, alterations in the shape of the heart, and the movement of the heart within the chest. The cardiac physical signs are related to one or more of these consequences of the contraction and relaxation of cardiac muscle. Cardiac catheterization provides information about the pressures within the heart, and the measurement of cardiac output determines flow over several cardiac cycles, but these technics are not completely satisfactory for the study of the production of physical signs. For this purpose, measurements of pressure should be supplemented by continuous measurements of flow from chamber to chamber within the heart, volume, and the exterior shape and position of the heart. Unfortunately, these ideal circumstances cannot be achieved in man, but radiologic technics provide a fair approximation of this ideal. Cineangiocardiograms obtained at 60 frames per second provide enough information about the movements of the heart during each cardiac cycle to be of value in the study of the origin of physical signs. Intracardiac pressure and sound can be recorded and correlated with cineangiocardiography, which provides temporal information about the flow of blood through the heart and great vessels during a single cardiac cycle. Though instantaneous flow is not measured in absolute terms, it is possible with accuracy to time the onset of flow into a chamber or great vessel. In many cases, these changes in flow correspond to changes in pressure, but this is not always so. The opening and closing of valves can be observed directly and therefore timed precisely by cineangiocardiography. This precise timing of valve function is not possible by pressure measurements alone, as valve function is merely one of many factors contributing to changes in intracardiac pressure.

Common cineangiocardiographic, hemodynamic, and phonocardiographic studies demonstrate the temporal correlation between cardiac events on the one hand and sound on the other; but this demonstrated temporal correlation does not however prove causation. For example, the opening snap of the mitral valve can be recorded at a time when the front of contrast material contained behind the mitral valve has reached the point of maximum descent into the ventricle, but it is still not possible to know whether the sound is produced by the tensing of the valve leaflets or the sudden change in velocity of the bolus of blood. Though of some theoretical importance, this difficulty is of little practical significance, as the clinician uses physical signs to draw inferences about the functional anatomy of the heart, and the cineangiographic correlations serve to strengthen this relationship whatever the physical mechanism for the production of sound may be.

The methods used have been reported in detail elsewhere but in essence consist of the standard technics of right heart catheterization and the retrograde femoral and transseptal technics for left heart catheterization. An 8-inch image amplifier equipped with a television system and a 35-mm. motion picture camera were employed. Correlation between the motion picture film and records of pressure and sound was made possible by a signal generated by the R wave of the
electrocardiogram, which made a simultaneous mark on the pressure record and the motion picture film. Films were analyzed and correlations made by the study of the original 35-mm. film on an analytical projector. The film projected was a 16-mm. copy of the original 35-mm. negative which permits the addition of labeling, titling, and other supplementary marking. The value of cineangiocardiology in the interpretation of physical signs can only be appreciated by viewing the sequences in motion. Reproduction of a single frame is not a satisfactory substitute. We have attempted to simulate appropriate sequences from the motion picture with drawings made by a medical artist, Mr. Leon Schlossberg, after he viewed the motion picture.

**Normal Mitral Valve**

For mitral valve cineangiocardiology, the patient is turned into the right anterior oblique position. Figure 1 is not a drawing of a cineangiogram but rather an anatomic drawing designed to show the mitral valve as it appears when viewed from the right anterior oblique projection. The aortic leaflet of the mitral valve is toward the observer, and the mural leaflet is behind it. The anterior commissure is up and to the right and the posterior commissure down and to the left. It is apparent that in this projection, the leaflets are superimposed and will not be seen to separate as the direction of their motion in the act of opening is parallel to the x-ray beam. The mitral valve may be considered to be a sleeve, the walls of which are collapsed during ventricular systole. As ventricular pressure falls, blood from the atrium moves down and separates the two walls of the sleeve.

The action of the normal mitral valve is outlined in figure 2. Contrast material is injected into the left atrium through a catheter positioned in that chamber by the transseptal technic. The normal mitral valve is seen only in systole and is seen as a smooth, crescentic indentation in the left atrial silhouette (fig. 2A). During diastole, the normal valve becomes invisible radiographically (fig. 2B). The leaflets separate completely, and radiopaque material flows freely into the left atrium across the full diameter of the mitral annulus. The normal mitral valve in no way impedes the flow of blood into the ventricle. At the end of the period of passive ventricular filling, atrial systole occurs, and the atrial shadow shrinks (fig. 2C). With atrial systole, the mitral valve leaflets move toward closure, and the mitral valve reappears as a line of difference in roentgenographic density. With the onset of ventricular systole, the contrast gradient at the atroventricular ring is clearly seen (fig. 2A). The mitral valve begins to close prior to the onset of ventricular systole in the patient with normal sinus rhythm.

**Mitral Stenosis**

The origin of the four major physical signs of mitral stenosis can be demonstrated by cineangiocardiology. The stenotic mitral valve is visible throughout the cardiac cycle.
Normal mitral valve: Right anterior oblique projection with catheter in the left atrium. A. Systole. The plane of the closed mitral valve (M.V.) is seen separating the left atrium (L.A.) and left ventricle (L.V.). Contrast material can be seen in the initial segments of the pulmonary veins (P.V.). The left atrial appendage (L.A. app.) is seen overlying the aorta (Ao.). The leaflets of the aortic valve have moved to the open position. B. Early diastole. The plane of the mitral valve is no longer visible. Contrast material can be seen entering the left atrium across a broad front. The indentations in the ventricular silhouette represent the papillary muscles. C. End diastole-atrial systole. The atrial shadow has decreased in size, and the left atrial appendage has rocked upward. The contrast material in the left atrium has been diluted by pulmonary venous drainage, and the contrast material has been effectively transferred to the left ventricle, indicating that the mitral valve does not impair left atrial emptying.

(figs. 3A, 3B, and 3C), unlike the normal mitral valve, which is visible only in systole. The stenotic mitral valve never opens completely, and, therefore, throughout diastole there is a contrast density gradient across the valve that is analogous to the pressure gradient. Neither pressure nor the concentration of roentgenopaque material can reach equilibrium across the valve. During systole (fig. 3A), the stenotic mitral valve has the same position and configuration as the normal valve, but its abnormal thickness may be evident. With the onset of diastole, a front of contrast material contained by the valve moves down into the ventricle to form a hemispheric projection into the ventricular cavity (fig. 3B). When the descent is complete and the hemisphere fully expanded, contrast material can be seen entering the ventricle. The opening snap of the mitral valve occurs at this moment of maximum descent of the valve, which is also the moment of first flow through the valve (fig. 3B).

The beginning of the descent of the still closed mitral valve corresponds to the peak of the "V" wave of the left atrial pressure pulse. Opening of the valve and hence mitral flow does not begin until an average of 67 msec. later (fig. 3B). The moment of mitral valve opening does not correspond to a directional change in left atrial pressure but is often associated with a notch or change in the slope of the "y" descent. The opening snap is related temporally to the tensing of the tissue of the mitral valve at the end of its downward descent. This event also corresponds to a sudden change in the velocity of the bolus of blood moving downward from
the atrium into the ventricle. Either event or a combination of the two may be responsible for the opening snap.

Throughout diastole, contrast material can be seen flowing from the atrium into the ventricle through the stenotic mitral valve orifice (figs. 3B and 3C). This flow corresponds to the diastolic murmur. The flow through the mitral valve can be seen to accelerate with atrial systole at the time of presystolic accentuation of the murmur (fig. 3C). Atrial systole initiates closure of the stenotic as well as the normal mitral valve, but in the presence of a pressure gradient and continuous flow at the end of diastole, it seems unlikely that presystolic approximation is as complete as in the case of the normal valve. In the presence of mitral stenosis, ventricular systole begins while the dome formed by the fused leaflets of the mitral valve is still in the partially descended position. The abrupt rise in ventricular pressure forces the mitral valve upward toward the atrium. The accentuated first heart sound of mitral stenosis can be related to the unusually long excursion of the mitral valve to the closed position. An alternative explanation would be that a pressure gradient and forward flow persist to the end of diastole, and ventricular systole brings about a sudden reversal of both pressure gradient and flow, which may be responsible for the snapping character of the first sound in mitral stenosis without the valve tissue itself playing any significant role.

**Mitral Stenosis and Mitral Regurgitation**

Radiopaque material is injected into the left ventricle with the patient in the right

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**Figure 3**  
*Mitral stenosis, right anterior oblique projection. A. End systole. A small amount of contrast material is seen in the left ventricular cavity (L.V.) from the previous cardiac cycle. The plane of the mitral valve (M.V.) is clearly seen throughout the cardiac cycle and is seen as a thick, relatively radiolucent band. B. Early diastole, at the moment of mitral valve opening, which is also the moment the opening snap occurs. The initial part of the "y" descent from the "V" wave of the left atrial pressure pulse occurs between stages A and B and thus before the mitral valve has fully opened. C. End diastole, at the moment of atrial systole, which is recognized cineangiographically by reflux of contrast material into the pulmonary veins and contraction of the left atrial appendage. The flow of blood through the mitral valve is accelerated by atrial systole, and this acceleration corresponds to the presystolic accentuation of the diastolic murmur.*

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Mitral stenosis and regurgitation, right anterior oblique projection. A thickened, stenotic, and regurgitant mitral valve is depicted as a relatively radiolucent barrier separating the left ventricle and left atrium. A jet of the contrast material which was injected into the ventricle can be seen entering the left atrium.

Figure 4

oblique projection (fig. 4). The left ventricle can be seen to become opaque during dia-
stole. With the onset of systole, the mitral valve appears as a line forming the upper posterior border of the opaque ventricle. At one point, the smooth front of the mitral valve is interrupted, and at this point a stream of contrast material can be seen crossing the plane of the valve and entering the cavity of the left atrium. This jet appears as soon as left ventricular pressure exceeds left atrial pressure and continues throughout sys-
tole. This jet corresponds temporally to the pansystolic murmur of mitral regurgitation. The plane of the valve is well seen until both chambers become opacified with opaque ma-
terial. The persistence of a front of contrast material throughout systole is not seen in the presence of severe degrees of mitral regurgitation, and the persistence of this front of contrast material indicates that valve tissue is present. In the presence of severe mitral re-
gurgitation, the plane of the mitral valve is never well seen, and in such cases, contrast material is seen to flow freely back and forth across the plane of the mitral valve. The left atrium and left ventricle rapidly become opacified to the same degree following injection into either the left atrium or left ventricle. In these cases of gross mitral insufficiency, contrast material can be seen to re-
flux into the pulmonary veins with ventricular systole. This cineangiocardiographic event corresponds to the tall “V” wave of the left atrial pulse seen in this disease.

Normal Aortic Valve

The aortic valve is seen with the patient in the left anterior oblique position (fig. 5). Contrast material can be injected above or below the valve, but the details of the aortic valve leaflets are best seen following aortic injection as in figure 5. The right and left coronary leaflets are seen to move back to-
ward the aortic wall with systole (fig. 5A), but the plane of attachment of the noncoro-
nary leaflet is perpendicular to the x-ray beam, and therefore its motion is not appre-
associated. All three leaflets can be seen in diastole (fig. 5B). Note the position of the aortic leaflet of the mitral valve in the subvalvular portion of the left ventricular outflow tract.

**Congenital Aortic Stenosis**

This condition is characterized cineangiocardiographically by the presence of a mobile dome formed by the still pliable leaflets of the aortic valve that are fused at the commissures. The dome can be seen both with aortic and left ventricular injections of contrast material. Ventricular injection is depicted in figure 6. In systole (fig. 6A), a jet can be seen issuing from the apex of this pliable dome. This jet often appears to impinge on the wall of the greater curvature of the aorta, which frequently shows poststenotic dilatation. This lesion is at times responsible for severe aortic obstruction and is compatible with survival into adult life. The cineangiogram reproduced was from a 23-year-old woman with a systolic gradient of 130 mm. Hg.

Patients with congenital aortic stenosis with a mobile valve have characteristic physical findings, and it is the mobility of the valve that appears to be responsible for these signs. This condition is characterized by an ejection click and loud aortic second sound in the presence of hemodynamically severe aortic stenosis. The ejection click is often very loud, is well heard at the apex, and may be mistaken for a split first heart sound. These physical findings are often incorrectly interpreted as indicating that severe aortic stenosis cannot be present. On the contrary, they are consistent with severe stenosis, but indicate that pliable, movable valve tissue is present. This is the only form of aortic stenosis that can be treated successfully by simple aortic commissurotomy, and this important diagnosis can be made on physical examination. The mechanism of production of the ejection click of the congenitally stenotic aortic valve probably is similar to that of the opening snap of the mitral valve. Both appear to be correlated with the presence of a movable diaphragm that is tensed during a change in the velocity of a column of blood. The ejection click can be recorded coincident with the events depicted in figure 6A; the accentuated or normal second sound correlates with figure 6B.

**Calcific Aortic Stenosis**

This condition often represents the end stage in patients with congenital aortic stenosis, though other etiologic conditions may also be responsible. We have studied patients illustrating the various stages in this transi-

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**Figure 6**

*Congenital aortic stenosis, left anterior oblique projection. Contrast material injected into the left ventricle. A. Systole. The dome-shaped aortic valve (Ao.V.) is seen projecting upward into the aorta. B. Diastole. This dome collapses.*

**Figure 7**

*Calcific aortic stenosis, left anterior oblique projection. Contrast material injected below the aortic valve. The aortic valve (Ao.V.) is immobile and has the same contour in systole (A) as in diastole (B).*
tion from the mobile congenitally stenotic valve (fig. 6) to the fixed, thickened, immobile valve of calcific aortic stenosis (fig. 7). Cineangiocardiography reveals these valves to be thick with irregular surfaces, but their most important characteristic is their immobility. There is little change in the position or contour of the valve between systole (fig. 7A) and diastole (fig. 7B). A jet can be seen to issue from an opening in the center of the thick partition that separates the ventricle and aorta. On physical examination, these patients have signs of left ventricular hypertrophy and the thrill and murmurs of aortic stenosis. There is no ejection click and no second sound. The absence of these findings reflects the absence of mobility.

Aortic Regurgitation with an Apical Diastolic Murmur

Aortic regurgitation is seen radiographically as a stream of contrast material flowing retrograde through the aortic valve following the supravalvular injection of the media (fig. 8). This regurgitant stream can be seen to be deflected and indented by a stream of blood entering the left ventricle from the left atrium during diastole. Thus, during diastole, the ventricle is being filled by two streams of blood which intersect at an acute angle, and between these intersecting streams lies the aortic leaflet of the mitral valve. The motion of this leaflet can be observed as it oscillates between the two streams and deforms the roentgenopaque regurgitant stream. This observation merely confirms the impression of Austin Flint, who postulated that the murmur was produced by the "vibration of the mitral curtains."

Conclusion

Cineangiocardiography provides a method whereby cardiovascular physical signs can be correlated temporally with mechanical events within the heart.

References


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