Fundamentals of clinical cardiology

Paradoxical splitting of the second heart sound: An informative clinical notation

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Potain,[1] in 1866, observed that the second heart sound (S_2) was not pure but split into two components during phasic respiration. As so frequently happens, this valuable observation lay dormant for many years until first objectively demonstrated by Katz[2] and later popularized and emphasized by Leatham[3,4] and others. With the development of cardiac diagnostic studies, the hemodynamic explanation of the phenomenon has been clarified. It is now well recognized that, normally, the first component of S_2 is due to closure of the aortic valve, and the second is due to closure of the pulmonary valve.

In the past several years numerous publications have appeared concerning normal and abnormal splitting of the second heart sound.[4-11] The majority has been concerned with the phenomenon of abnormal, *persistent* splitting of S_2 and has stressed the importance of this finding. We have recently become aware of how frequently abnormal, *paradoxical* splitting of the second heart sound is encountered. In this paper we will review briefly the general topic of normal and abnormal splitting of S_2, contrast abnormal persistent with paradoxical splitting, and propose a simple, logical classification which is applicable to both types. Our main purpose, however, is to outline the circumstances in which paradoxical splitting is found. As will be discussed, when paradoxical splitting is present without obvious cause, important information is obtained which can help clarify difficult diagnostic problems.

Normal "physiologic" splitting

Splitting of the second heart sound into two distinct components occurs whenever there is asynchronous closure of the aortic and pulmonary valves. Although certain facets of the explanations cited are less well established than others, the following concept in our hands has served to best explain the clinical variations encountered. Normally, during inspiration, increasing negative pressure within the thorax augments systemic venous return to the right side of the heart. Simultaneously, the capacity of the pulmonary vascular bed increases, which results in diminished pulmonary venous return to the left side of heart. This disparity of volumes imposed on the ventricular pumps during inspiration results in a prolonged period of right ventricular ejection and a (relatively) shortened period of left ventricular ejection. Thus, closure of the pulmonary valve is delayed and separated from closure of the aortic valve.

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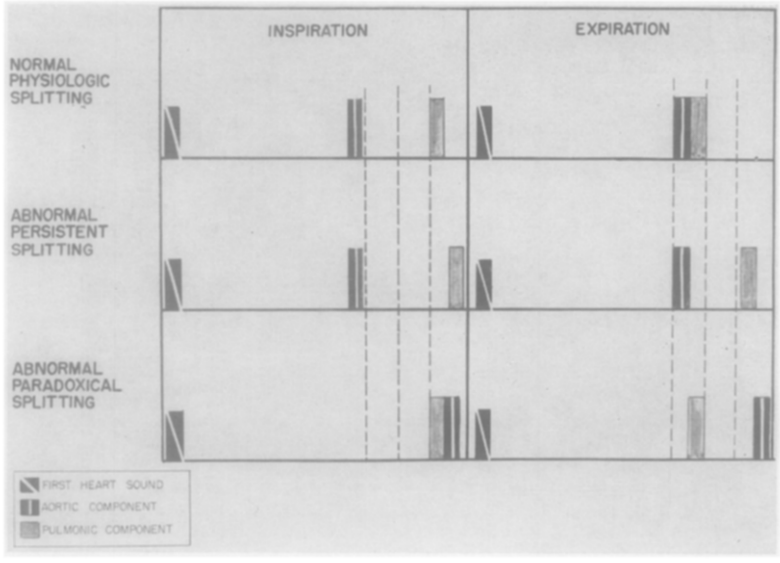


Fig. *1*. See text.

During expiration, systemic venous return decreases and pulmonary venous return increases. Therefore, the disparity of volumes is reversed, and the closures of the aortic and pulmonary valves occur at, or about, the same time. These hemodynamic events are readily recognized on auscultation in the "pulmonary area."[12] Thus, the sounds of valve closure comprising S_2 are separated maximally during inspiration and minimally or not at all during expiration, as shown in Fig. 1. (It is to be noted that the apparent "movement" of A_2 in the normal heart is exaggerated for the purpose of emphasis.)

Abnormal splitting

Variation from the normal respiratory relationships of aortic and pulmonary valve closure results in abnormal splitting of S_2. It may be generalized that any circumstance that either significantly *prolongs* or *shortens* the normal ejection period of one ventricle, without significantly influencing the other, will change the relationship of aortic and pulmonary valve closure, and abnormal splitting of S_2 will result. If the period of right ventricular ejection is prolonged, abnormal *persistent* splitting will occur; if the period of left ventricular ejection is prolonged, abnormal *paradoxical* splitting will be present. We have found that the following classification (Table I), modified from Leatham,[3] is useful for tabulating these circumstances. It is applicable to either right or left ventricular involvement and allows the clinician a format for analyzing the findings in a particular case.

It can be seen that in all situations, except diminished resistance to ventricular ejection, a prolonged period of ejection of the involved ventricle will result.

Abnormal "persistent" splitting

If the period of right ventricular ejection is *prolonged*, for any reason, or the period of left ventricular ejection is *shortened*, closure of the pulmonary valve will be relatively or absolutely "delayed" and separated from closure of the aortic valve. This separation will be present at all times, without refer-

Table I. *Abnormal splitting of* S_2

I.	"Electrical causes"
II.	"Hemodynamic causes"
	A. Volume loads
	1. Recirculation shunts
	2. Semilunar valve insufficiency
	B. Pressure loads
	1. Outlet obstruction
	2. Vascular hypertension
	C. Ventricular dysfunction
	D. Diminished resistance to ventricular ejection

ence to the phases of respiration. The degree of separation, however, may be further modified by the disparities in normal respiratory volume (Fig. 1). The most common circumstances in which such abnormal "persistent" splitting is seen are well known and have been thoroughly discussed in numerous publications. They will be reviewed here only by tabulation (Table II) in the above classification.

Abnormal "paradoxical" splitting

Utilizing the same generalization previously presented, one may readily visualize how paradoxical splitting can occur. Thus, any circumstance that sufficiently prolongs the period of left ventricular ejection will, of necessity, delay closure of the aortic valve and cause the aortic component of S_2 to "follow" the pulmonary component.[19] The sound of closure of the aortic valve will then occur temporally "later" in both phases of respiration. In such a circumstance, the normal increase in volume imposed on the right ventricle during inspiration results in a delay in closure of the pulmonary valve, and the pulmonary component blends with the aortic. During expiration, diminished systemic venous return allows earlier closure of the pulmonary valve, and the components of S_2 separate. Thus, in contrast to normal (Fig. 1), S_2 becomes single, or nearly so, with inspiration and splits into two components during expiration—reversed or paradoxical splitting.

Table II. *Abnormal "persistent" splitting of* S_2

I. "Electrical causes"
   Right bundle branch block[3,10]
II. "Hemodynamic causes"
   A. Right ventricular volume loads
      1. Recirculation shunts (left-to-right shunts)
         a. Atrial septal defect[10,11,13]
         b. Anomalous pulmonary venous drainage[32]
         c. Ventricular septal defect[14]
      2. Pulmonary valve insufficiency[15,16]
   B. Right ventricular pressure loads
      1. Outlet obstruction[3,9]
         a. Valvular
         b. Subvalvular } Pulmonary stenosis
         c. Supravalvular
      2. Pulmonary hypertension—primary or secondary (narrow splitting and accentuated pulmonic component)[33-35]
   C. Ventricular dysfunction
      1. Right ventricular failure[10,17]
   D. Diminished resistance to left ventricular ejection
      1. Mitral insufficiency[18]
      2. Ventricular septal defect[14]

The clinical conditions that give rise to paradoxical splitting of S_2 can also be conveniently tabulated (Table III) in the general classification previously presented. The majority represent situations that will prolong the period of left ventricular ejection. One exception is the circumstance of paradoxical splitting of S_2 due to a shortened period of right ventricular ejection. This could be seen in isolated tricuspid insufficiency. However, this lesion must be extremely rare. The authors have not encountered it, and it is included as a theoretical (although logical) possibility.

The general categories (Table III) will be briefly reviewed and representative phonocardiograms presented.

"Electrical causes" of paradoxical splitting. Paradoxical splitting of S_2 was recognized shortly after Wolferth and Margolies[20] demonstrated that ventricular asynchrony accompanies complete left bundle branch block. Today, this is a well-recognized clinical entity and it is the rare patient with left bundle branch block who does not exhibit this abnormality. Indeed, Gray[19] has stated: "A recognizable delay of aortic closure with paradoxical splitting of the second heart sound may be regarded as an essential clinical feature of left bundle branch block." The frequency with which paradoxical splitting is found in left bundle branch block is strong evidence that "mechanical" delay *must* accompany the "electrical" delay, and tends to refute the studies[21] which did not (experimentally) confirm ventricular asynchrony in left bundle branch block.

Paradoxical splitting of S_2 is also occasionally found in the Wolff-Parkinson-White syndrome (Fig. 2). It is most frequently present in the "Type B" form and suggests, as pointed out by March and associates,[22] that right ventricular depolarization and ejection occur prematurely in this form of Wolff-Parkinson-White

syndrome, resulting in a "relatively delayed" period of aortic valve closure. The presence of paradoxical splitting in these conduction disturbances has no real clinical importance.

"Hemodynamic causes" of paradoxical splitting.

LEFT VENTRICULAR VOLUME LOADS. The literature has emphasized the effect of right ventricular volume loads (ASD, VSD, etc.) on splitting of S_2, but minimal mention has been made of the effect of left ventricular loads. It is reasonable to assume that any condition, congenital or acquired, which significantly increases left ventricular stroke volume is likely to increase the period of left ventricular ejection and result in delayed closure of the aortic valve. The presence of paradoxical splitting may, therefore, provide a "bedside estimate" of the volume load imposed on the left ventricle. The best known example of such volume load is patent ductus arteriosus. We, like Gray,[19] have found that the presence of paradoxical splitting in patent ductus arteriosus is a reasonable clinical index that the magnitude of shunt flow is substantial. When paradoxical splitting is found in this condition, one can infer that the ductus is large and that pulmonary vascular resistance is relatively low in order to allow a significantly increased left ventricular volume load.

Aortic regurgitation is another circumstance that imposes a volume load on the left ventricle. We have found that quite frequently the aortic closure component remains audible in spite of valvular damage. Thus, the severity of aortic valvular insufficiency can also be assessed by notation of S_2 splitting. When both components of S_2 are audible, the presence of physiologic splitting indicates a milder degree of regurgitation than is present when the splitting is paradoxical (Fig. 3).

LEFT VENTRICULAR PRESSURE LOADS. It would appear that the left ventricle can cope more efficiently with pressure loads than with volume loads. Thus, significant obstruction to left ventricular outlet or increased systemic vascular resistance is frequently seen without appreciable delay in the period of left ventricular ejection.[23-25] Absence of paradoxical splitting, therefore, does not preclude a serious left ventricular pressure load. When it is present, however, in aortic stenosis (Fig. 4) or hypertension, one can state with assurance that the lesion is severe. In hypertension, paradoxical splitting has been seen only when there is significant clinical impairment in left ventricular function.

LEFT VENTRICULAR DYSFUNCTION. When paradoxical splitting is found on physical examination, the presence of typical cardiac murmurs, hypertension, or readily defined electrocardiographic abnormality makes the cause apparent. We have become aware, however, that paradoxical splitting is frequently encountered when no obvious "electrical" or "hemodynamic" cause is discernible. It is in such cases of "unexplained" paradoxical splitting that we have found this bedside observation to be most rewarding. In keeping with the previous discussion, it is evident that some factor must be present to sufficiently prolong left ventricular ejection and cause paradoxical splitting. It would seem reasonable that any circumstance that results in impaired left ventricular myocardial contractility may result in prolonged ejection. Such

Table III. *Abnormal "paradoxical" splitting of* S_2

- I. "Electrical causes"
  - 1. Left bundle branch block
  - 2. Wolff-Parkinson-White syndrome
- II. "Hemodynamic causes"
  - A. Left ventricular volume loads
    - 1. Recirculation shunt
      - a. Patent ductus arteriosus
      - b. Aorto-pulmonary window
      - c. Anomalous systemic venous return (personally observed and confirmed)
    - 2. Aortic valve insufficiency
  - B. Left ventricular pressure loads
    - 1. Outlet obstruction
      - a. Valvular
      - b. Subvalvular
      - c. Supravalvular

      } Aortic stenosis[36]
    - 2. Vascular hypertension—systemic hypertension of any cause
  - C. Left ventricular dysfunction
    - 1. Myocardial infarction
    - 2. Left ventricular failure
    - 3. Coronary insufficiency
  - D. Diminished resistance to right ventricular ejection
    - 1. Tricuspid insufficiency

lesions may be inflammatory, "ischemic," or necrotic left ventricular disease. We have elected to group such conditions as examples of "left ventricular dysfunction."

It is generally accepted, and well publicized, that right ventricular failure is accompanied by abnormal persistent splitting of S_2.[10] Less well known is the fact that left ventricular failure, from any cause, is usually accompanied by paradoxical splitting. In florid heart failure, however, the presence of pulmonary râles and rhonchi and the relatively muffled heart sounds make critical auscultation difficult. In such cases the problem is obvious, and the notation of paradoxical splitting would add no particularly useful information. In subtle left ventricular failure, however, this observation can be of great help in resolving a problem case and directing proper management.

We have found that paradoxical splitting after myocardial infarction is almost the rule, and we are surprised that it has not been previously noted.[26,27] The correlation has been so good that we now question the presence of acute myocardial damage if paradoxical splitting is absent. Conversely, the notation of paradoxical splitting in patients with suggestive chest pain, but normal electrocardiograms, has often correctly dictated appropriate treatment for acute myocardial infarction. In the usual patient with acute myocardial infarction, paradoxical splitting persists for 12 to 48 hours. It may disappear transiently during this time, but, frequently, the exertion of merely rolling over in bed will cause its return. We are presently studying the possible prognostic and therapeutic significance of this sign in acute myocardial infarction.[28]

Even more fruitful has been the observation that paradoxical splitting frequently accompanies episodes of angina pectoris. Wood[29] has also noted this phenomenon. This finding has often helped us to clarify the all too common clinical problem of "atypical chest pain." Currently, we are correlating the notation of S_2 splitting with electrocardiographic changes developed on exercise.[30] To date, the correlation of a positive exercise test and transition from physiologic to paradoxical splitting has been impressive.

The following brief case reports indicate some of the diverse clinical circumstances in which the notation of paradoxical splitting has proved to be helpful. They are included as examples of the value of this auscultatory observation in two basic areas: (1) the decision whether a left ventricular abnormality is actually present; (2) the conclusion whether an acute cardiopulmonary problem is basically cardiac or pulmonary.

Case reports

Case I. A 14-year-old boy was hospitalized on the Orthopedic Service with a diagnosis of septic arthritis and was acutely ill on admission. A painful, tender, and swollen right ankle and hip had been present for 1 day, accompanied by an impetiginous dermatitis of both lower extremities. Cultures after arthrotomy of the right hip were sterile. At this time, paradoxical splitting of S_2 was heard and the diagnosis of rheumatic myocarditis was proposed. Twenty-four hours later, his originally normal electrocardiogram showed a Wenckebach conduction defect, which promptly responded to steroids. Antistreptolysin titers during the next 6 weeks declined from 1/500 to 1/125.

Case II. A 37-year-old airman was hospitalized because of myalgia, low-grade fever, and nausea and vomiting of 3 days' duration. The early observation of a friction rub at first appeared to resolve the problem diagnostically. However, within 24 hours, paradoxical splitting of S_2 was noted by the resident physician (Fig. 5). This finding was thought to indicate the presence of active myocarditis. Despite repeated observation, it was 48 hours before cardiomegaly, abnormal electrocardiogram, and prolonged Decholin circulation time developed. Venous pressure remained normal throughout his acute illness. Subsequently, the patient manifested extreme lability of the blood pressure, tachycardia, dyspnea, and mental confusion, and a viral encephalomyocarditis was diagnosed presumptively. After 5 days of serious illness, improvement was first heralded by the return of physiologic splitting of S_2. Thus far it has not been possible to establish culturally or serologically any etiological agent.

Case III. A 42-year-old officer was hospitalized because of possible myocardial infarction. Chest pain, although suggestive in character, had been unusually brief in duration. The electrocardiograms showed only elevation of the S-T segment in the right precordial leads. One determination of SGOT was elevated, but other laboratory findings were normal, and the vital signs were completely stable. Because of the presence of paradoxical splitting of S_2, myocardial infarction was considered probable, and the patient was treated accordingly. During the ensuing 2 weeks, there were continuing nonspecific electrocardiographic changes, but it was 18 days before the electrocardiogram became clearly diagnostic of diaphragmatic myocardial infarction.

Case IV. A tense, 43-year-old soldier was seen in consultation because of a vague chest pain. On the basis of a detailed clinical history, his complaint was

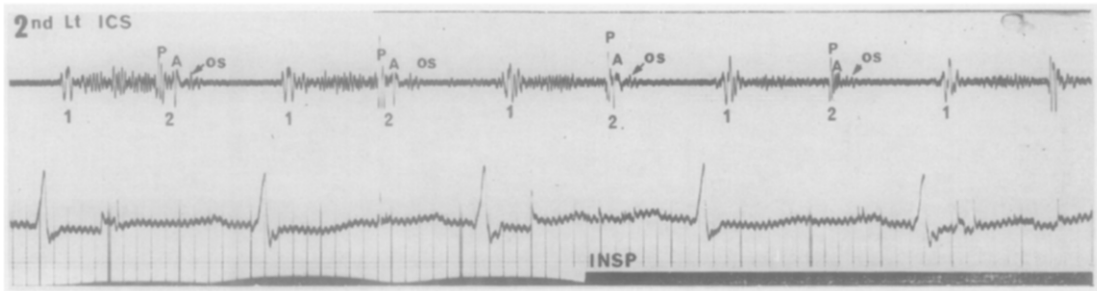


Fig. 2. Paradoxical splitting of S_2 in Wolff-Parkinson-White syndrome (Type B). (All phonocardiograms included were recorded with a Sanborn Twin-Beam Model 62 from which the upper portions of the time lines were deleted.)

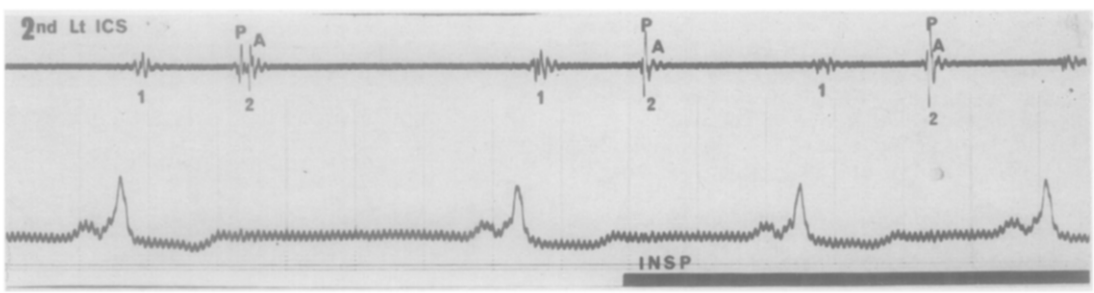


Fig. 3. Paradoxical splitting of S_2 in aortic insufficiency (post-traumatic). The phonocardiogram demonstrates the paradoxical relationship of the sounds of aortic (*A*) and pulmonary (*P*) valve closure. An aortic ejection murmur (*sm*) is heralded by an ejection click (*ec*). There is a pandiastolic murmur (*dm*) of aortic insufficiency.

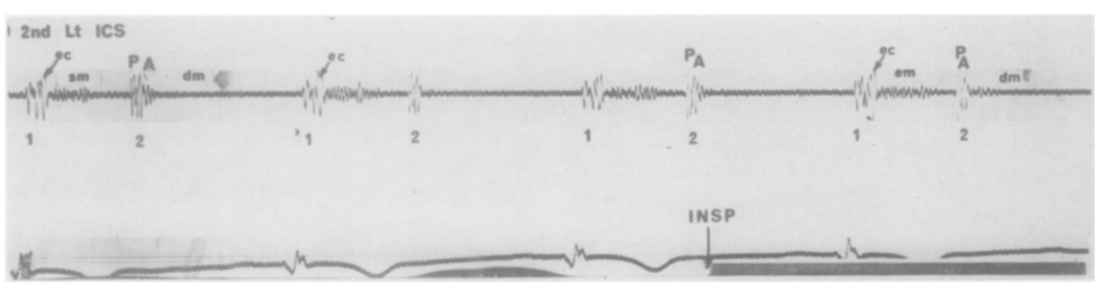


Fig. 4. Paradoxical splitting in aortic stenosis (combined aortic and mitral stenosis). The paradoxical relationship of the sounds of aortic (*A*) and pulmonary (*P*) valve closure is easily seen. A well-defined opening snap (*OS*) is also readily identified.

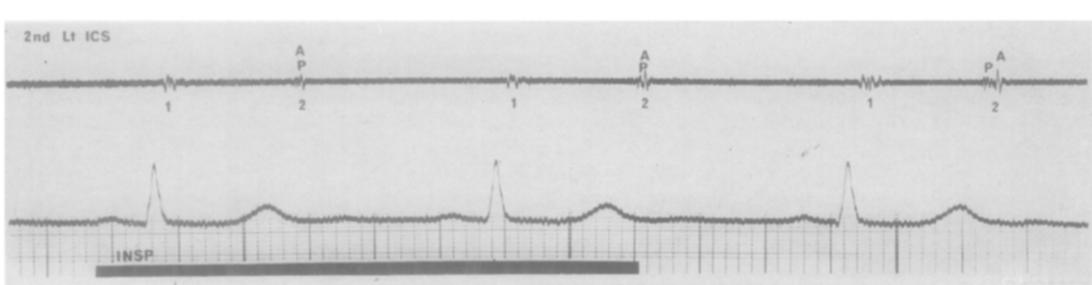


Fig. 5. Paradoxical splitting in Case II.

considered to be noncardiac. The resting electrocardiogram was normal, and a double Master two-step test showed only minimal "junctional" S-T segment depression in the left chest leads. After the exercise test the patient stated that he was "exhausted" and he appeared to be hyperventilating. Approximately 5 minutes later, it was noted that the heart tones were of good quality and the blood pressure was normal. There was, however, paradoxical splitting of S_2, which had not been present previously. In spite of this observation, because of the prominence of functional elements, cardiac clearance for continued duty was recommended. Approximately 5 months later the man was hospitalized with acute myocardial infarction characteristic in all regards.

Case V. A 27-year-old white woman with mitral stenosis, severely symptomatic, was referred for consideration of mitral valvotomy. The routine laboratory studies were normal, except for a sedimentation rate of 24 mm. Mild enlargement of the left atrium was demonstrated on chest x-ray examination, and the electrocardiogram showed only minimally notched P waves. Decholin circulation time was 16 seconds. The physical findings were characteristic of significant mitral stenosis, except for the presence of paradoxical splitting of S_2 after she had walked leisurely on the level. Because of this finding, the possibility of continuing subclinical rheumatic activity was considered.[31] Surgery was postponed, and 3 months later the second heart sound exhibited physiologic splitting for the first time. Symptomatically, the patient was markedly improved.

Case VI. A 68-year-old man had abrupt onset of dyspnea with cyanosis 5 days after herniorrhaphy. Tightness in the chest was described, but it was not severe. Venous pressure was 18 cm., and the Decholin circulation time was 26 seconds. The electrocardiogram was abnormal, with S-T segment elevation and deep inversion of the T waves in the right precordial leads. The presumptive diagnosis of pulmonary embolus was questioned because of the early presence of paradoxical splitting of S_2. Deterioration continued, and death occurred 4 hours after the onset of symptoms. A careful search at postmortem examination failed to reveal pulmonary emboli or pulmonary infarction. There was, however, massive acute myocardial infarction of the anteroseptal surface of the heart.

Discussion

The notation of splitting of S_2 has particular value in that it is a *bedside* observation which does not require special equipment, undue expenditure of time, or particular talent to appreciate. As in any type of physical examination, proficiency is acquired only through diligence and practice. Thus, in examining for splitting of the second heart sound, only "attentive auscultation" can be rewarding.

Several points deserve emphasis:

1. Splitting of S_2 may not be *best* heard in the familiar "pulmonary area." Indeed, it may be inaudible in this location but well heard several centimeters distant (upper left sternal border, mid-sternum, or even lower left sternal border). Splitting of S_2, like gold, "is where you find it."

2. Paradoxical splitting due to "left ventricular dysfunction" may not be so readily appreciated as the paradoxical splitting due to other well-known causes (such as left bundle branch block). Yet, as we have indicated, it is in this circumstance that the observation is most rewarding. Often, the examiner cannot distinguish two discrete components of S_2, but can readily tell that S_2 is "more pure" on inspiration and "less pure" on expiration.

3. Obviously, cardiac lesions may be combined, which may be "additive" in prolonging ventricular ejection (i.e., ASD plus pulmonary stenosis[3]), or one may tend to "cancel" the effect of another (LBBB plus right ventricular failure).[10] We have found that the use of a classification such as the one proposed has allowed us to analyze more thoughtfully such combinations.

Summary

In recent years, numerous publications have emphasized the information that can be gained by careful notation of splitting of the second heart sound. Most have dealt with abnormal, *persistent* splitting of S_2, and there has been infrequent mention of abnormal, *paradoxical* splitting. In this paper we have contrasted the hemodynamic bases for these differences in splitting, proposed a simple classification applicable to either type, and briefly outlined some experiences in clinical problems accompanied by paradoxical splitting. Our experience to date has indicated that notation of "unexplained" paradoxical splitting of S_2 may be a valuable "bedside indicator" of left ventricular dysfunction.

Addendum

Since this paper was submitted for publication, Yurchak and Gorlin[37] have reported their experiences with paradoxical splitting of the second heart sound in coronary heart disease. That their cases, as well as ours, were not documented by phonocardiography emphasizes the dif-

ficulty in recording what is readily appreciated on auscultation, and further directs attention to the primary value of this finding as a *bedside* auscultatory observation.

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