The Mechanism of Respiratory Variation in Splitting of the Second Heart Sound

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Many recent reports have stressed the importance of auscultatory analysis of the second heart sound as a part of routine cardiac evaluation. In spite of recognized changes in the second sound, which occur in a variety of disease states, there is still disagreement concerning the mechanism of normal respiratory splitting of the second sound.1, 2

Normal inspiratory splitting of the second heart sound was first described by Potain nearly a century ago.3 It is only in the last decade, however, that this has become widely recognized as a physiologic finding in both adults4 and children.5 The first component of the second sound represents aortic valve closure and is transmitted over the entire precordium. It normally represents the only portion of the second sound heard at the apex and primary aortic area.6 The second or pulmonic component of the second sound is best heard over the pulmonic area and is transmitted to a limited extent along the left sternal border, but not to the apex or primary aortic area. Thus, the optimum location for clinical and phonocardiographic study of physiologic splitting is over the pulmonic area.

That splitting of the second heart sound represents asynchronous closure of the aortic and pulmonary valves can be illustrated in several ways. Leatham7 recorded the heart sounds simultaneously at the apex and pulmonic areas and clearly showed the coincidence of aortic closure with the first component of the second sound and pulmonic closure with the second. Taking into account the delay in carotid pulse transmission, Perloff and Harvey8 demonstrated the synchronism of the first and second components of the second sound with the dicrotic notches of the carotid and pulmonary arterial pulses, respectively. Moreover, using intracardiac phonocardiographic technics, Rogers et al.9 noted that the second component disappeared on removal of the pulmonary valve.

Normal respiratory variation is characterized by increased splitting in inspiration and approximation, or even fusion, of the two components during expiration. The usual explanation attributes inspiratory splitting solely to delayed pulmonic closure resulting from the increased stroke volume and lengthened ejection time of the right ventricle in this phase of respiration.1, 7, 9, 10 This is a consequence of augmented right-sided filling secondary to the increase in negative intrathoracic pressure.


Figure 1
SPLITTING OF THE SECOND HEART SOUND

racic pressure accompanying inspiration. Leatham\(^6\) has raised the possibility that a decrease in pulmonary artery pressure in inspiration may also be a factor in the delay in pulmonary valve closure.\(^*\) He believes, however, that the inspiratory increase in right ventricular output is more important. Recent work by Boyer and Chisholm\(^2\) has indicated that expiratory lengthening of left ventricular systole also contributes to respiratory variation in second sound splitting. This view has not achieved general acceptance.

Methods and Materials

In order to study the mechanism of physiologic splitting, phonocardiograms were obtained from 80 normal children ranging in age from 5 to 15 years. Phonocardiograms were made on all patients while at rest and in the recumbent position. A Sanborn Twin-Beam apparatus with the standard dynamic microphone and a paper speed of 75 mm. per second was employed. Tracings were obtained over the pulmonic area with both the medium-sized (3.8 cm.) bell and the black diaphragm. A simultaneous electrocardiogram, lead I, was recorded for timing purposes. All tracings were made as the subject respired normally. An operator marked the tracing during each inspiratory portion of the respiratory cycle. Twelve of the 80 tracings were discarded because of a lack of clarity of one or both of the components of the second sound. The 68 others were deemed satisfactory for analysis. In all but eight of these, the tracings obtained with the diaphragm end piece were analyzed.

With the tip of the R wave of the electrocardiogram as a constant point of reference, the time intervals between the R wave and the first and second components of the second sound were measured to the nearest .005 second (fig. 2). In each subject five cardiac cycles occurring at the peak of normal inspiration and five at the peak of normal expiration were measured. An average “R-A\(_2\)" and “R-P\(_2\)" interval was then obtained during normal inspiration and expiration. The contribution of the movement of each component of the second sound to respiratory splitting was then assessed.

With a paper speed of 75 mm. per second, one can measure individual intervals confidently only to the nearest .005 second (.375 mm.). For each subject five separate “R-A\(_2\)" and “R-P\(_2\)" measurements were averaged and then expressed to the nearest .001 second. In the text and figures all intervals expressed to the thousandths of a second are mean values and should not be interpreted as indicating that individual measurements of this accuracy can be attained with this method.

Results

The results of the analysis of these data are presented in figures 3 and 4 and show that a significant contribution to inspiratory splitting is made by earlier aortic valve closure. The movement of “A\(_2\)" ranged from zero to .016 second, with an average of .007 second (S.D. = .004 second). Its movement contributed an average of 35 per cent (S.D. = 17 per cent) to the difference between the split-
Figure 3
Schematic representation of respiratory variation in splitting of the second sound showing the average measurements found in this series of normal children. Note the contribution of earlier aortic closure to inspiratory splitting.

ting interval in inspiration and expiration (fig. 5). During normal respiration we found an average expiratory splitting of .020 second (S.D. = .008 second) with a range of from .009 to .039 second. In inspiration, splitting averaged .040 second (S.D. = .009 second) with a range of from .020 to .063 second.

Discussion
Our findings are in agreement with those of Boyer and Chisholm in demonstrating a significant contribution of earlier aortic valve closure to inspiratory splitting of the second heart sound.

Lauson et al., Wiggers, and Williams and Gropper maintained that during inspiration an increase in pulmonary vascular capacity greater than that needed to accommodate the increased right heart output occurs. As a consequence, in inspiration pulmonary venous return to the left heart is decreased. This is associated with a transient fall in left-sided output (fig. 1) and an accompanying shortening of left ventricular ejection time and hence slightly earlier closure of the aortic valve. On the other hand, as expiration begins, the pulmonary vasculature recoils, thus increasing pulmonary venous return and augmenting left ventricular stroke volume (fig. 1) and ejection time.

Maximal splitting is noted at the peak of inspiration and corresponds to that phase of the respiratory cycle in which right ventricular stroke volume is greatest and left-sided output is relatively low (fig. 1).

*Circulation, Volume XXIV, August 1961

*Since this article was submitted for publication, Shafter (Am. J. Cardiol. 6: 1013, 1960) has reported on a study of the respiratory variation in splitting of the second sound. He suggested another explanation for the expiratory increase in left ventricular stroke volume, noting that there was some evidence that expiratory decrease in pulmonary vascular capacity did not occur. He pointed out that the augmented inspiratory right heart output requires a few seconds to reach the left heart. Thus, the apparent expiratory prolongation of left ventricular systole may be merely coincidental, reflecting instead the delayed effect of the augmented right heart output of the preceding inspiratory cycle.
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It is not unexpected that the aortic component of the second sound shows less movement than the pulmonic. Shuler et al. and Dupee and Johnson suggested that a threefold inspiratory increase in right ventricular stroke volume may occur but that expiratory augmentation of left-sided output does not exceed 50 per cent. These large fluctuations, however, were noted under artificial experimental conditions. We believe that the data of Lauson et al., showing a 25 per cent insipimental condition. We believe that the data of Lauson et al., showing a 25 per cent inspiratory increase in right ventricular stroke volume and a 7 per cent expiratory increase in left ventricular stroke volume, more closely reflect the situation in the human subject. The greater respiratory variation in right-sided volumes probably reflects the inherent greater distensibility of the right ventricle. In addition, the large capillary volume of the lungs represents an effective buffering reservoir that tends to prevent sudden extreme changes in the pulmonary venous return.

The distinction of pathologic from physiologic splitting of the second sound is not always simple. The auditory threshold for the clinical recognition of splitting approximates .02 second. Thus, in many normal subjects the second sound is appreciated as split, although usually variably so, throughout the respiratory cycle. Moreover, a recent report has shown that some individuals exhibit a widely and nearly constantly split second sound while in the supine position. When upright, however, these patients demonstrate the expected respiratory variation in splitting. These observations suggest that further phonocardiographic studies of a normal population with respect to positional changes in splitting of the second sound would be of value.

Analysis of the second sound is of importance in the evaluation of patients with a variety of congenital and acquired cardiac diseases. Changes include abnormally wide splitting on a "mechanical" basis (e.g., atrial septal defect, pulmonic stenosis, ventricular septal defect, mitral regurgitation) or on an "electrical" basis (complete right bundle-branch block). On the other hand, abnormally narrow or reversed splitting also occurs. The "mechanical" type is noted in patent ductus arteriosus and aortic stenosis whereas the "electrical" variety is present in complete left bundle-branch block.

A common clinical situation in which knowledge of normal splitting of the second sound is particularly useful is in distinguishing the patient with an atrial septal defect.

Figure 4

Histogram showing variation in splitting interval with normal respiration. Note that in 51 of 68 subjects splitting variation ranged between .015 and .025 second.

Figure 5

Scatter diagram showing contribution of movement of aortic valve closure to total variation in splitting of second sound. The slope (.35) indicates an average contribution of 35 per cent.

Circulation. Volume XXIV, August 1961
from the patient with an innocent pulmonary ejection murmur. In the vast majority of patients with a hemodynamically significant atrial defect wide and constant splitting (.04 second or more) of the second heart sound is present.

**Summary**

This study of 68 normal children has shown that in the supine position and under conditions of normal respiration splitting of the second sound averages .02 second in expiration and .04 second in inspiration. Inspiratory increase in splitting of the second sound is the result not only of a delay in pulmonic valve closure but also of earlier aortic closure. Movement of the aortic component is responsible, on the average, for 35 per cent of the difference in splitting of the second sound in inspiration and expiration.

**Acknowledgment**

We are indebted to Mr. Robert L. Nicks, Superintendent, Methodist Home for Children, Raleigh, North Carolina, for allowing us to use children of the Home as subjects for this study. We also gratefully acknowledge the assistance of Dr. H. B. Wells, Department of Statistics, School of Public Health, University of North Carolina.

**References**

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Circulation. 1961;24:180-184
doi: 10.1161/01.CIR.24.2.180

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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