On the Genesis of Heart Sounds

Contributions Made by Echocardiographic Studies

IN SPITE OF THE REMARKABLE ADVANCES in understanding of cardiac physiology resulting from cardiac catheterization, angiography, intracardiac phonocardiography and echocardiography, the subject of the genesis of heart sounds is more controversial than ever. With respect to the first heart sound (S1), two major schools of thought have emerged: the first espouses the classical theory which attributes the major elements of S1 to vibrations dependent on closure of the atrioventricular (A-V) valves. According to this theory, the two bursts of high frequency vibrations constituting S1 can be related to mitral and tricuspid closure and therefore referred to as M1 and T1, respectively. In recent years this theory has come under sharp attack by Luisada and co-workers. They have pointed out a distinct time lag between the crossover of pressures in the left atrium and ventricle and the appearance of the initial major group of high frequency vibrations of S1. These investigators have found a close relationship between the onset of the first group of high frequency vibrations and the early phase of rise of left ventricular pressure and the amplitude of these vibrations has been related to the left ventricular dp/dt during isometric systole. Furthermore, the role of the right ventricle in the genesis of the second burst of high frequency vibrations constituting S1 has been denied. Experiments in animals have demonstrated to the satisfaction of Luisada and co-workers that the right side is incapable of producing the sound designated T1 in classic nomenclature. The second group of high frequency vibrations of S1 is attributed therefore not to tricuspid closure but rather to an additional left sided event — the ejection of blood into the root of the aorta or the aortic root factor.

The ability of echocardiography to track the movement of all the heart valves accurately and without significant time lag has provided a new opportunity to examine the subject of the genesis of heart sounds — not only S1 but other sounds as well. The technique employed in laboratories concerned with this problem has entailed the simultaneous recording of phonocardiograms from two areas, echoes of the heart valves, an ECG and a carotid and apex pulse tracing at fast paper speed. Waider and Craigie used this method to study a group of 49 subjects with split first heart sounds. In a variety of situations including normal subjects, patients with atrial septal defect, RBBB, Ebstein's anomaly, and mitral stenosis, the simultaneous occurrence of mitral valve closure with M1 and tricuspid valve closure with T1 could be demonstrated.

Burgraf and Craigie studied a group of patients with complete heart block where an opportunity is afforded to relate the amplitude of the components of S1 to valve movements and to indirect indices of left ventricular contraction such as the pre-ejection period (PEP) over a wide range of P-R intervals. At P-R intervals less than 0.20 sec S1 was loud and followed a large amplitude of excursion of the mitral valve in its closing movement. With longer P-R intervals (0.20 — 0.50 sec) the mitral valve would close following atrial systole but would then drift open again before ventricular systole. A second brisk closing movement of the valve occasioned by ventricular contraction was again associated with a loud S1. The amplitude of S1 vibrations on the phonocardiogram was found to be closely related to the width of separation of valve leaflets at the onset of ventricular systole (r = 0.93). However, the relationship to PEP was poor (r = -0.12), indicating that the vigor of ventricular systole as measured by this indirect index had relatively less influence on the intensity of S1. In three instances it was possible to record movements of the tricuspid as well as the mitral valve. Since the movements of the two valves with various P-R intervals were not always parallel, it was possible to establish a contribution of tricuspid closure to S1 under circumstances where the mitral valve was already closed and silent.

The contribution of opening of the semilunar valves to the constellation of sounds comprising S1 was also studied by Waider and Craigie using the echo-phonocardiographic technique. An ejection sound of aortic origin was noted in cases of aortic valve deformity and this could be shown to
occur synchronously with the achievement by the valve of its fully open position, confirming the angio-phonocardiographic observations of Ross and Criley in 1964. However, in other instances where the aortic valve was presumably normal its opening could be shown echocardiographically to follow the inscription of the major components of S2 by several msec thus eliminating aortic root factor as a source of the sound.

Similarly, ejection sounds of right sided origin were found to occur precisely with the achievement of a fully open position by the pulmonic valve. This was true both with deformed valves, as in pulmonary valve stenosis, and in pulmonary hypertension. In the latter situation, where the ejection sound has in the past been attributed to a vascular origin, Waider and Craig and Sakamoto et al.7 found that it occurs at the very onset of swelling of the main pulmonary artery and not after this vessel has become dilated. This observation was made by recording simultaneously heart sounds, pulmonary valve movement by echo and movement of the chest wall over the pulmonary artery by a pulse transducer.

These studies then, support the concept that S2 consists of two major elements dependent on mitral and tricuspid closure as enunciated by Leatham in the classic theory of the origin of the first heart sound.1 The actual contact of the valve cusps is not believed to be the source of sound but rather the sudden deceleration of a mass of blood within the ventricle, which sets the whole “cardiohemic system” into vibration with resulting vibrations in the audible range. The movement of the mitral leaflets alone would be unable to account for the energy required to produce the first sound, according to experiments in dogs by MacCanon and co-workers.9

The second sound was attributed by Rouanet 140 years ago to closure of the semilunar valves.10 This explanation has been generally accepted to the present time.1 Recent echocardiographic studies by Chandraratna et al., however, have reported a discrepancy of 5 to 25 msec between aortic valve closure and A2.11 This observation would be consistent with the hypothesis that deceleration of a column of blood in the root of the aorta at the termination of systole leads to sound-producing vibrations audible as S2.6,12 Similarly by echo-phonocardiographic techniques a larger delay between pulmonary valve closure and P2 has been found by Chandraratna et al.11 and by Sakamoto et al.13 The longer interval between valve closure and sound production on the right side is attributed to the increased compliance of the pulmonary arteries and is consistent with the observations of Luisada10 and Shaver et al.14 With pulmonary hypertension the interval between valve closure and sound is abbreviated.5,14 In spite of the above, our own observations disclose an unvarying simultaneity of aortic valve closure and the initial high frequency vibrations of A2.

Opening Snaps: The opening snap (OS) of the mitral valve has been recognized for many years as being an important physical sign of mitral stenosis. It has been attributed to the opening of the thickened deformed valve in early diastole. The snap does not occur simultaneously with the pressure crossover between atrium and ventricle but later when the valve has moved to the full extent of its trajectory toward the ventricular chamber and has come to a sudden halt. In this respect the opening snap is like S1, the genesis of which is dependent on pressure crossover but actually occurs when the valve has been stopped in its closing movement some 20-40 msec later.15 By echo-phonocardiography, the relationship of the abrupt cessation of forward movement of the valve in mitral stenosis and the appearance of the OS is a most reliable and reproducible phenomenon. This explanation of the OS has been staunchly contested by Rodbard who has maintained that the sound is in fact due to reclosure of the valve.16 Combined echo-phonocardiographic studies do not support the closure theory.17 The relationship between the snap and the termination of forward (opening) movement of the valve has been observed uniformly in this condition.

The fact that opening snaps are not confined to mitral stenosis has been demonstrated by phonocardiography. Leatham described opening snaps in atrial septal defect18 and Nixon found them to be a frequent occurrence in mitral regurgitation.19 Millward et al. studied six patients with nonstenotic mitral opening snaps.20 These included tricuspid atresia, thyrotoxicosis, left to right shunt, complete heart block and mitral regurgitation. In all instances the snap occurred simultaneously with the achievement by the valve of a fully open position. The common feature of these pathophysiologic conditions was excessive flow in early diastole across a normal valve presumably resulting in a forceful opening movement and acceleration of a large inflow to the left ventricle. Similarly an opening snap of the tricuspid valve in atrial septal defect occurs coincident with full opening of the normal valve.

For those concerned with the study and teaching of physical signs in cardiology it is of interest that all the sounds discussed above — first sound, second sound, ejection sounds and opening snaps — are dependent on the respective valves reaching a fully closed or fully open position. Thus a unitarian theory can be supported for these heart sounds to the effect that their genesis depends on the deceleration of a mass of blood with resultant vibrations involving the heart chambers, valves and contents. This would be consistent with the postulates of Rushmer.8 The click of the click-murmur syndrome is probably explained on a similar basis. Criley et al. demonstrated several years ago by nonsimultaneous angiocardiographic and phonocardiographic techniques that the clicks and murmurs seemed to occur near or shortly after the time of maximal prolapse.21 Recently echo-phonocardiographic studies by Winkle et al. have amplified these observations and have shown that the peak intensity of the clicks usually coincided with the point of maximal prolapse.22 The appearance of multiple clicks and clicks in the absence of demonstrable prolapse may be explained by the complexity of the mitral valve apparatus and our inability to visualize all portions of the valve simultaneously with a single ultrasonic beam.

There are some notable exceptions to the explanations offered above for the genesis of heart sounds. The vibrations discussed above — first and second sound, ejection sounds, clicks, and opening snaps — are all of high frequency and, by auscultation, sharp and of brief duration. The lower frequency third (S3) and fourth (atrial) (S4) sounds have a different genesis. The third sound occurs in a variety of circumstances where the common denominator physiologically
is rapid ventricular filling. This is seen in the normal youthful subject and in mitral regurgitation and also in circumstances where filling in later diastole is impeded by pericardial constriction or elevated pressure as in heart failure. In echo-phonoangiographic records, the third sound occurs during the E-F or closing slope of the mitral valve in early diastole. Although the steepness of the slope may increase at the time of S₁ no distinctive role of the valve in the production of the sound has been delineated by this technique.

Similarly, the S₂ occurs after the mitral valve has reopened in late diastole in response to atrial contraction. The sound occurs during the A-C period in echocardiographic records, while the valve is in the act of closing, and no significant alteration in valve motion can be detected at the time of sound production. Although the pathogenesis of S₂ remains incompletely understood its significance in terms of disturbed physiology is as an indicator of an alteration in ventricular compliance. It occurs when atrial systole leads to a disproportionate rise in pressure in the ventricle in late diastole. It remains therefore a bedside observation of significance and not an obsolete part of the physical examination.

In summary, the investigations cited above and many more indicate a new opportunity afforded by noninvasive techniques to investigate some very fundamental aspects of the physical examination which have been of interest to clinicians for decades. The results of these studies serve to increase one's confidence in bedside observations by providing a heightened awareness of the physiologic basis of physical signs.

References

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