Physiologic Splitting of the Second Heart Sound

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An increase in the magnitude of splitting of the 2 components of the second heart sound, as heard in the pulmonary area during inspiration, has been recognized for nearly a century. Recent reports suggest that this phenomenon is due to a relative delay of pulmonary valve closure when the increased venous return during inspiration causes prolongation of right ventricular mechanical systole. However, it seems reasonable from physiologic studies that inspiratory shortening of left ventricular systole, resulting in an earlier aortic closure sound, could contribute to the splitting. Indeed, figure Q of a review by Leatham suggests some contribution from movement of the aortic component.

In order to test this hypothesis heart sound recordings were obtained from young normal subjects with easily perceptible second heart splitting. For purposes of measurement the first sound and second sound components were required to have early fast deflections which were reasonably constant in form throughout respiration. Twenty persons were found who fulfilled these criteria. Precordial recordings were simultaneously obtained from the apex and an area of the base which allowed the greatest discrimination of second sound components. Signals from Altec capacitor microphones, electrocardiograms and pneumogram were recorded on a Hathaway oscillograph, (model 8-14C), at a paper speed of 100 mm. per second.

The intervals between the first heart sound and the first (aortic) component of the second sound (1-2 A) and the interval from the first sound to the second (pulmonary) component of the second sound (1-2 P) were measured with an accuracy of ± 3 msec. Changes in these intervals were calculated for each of at least 3 consecutive respiratory cycles.

The mean change of all the 1-2 A intervals during a respiratory cycle was 11.7 msec. while that of the 1-2 P was 11.4 msec.

Such changes were always opposite in sign. Thus early aortic valve closure contributes as much as delayed pulmonic valve closure to the maximal second sound splitting of inspiration. Similarly, the reason that splitting is minimal in expiration is that aortic closure is delayed and pulmonic closure occurs early. In the example shown in figure 1 the variation of the 1-2 A, 13 msec., actually exceeds that of the 1-2 P which is only 4 msec.

We believe that this method may be a fruitful one for studying abnormal relationships between the 2 ventricles, as in atrial septal defect.

Fig. 1. Respiratory variation in duration of 1-2A and 1-2P.
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