

'Fixed' splitting of the second heart sound in ventricular septal defect

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Eleven cases of catheter proven ventricular septal defect with left-to-right shunting in the absence of pulmonary hypertension were studied by phonocardiography. All showed wide and relatively fixed splitting of the second heart sound. Reasons for the relative fixation of the split are suggested, and the point is made that such fixed splitting is not necessarily a sign of atrial septal defect.

The wide and fixed splitting of the two components of the second heart sound in patients with atrial septal defect is well established and is regarded as an important clue to the diagnosis of this condition (Aygen and Braunwald, 1962; Dexter, 1956). In one study of 115 patients with atrial septal defect, 87 showed no respiratory variation of these two components while in 28 there was an average increase of only 0.01 sec an inspiration (Johnston, 1957). The second heart sound in patients with ventricular septal defects, on the other hand, though recognized as being widely split, is generally thought to show normal movement with respiration. Leatham investigated 23 patients with ventricular septal defects of varying size, and found that in 17 of these subjects the second heart sound was widely split. On inspiration the width of the splitting increased 'normally' - that is, the split was wide but not fixed. However, the average amount of increase in splitting on inspiration in these patients was only 0.02 sec (from an average of 0.045 sec in expiration to an average of 0.065 in inspiration) (Leatham and Gray, 1956). This variation in patients with ventricular septal defect may therefore be less than normal, that is, relatively fixed. In 115 normal patients studied by Aygen and Braunwald (1962), the average respiratory variation was 0.0378 sec. In their patients with ventricular septal defect the 'relationship between the

two components of the second heart sound was observed to fall into the normal range with only an occasional exception'. Figures are not provided.

Because it was our clinical impression that the respiratory variation of the A_2 - P_2 interval in patients with ventricular septal defect was less than normal, we undertook the following study.

Subjects and methods

Patients with ventricular septal defect were studied by phonocardiography. The criteria used for patient selection were:

- 1 Presence of a ventricular septal defect with left-to-right shunt confirmed by cardiac catheterization.
- 2 Absence of additional cardiac defects (for example, atrial septal defect, pulmonary stenosis, etc).
- 3 Absence of complete right or left bundle-branch block.
- 4 Absence of pulmonary hypertension due to raised pulmonary vascular resistance. Two patients with modestly raised pulmonary artery pressures associated with large left-to-right shunts were included.
- 5 Patient age sufficient to allow co-operation during sound recordings.

Phonocardiograms were recorded at the pulmonary valve area and at the left sternal edge during and after held expiration and inspiration and during a prolonged period of exaggerated respiration. Valsalva manoeuvre was avoided. All recordings were made on a Cambridge Multi-channel Recorder (Type 72112) using mid-frequency filters and paper speeds of 75 and 100 mm/sec. All tracings were then measured to determine the minimum and maximum splitting

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of the A_2 - P_2 interval achieved during the above respiratory manoeuvres. Results are shown in the Table.

Results

All patients studied had a respiratory variation of the A_2 - P_2 interval of 0.02 sec or less. Five patients showed a variation of 0.01 sec or less. The mean interval on expiration was 0.035 sec, increasing to a mean of 0.051 sec on inspiration. Thus, the average inspiratory increase was 0.016 sec. Though the number of patients studied is not large enough to draw definite conclusions, no relation between shunt size and either the degree of splitting or the respiratory variation of the split was apparent.

Discussion

Our figures and those of others (Johnston, 1957; Leatham and Gray, 1956) indicate that in many patients with ventricular septal defect the normal respiratory variation in the A_2 - P_2 interval may be blunted. In those patients in whom the change was 0.01 sec or less, the situation might justifiably be described as fixed splitting of S_2 since this slight degree of variation was observed in 28 of the 115 patients with atrial septal defect studied by Aygen and Braunwald.

It is generally accepted that for two auscultatory sounds to be distinguished as separate, the minimal interval between them must be at least 0.02 sec (Leatham and Segal, 1962). It is, therefore, difficult to understand the reason for the wide clinical acceptance of the concept that the respiratory variation of the A_2 - P_2 interval in ventricular septal defect is normal when, as our small series suggests, the change in the interval is so often below the limits required even for the discrimination of two discrete consecutive sounds. The explanation for this circumstance may be found in an analysis of the behaviour of the murmur. In expiration, there is an increase in return of pulmonary flow to the left heart and, consequently, an increase in shunt flow and, therefore, in the intensity of the associated pansystolic murmur which spills through A_2 . This increase in enveloping noise tends to mask A_2 , leaving P_2 to be perceived as a single or narrowly split second sound. In inspiration, with increased pooling of blood in the lungs, there is a decrease in return to the left heart, a diminution in shunt flow, and the intensity of the murmur is accordingly reduced. This results in a more sharply defined A_2 which can easily be distinguished from the late appearing P_2 (see Fig.). The outcome of these alterations is the impression of an A_2 - P_2

TABLE Respiratory variation of A_2 - P_2 interval in ventricular septal defect

Case No.	Q_p/Q_s (l./min)	PA (mmHg)	Minimum split expiration (sec)	Maximum split inspiration (sec)	Variation (sec)
1	1.8/1	20	0.030	0.050	0.020
2	4/1	55	0.025	0.035	0.010
3	3.5/1	46	0.030	0.050	0.020
4	1.6/1	18	0.050	0.060	0.010
5	4.8/1	30	0.030	0.050	0.020
6	Shunt demonstrable on ascorbates only	25	0.020	0.030	0.010
7	4.2/1	25	0.030	0.050	0.020
8	1.4/1	22	0.035	0.055	0.020
9	1.5/1	20	0.050	0.020	0.020
10	3.2/1	25	0.050	0.060	0.010
11	3.1/1	18	0.060	0.060	0.000
Mean	—	—	0.035	0.051	0.016

Abbreviations: Q_p - pulmonary flow; Q_s - systemic flow; PA - mean pulmonary artery pressure.

interval which, though widely split, moves normally with respiration.

The explanation which is widely accepted for the fixity of the split second sound in patients with atrial septal defect was first proposed by Aygen and Braunwald (1962). These authors suggested that in atrial septal defect the communicating atria act as a common reservoir receiving blood from both the systemic and pulmonary veins. The relative compliances of the right and left ventricles determine the relative amounts of this common input apportioned to the greater and lesser circulations (Levin *et al.*, 1967). Since compliance presumably does not change acutely, the amount of blood delivered to each side remains relatively constant throughout the respiratory cycle. The composition of the blood, however, does vary with the cycle, the right ventricle receiving relatively more of the systemic return during inspiration and relatively more of the pulmonary return via the shunt during expiration. Thus, despite qualitative changes, the ratio of RV filling/LV filling and hence of RV output/LV output undergoes little of the change seen in the normal situation where the cardiac chambers are fully separated. This results in a situation in which right ventricular output always exceeds left ventricular output by an amount which is ultimately determined by the degree that right ventricular compliance exceeds left ventricular compliance. Right ventricular events (that is, P_2) therefore are delayed more than normal as compared with the analogous left ventricular events (that is, A_2) but remain relatively free of the influence of respiration - that is, the second sound is widely split and fixed.

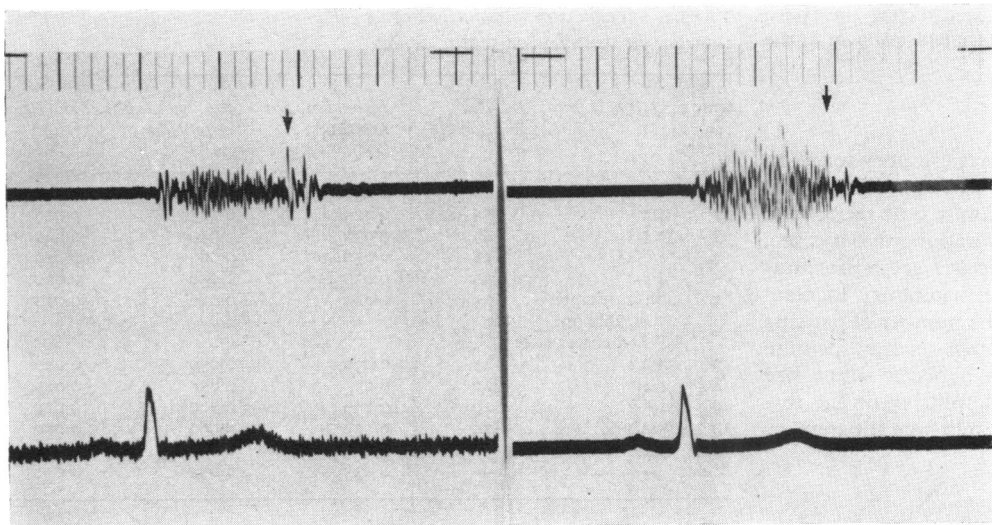


FIG. The aortic closure sound (\downarrow) so clearly audible in inspiration (left) becomes masked by the murmur in expiration (right) giving the impression that the two components of the second sound move together normally.

This sequence of events was not felt to apply to the dynamics of ventricular septal defect because the shunt was thought to be systolic in timing and therefore unable to influence right ventricular preload. More recent work (Wood, 1958) has shown that while the major part of shunt flow may take place in systole, a substantial amount may occur in diastole as well. Respiratory variation in blood passing across the interventricular septum in diastole may be sufficient to cancel respiratory variation in ventricular outputs. Thus, the increase in systemic venous return to the right ventricle in inspiration should result in reduced diastolic shunt flow for that cardiac cycle, and the reduction in systemic venous return via the normal pathways in expiration should allow an increase in the diastolic left-to-right shunt. This situation, resulting in a degree of reciprocal filling of the right ventricle via the normal and the shunt route, could produce consequences analogous to those seen in atrial septal defect – that is, presentation to the right ventricle of an excess quantity of blood which is relatively constant in amount throughout the respiratory cycle but is made up of different proportions of venous and oxygenated blood in different phases of the cycle. If the diastolic shunt through the ventricular septal defect were exactly equal in amount to the flow through normal pathways, one would expect the reciprocal relation described above to be an exact one, producing that total independence of right ventricular events from those of

respiratory cycle (and hence unvarying fixation of the A_2 - P_2 interval) that characterizes the majority of atrial septal defects. The fact that the splitting of S_2 in our cases was not exactly fixed but varied from 0.01 to 0.02 sec with respiration suggests that diastolic shunting is not precisely matched to flow through normal pathways, being sufficiently less to damp the normal respiratory variation while not completely abolishing it. This is probably because the amount of blood flow across a small ventricular septal defect is strictly limited by the size of the hole. The degree of fixity of the splitting of S_2 in ventricular septal defect may be a measure of the amount of the shunt taking place in diastole – that is, the more shunting in diastole, the more fixed the A_2 - P_2 interval. These hypotheses should be amenable to testing by fiberoptic catheter studies of shunting during the respiratory cycle but we have been unable as yet to carry these out. In any event, our cases should serve as a reminder that a relative degree of fixation of a widely split second sound is not necessarily a guarantee of the presence of an atrial septal defect.

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