MEDICAL GRAND ROUNDS

Parkland Memorial Hospital

March 30, 1978

VIGNETTES IN CARDIAC AUSCULTATION

PART I

Charles B. Mullins, M.D.
William Shapiro, M.D.

"The beginning of the physician's secret: knowledge of the heart's movement and knowledge of the heart."

from Ebers Papyrus - circa 16 centuries before the Christian era.

PART I

TABLE OF CONTENTS

1. Cardiac Cycle 1

2. First Heart Sound 2

3. Second Heart Sound and Splitting of the Second Heart Sound 4

4. Third and Fourth Heart Sounds (Gallops) 8

5. Ejection Clicks 9

6. Mitral Regurgitation 10

7. Click-Murmur or Mitral Valve Prolapse Syndrome 13

8. Mitral Stenosis 18

This is the first of a two part series of clinical correlates of cardiac auscultation to be presented as a review of cardiac auscultation and to report recently developed theories which explain auscultatory events in the normal and diseased heart.

A picture is worth a thousand words; therefore, we have selected pertinent graphics which explain the auscultatory events that will be discussed and demonstrated.

A brief review of the cardiac cycle will place auscultory events into perspective.

1. Cardiac Cycle

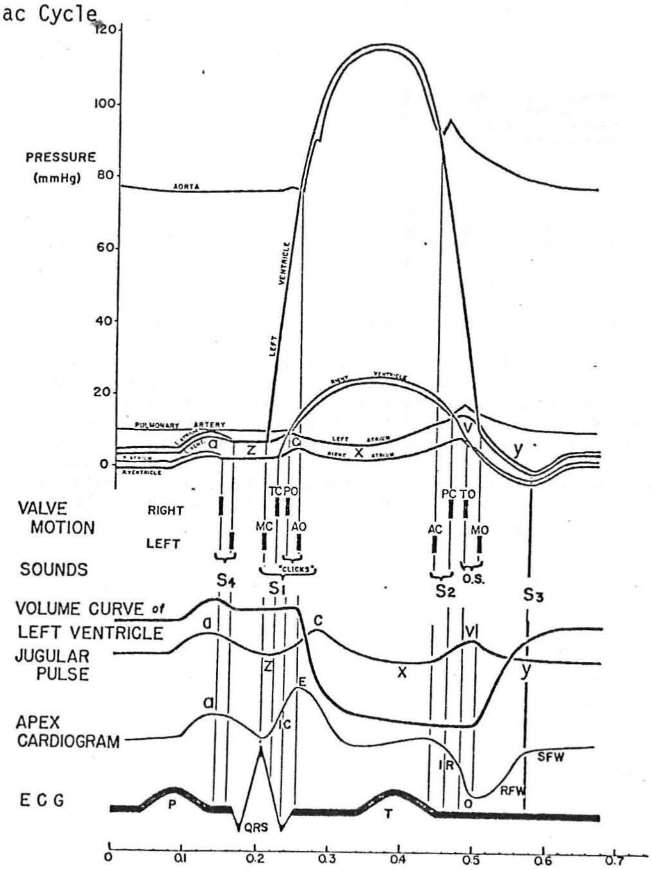
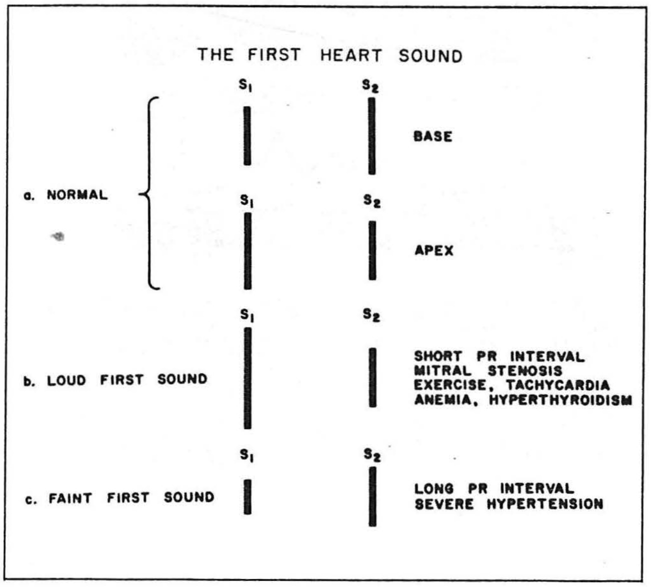


Diagram of the cardiac cycle, showing the pressure curves of the great vessels and cardiac chambers, valvular events and heart sounds, left ventricular volume curve, jugular pulse wave, apex cardiogram (Sanborn piezo crystal), and the electrocardiogram. For illustrative purposes, the time intervals between the valvular events have been modified and the Z point has been prolonged. Valve motion: *MC* and *MO*, mitral "closure" and opening; *TC* and *TO*, tricuspid "closure" and opening; *AC* and *AO*, aortic "closure" and opening; *PC* and *PO*, pulmonic "closure" and opening; *O.S.*, opening snap of atrioventricular valve(s). Apex cardiogram: *IC*, isovolumetric contraction wave; *IR*, isovolumetric relaxation wave; *O*, opening of mitral valve; *RFW*, rapid-filling wave; *SFW*, slow-filling wave. See text for details.

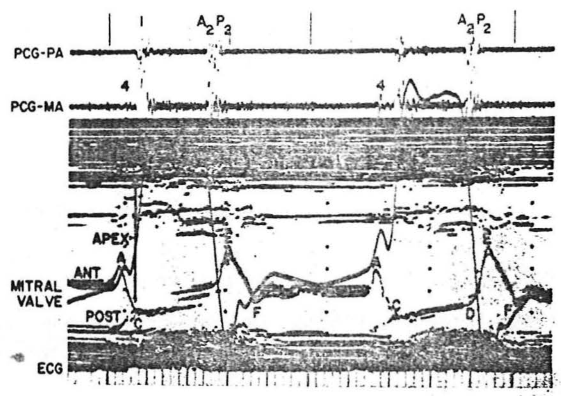
from Hurst

2. First Heart Sound



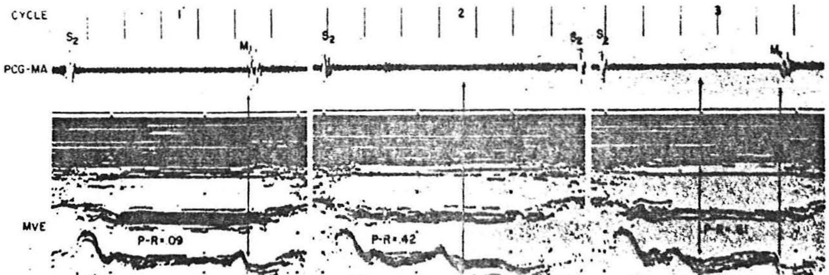
a. The second sound is characteristically louder than the first sound at the base, and although the first sound is usually more prominent than the second at the apex, this relationship is less constant and less reliable for timing purposes.
b. A loud first heart sound is heard in the presence of a short P-R interval, mitral stenosis, exercise, tachycardia, anemia, hyperthyroidism, and hyperkinetic states in general.
c. A faint first heart sound usually occurs in the presence of a long P-R interval, and it frequently occurs in severe hypertension.

from AHA



The P wave of the ECG initiating atrial systole can be seen in relationship to several events: mitral leaflets opening (A), the A wave of the apexcardiogram and the fourth heart sound (4). The mitral valve begins its closing movement in the wake of atrial systole and final closure is achieved by ventricular systole. The onset of ventricular systole is known to be precisely synchronous with the upstroke of the apexcardiogram.[62] Synchrony of the closure of the leaflets, or C point in the mitral valve echo, and the initial high-frequency of S_1 is marked by the vertical arrow. During ventricular systole the leaflets of the mitral valve remain in apposition. They separate abruptly in early diastole (D point) with the anterior leaflet achieving its fully open position at the E point. In diastole, the leaflets partially close (F) and are then reopened by the next atrial systole. PCG PA = Phonocardiogram in the pulmonic area, MA = mitral area, 1 = first heart sound, 4 = fourth heart sound, A_2P_2 = aortic and pulmonic components of second heart sound. (From Craige, E., and Fortuin, N. J.: Genesis of heart sounds and murmurs as demonstrated by echocardiography. In: *Ultrasound in the Diagnosis of Cardiovascular-Pulmonary Disease* (Joyner, C. Ed.). Chicago, Year Book Medical Publishers, 1974. Used with permission.)

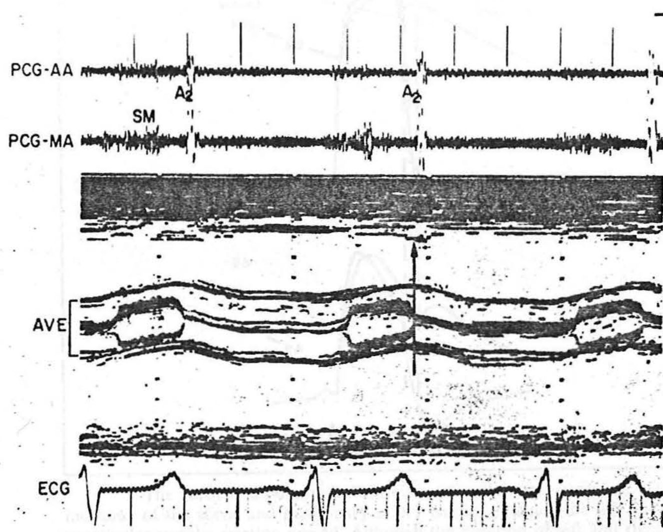
from Yu & Goodwin



Mitral valve patterns at different P-R intervals. Subject SM. Cycle 1—**short P-R with loud M_1.** Cycle 2—intermediate P-R with absent first heart sound. Cycle 3—long P-R with reopening and secondary closure of mitral valve coincident with a softer M_1. MA = Mitral area, MVE = mitral valve echogram, PA = pulmonic area, PCG = phonocardiogram, S_2 = second heart sound, M_1 as in Figure 1. Tall vertical arrows indicate time of mitral valve closure. P and QRS of electrocardiogram indicated by short arrows. (From Burggraf, G. W., and Craige, E.[2] Used with permission.)

from Yu & Goodwin

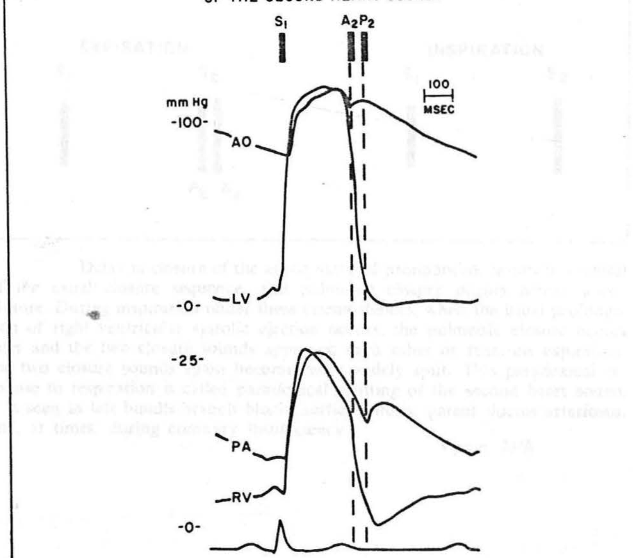
3. Second Heart Sound and Splitting of the Second Heart Sound



Echophonocardiogram from a woman in uremia. The aorta (AVE) is of normal dimensions. The aortic valve is clearly shown with its box-like separation of cusps in systole and closure at the time of A_2 (arrow). A systolic murmur (SM) is also seen.

from Yu & Goodwin

NORMAL PRESSURE-SOUND RELATIONSHIPS OF THE SECOND HEART SOUND.

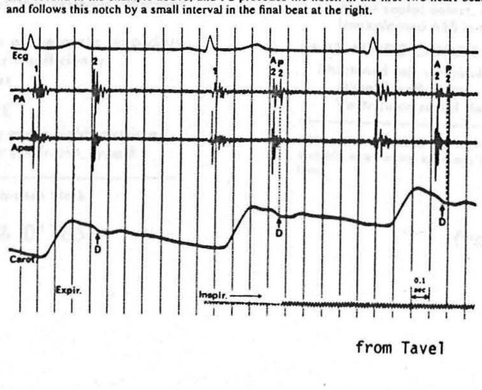


The aortic and pulmonic closure sounds which are coincident with the incisurae of the aortic and pulmonary artery trace, punctuate the left and right ventricular systolic ejection period. Although the duration of left and right ventricular electromechanical systole are nearly equal, right ventricular systolic ejection time is longer than the equivalent event occurring on the left side of the heart. As a result, the pulmonic sound occurs slightly later than aortic closure sound.

from AHA

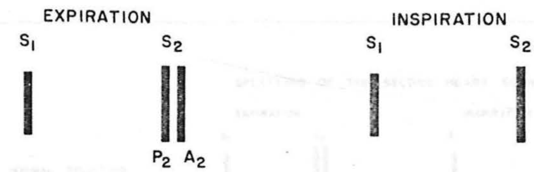
Normal respiratory variation of splitting of the second heart sound. Sounds are recorded simultaneously at the pulmonary area (*PA*) and apex, together with the ECG and carotid pulse. On expiration, A2 and P2 are superimposed (*far left*), but, with inspiration, the two heart beats on the right show increasing separation of A2 and P2.

The tracing again demonstrates the normal relationship of the second heart sound to the carotid pulse: A2 precedes the dicrotic notch by a small but constant interval, measuring 0.04 second in the example above, and P2 precedes the notch in the first two heart beats and follows this notch by a small interval in the final beat at the right.



from Tavel

PARADOXICAL SPLITTING OF THE SECOND HEART SOUND



Delay in closure of the aortic valve, if pronounced, results in reversal of the usual closure sequence, and pulmonic closure occurs before aortic closure. During inspiration under these circumstances, when the usual prolongation of right ventricular systolic ejection occurs, the pulmonic closure occurs later and the two closure sounds approach each other or fuse; on expiration, the two closure sounds again become more widely split. This paradoxical response to respiration is called paradoxical splitting of the second heart sound. It is seen in left bundle-branch block, aortic stenosis, patent ductus arteriosus, and, at times, during coronary insufficiency.

from AHA

PARADOXICAL SPLITTING OF THE SECOND HEART SOUND

DELAYED AORTIC CLOSURE

- Delayed electrical activation of the left ventricle
  - Complete LBBB (proximal type)
  - Right ventricular paced beats
  - Right ventricular ectopic beats
- Prolonged left ventricular mechanical systole
  - Complete LBBB (peripheral type)
  - Left ventricular outflow-tract obstruction
  - Hypertensive cardiovascular disease
  - Arteriosclerotic heart disease
    - Chronic ischemic heart disease
    - Angina pectoris
- Decreased impedance of the systemic vascular bed (increased hangout)
  - Post-stenotic dilatation of the aorta secondary to aortic stenosis or insufficiency
  - Patent ductus arteriosus

EARLY PULMONIC CLOSURE

- Early electrical activation of the right ventricle
  - Wolff-Parkinson-White syndrome, type B

Abbreviation: LBBB = left bundle-branch block.

from Shaver & O'Toole

WIDE PHYSIOLOGICAL SPLITTING OF THE SECOND HEART SOUND

DELAYED PULMONIC CLOSURE

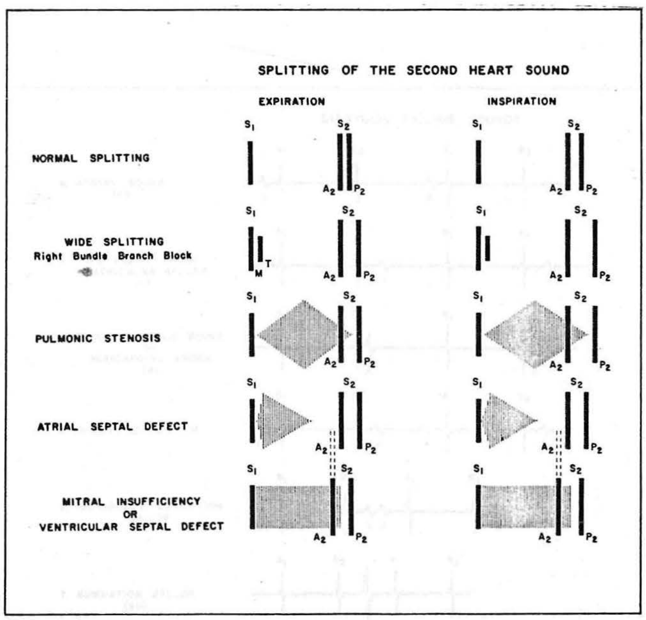
- Delayed electrical activation of the right ventricle
  - Complete RBBB (proximal type)
  - Left ventricular paced beats
  - Left ventricular ectopic beats
- Prolonged right ventricular mechanical systole
  - Pulmonic stenosis with intact septum (moderate to severe)
  - Acute massive pulmonary embolus
  - Pulmonary hypertension with right-heart failure
- Decreased impedance of the pulmonary vascular bed (increased hangout)
  - Normotensive atrial septal defect
  - Idiopathic dilatation of the pulmonary artery
  - Pulmonic stenosis (mild)
  - Atrial septal defect, postoperative (70%)
  - Unexplained AES in the normal

EARLY AORTIC CLOSURE

- Shortened left ventricular mechanical systole (LVET)
  - Mitral insufficiency
  - Ventricular septal defect

Abbreviations: RBBB = right bundle-branch block; AES = audible expiratory splitting; LVET = left ventricular ejection time.

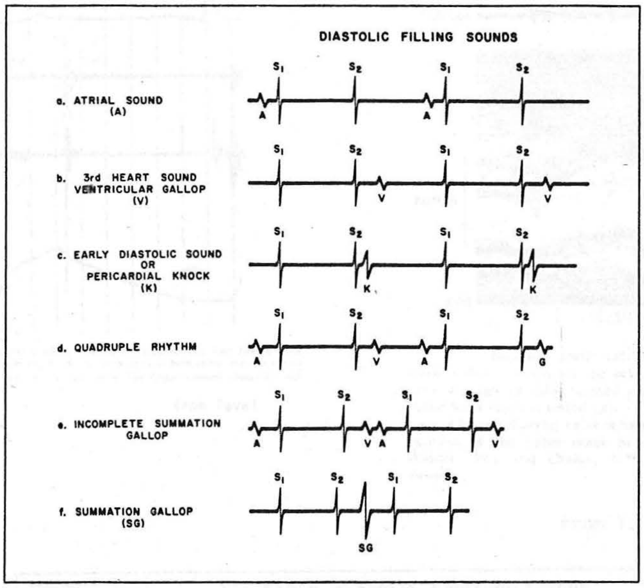
from Shaver & O'Toole



Wide splitting of the second heart sound may be due to late closure of the pulmonic valve as occurs in right bundle branch block, pulmonic stenosis and atrial septal defect; or to early closure of the aortic valve as occurs in mitral insufficiency and ventricular septal defect. In atrial septal defect, the splitting is both wide and fixed.

from AHA

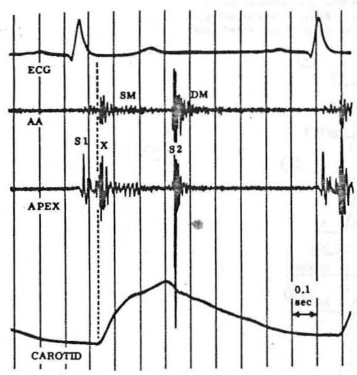
4. Third and Fourth Heart Sounds (Gallops)



a. An atrial sound (A) occurs in presystole in patients with hypertension, coronary artery disease, and long P-R intervals.
b. A filling sound may occur in early diastole in children and young adults, but it disappears with age. This sound is called a normal third heart sound. When it is heard in middle age, it is called a ventricular gallop and indicates myocardial failure or AV valve incompetence.
c. In constrictive pericarditis, a sound occurs in early diastole (K) which is earlier, louder, and higher-pitched than the usual ventricular gallop but is, in fact, an accelerated form of this filling sound.
d. If both an atrial (A) and ventricular (V) gallop are present, a quadruple rhythm results.
e. At faster heart rates these sounds occurring in rapid succession may give the illusion of a mid-diastolic rumble.
f. When the heart rate is sufficiently fast, the two rapid phases of ventricular filling reinforce each other and a very loud summation gallop (S.G.) may appear. This sound may be louder than the other two heart sounds.

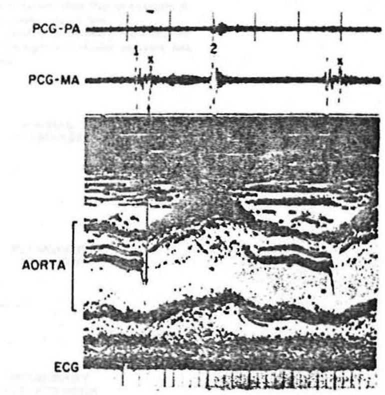
from AHA

5. Ejection Clicks (Sounds)



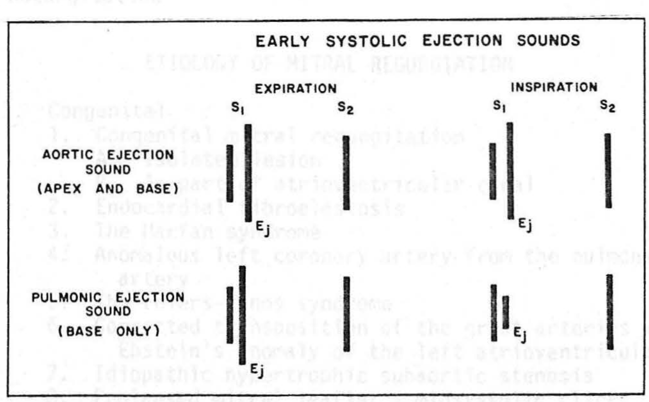
– Aortic ejection sound in aortic stenosis and insufficiency with dilated aorta in a 56-year-old male. The ejection sound (*X*) is easily seen at both aortic area (*AA*) and apex, but the first sound is not seen at the aortic area. The ejection sound coincides with the onset of the carotid upstroke.

from Tavel



Bicuspid aortic valve. Opening of the aortic valve is shown in the echocardiogram. The valve reaches its fully opened position at the time of the loud ejection sound (x). The presumed diagnosis of bicuspid aortic valve is based on the eccentric location of the valve cusps in diastole.[43] (From Waider, W., and Craige, E.[58] Used with permission.)

from Yu & Goodwin



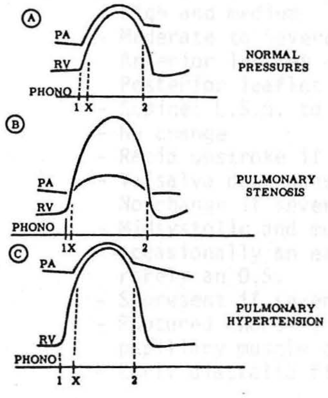
Aortic ejection sounds are heard at the apex as well as at the base and usually have little respiratory variation. Pulmonary ejection sounds, on the other hand, radiate poorly, are usually heard at the base only, and in the absence of severe pulmonary hypertension, diminish or disappear on inspiration. Aortic root ejection sounds are seen in coarctation of the aorta, aneurysm or dilatation of the aorta, hypertension and aortic insufficiency. Aortic valvular ejection sounds occur in aortic stenosis and are most easily heard when the valve is not severely calcified. Pulmonary root ejection sounds occur in idiopathic dilatation of the pulmonary artery, pulmonary hypertension, hyperthyroidism and anemia. Pulmonary valvular ejection sounds occur in pulmonary valvular stenosis, especially well heard in mild pulmonary stenosis.

from AHA

– Schematic representation of the timing of the pulmonary ejection sound (*X*) in relation to various hemodynamic alterations within the right heart. When the pulmonary artery pressure is high, the ejection sound is late and follows the QRS and the mitral first heart sound by a relatively long interval.

A, normal pulmonary and right-ventricular pressures, as might occur in uncomplicated atrial septal defect. **B**, pulmonary stenosis, with high right-ventricular pressure but low pulmonary arterial pressure, in which the ejection sound is earlier than that in example **A**. **C**, pulmonary arterial hypertension, in which the ejection sound occurs late.

These examples also help to explain the considerable delay of pulmonary valve closure in pulmonary stenosis (**B**), since P2 occurs only after the right-ventricular pressure has fallen below that of the pulmonary artery in early diastole.



from Tavel

6. Mitral Regurgitation

ETIOLOGY OF MITRAL REGURGIATION

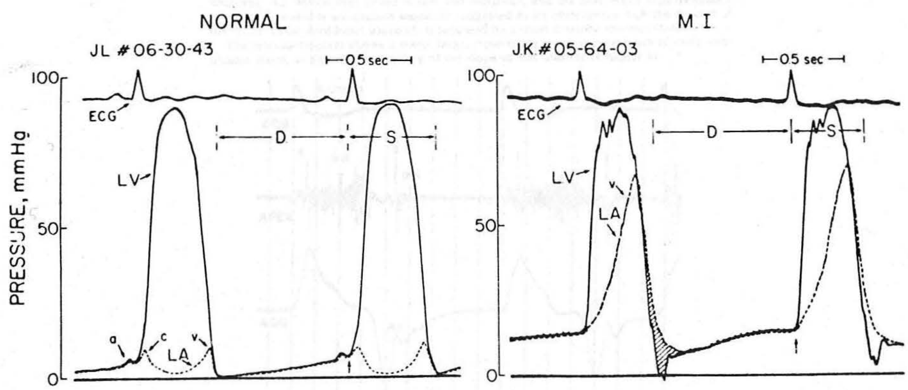
I. Congenital
   1. Congenital mitral regurgitation
      A. Isolated lesion
      B. As part of atrioventricular canal
   2. Endocardial fibroelastosis
   3. The Marfan syndrome
   4. Anomalous left coronary artery from the pulmonary artery
   5. The Ehlers-Danos syndrome
   6. Corrected transposition of the great arteries with Ebstein's anomaly of the left atrioventricular valve
   7. Idiopathic hypertrophic subaortic stenosis
   8. Prolapsed mitral leaflet - midsystolic click, late murmur syndrome - (Barlow's)

II. Acquired
   1. Rheumatic valvulitis
   2. Bacterial endocarditis
   3. Ruptured mitral chordae tendineae
   4. Papillary muscle rupture or dysfunction
   5. Calcified mitral annulus fibrosis
   6. Secondary to left ventricular dilatation

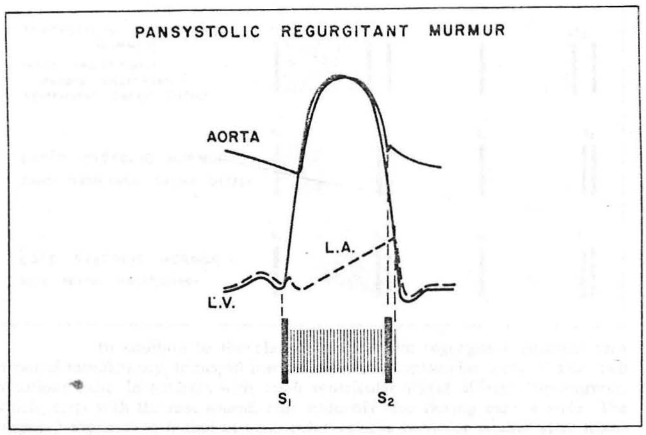
Mitral Regurgitation

1. Timing - Systolic
2. Duration - If holosystolic thru A_2; may be early, mid or late systolic
3. Intensity - 1 - 6+
4. Pitch & Quality - High and medium
5. Radiation - Moderate to Severe → axilla; Anterior leaflet → Spine; Posterior leaflet → aorta
6. Position - Supine; L.S.B. to apex
7. Respiration - No change
8. Peripheral Signs - Rapid upstroke if severe
9. Special Studies - Valsalva decreases murmur late, if minimal, No change if severe;
10. Clicks or O.S. - Midsystolic and multiple clicks; occasionally an early click; rarely an O.S.
11. S_3 and S_4 - S_3 present if severe; S_4 present if acute
12. Causes - Ruptured chordae, rheumatic, trauma, SBE, papillary muscle dysfunction
13. Other - Early diastolic flow rumble if severe

Hemodynamics of Mitral Regurgitation



NORMAL		M.I	
5	LAM	38	mmHg
10	V	70	mmHg
90/6	LV	90/15	mmHg
330	CI	115	L/min/M^2

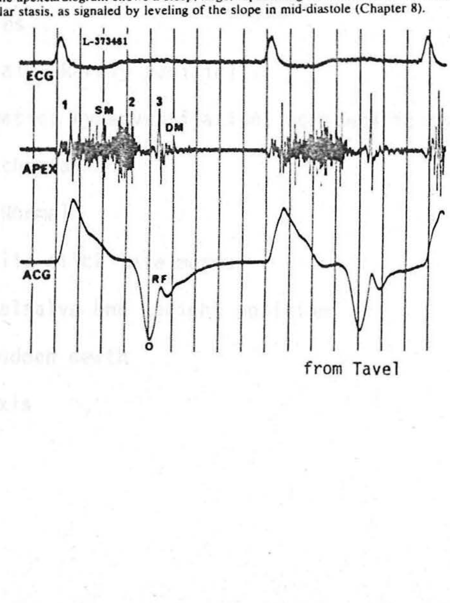


The pansystolic regurgitant murmur of mitral insufficency begins with and may replace the first heart sound. This murmur continues up to and through the aortic closure sound, since at that time ventricular pressure continues to exceed left atrial pressure.

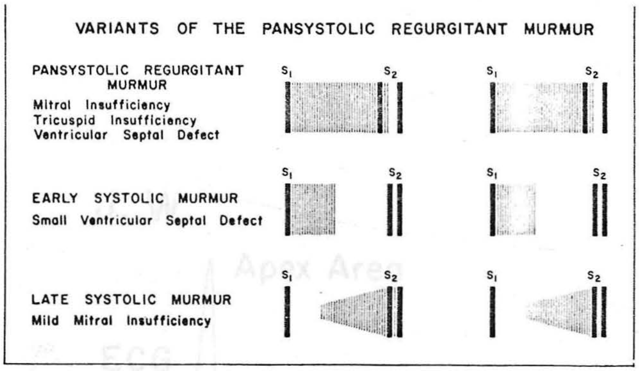
from AHA

– Pure mitral insufficiency in a 42-year-old male, showing typical sounds and apexcardiogram. A pansystolic murmur is present which continues through, and partially obscures, A2. Mitral first sound is soft and indistinct, and the first major high-frequency vibration is probably an ejection sound, as suggested by its coincidence with the *E* point of the ACG. Loud third heart sound (3) is followed by a short diastolic murmur (*DM*).

The apexcardiogram shows a steep, large, rapid-filling wave and evidence of early ventricular stasis, as signaled by leveling of the slope in mid-diastole (Chapter 8).



from Tavel



In addition to the classical pansystolic regurgitant murmur seen in mitral insufficiency, tricuspid insufficiency, and ventricular septal defect, two variations exist. In patients with small ventricular septal defects, the murmur, which starts with the first sound, may suddenly stop during early systole. The proposed explanation is that as ventricular volume becomes smaller after maximal ejection, the defect seals shut and the murmur ceases. The late systolic murmur might be considered to be the reciprocal of the former. In some cases of mild mitral insufficiency, the valve is apparently competent in early systole; but now, as ventricular volume decreases, the leaflets become incompetent and a murmur begins which builds up in late systole to become maximal at the time of the second heart sound.

from AHA

7. Click-Murmur or Mitral Valve Prolapse Syndrome:

"CLICK" LATE MURMUR SYNDROME

↑ Females

Congenital - Rarely familial

Asymptomatic; hyperventilation; nonspecific chest pain

ECG - Ischemia

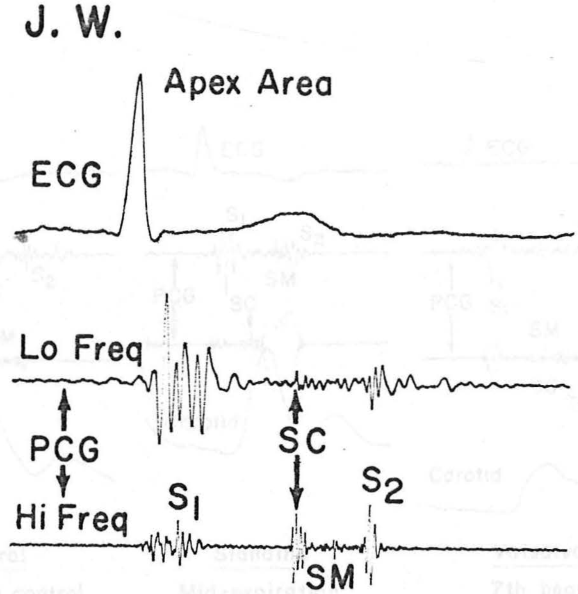
X-ray - Normal

Midsystolic click late murmur

↑ with Valsalva and upright position

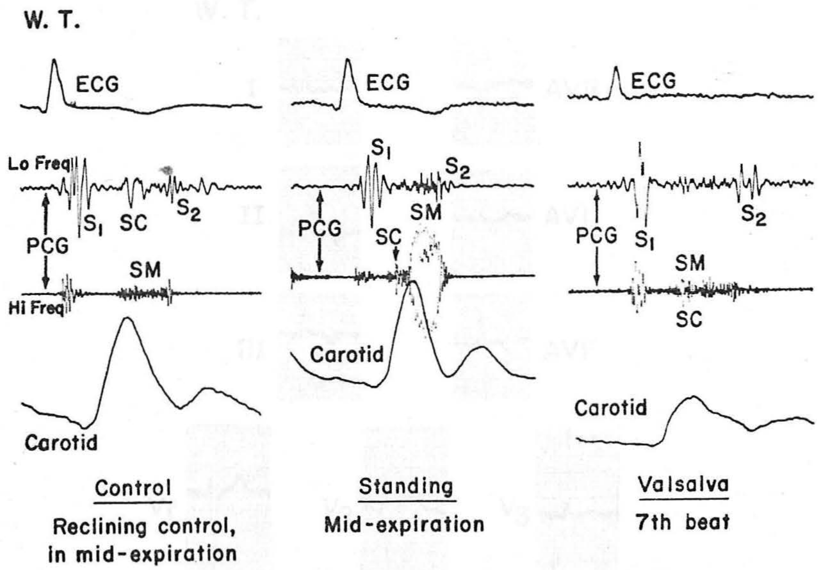
Rarely sudden death

Prophylaxis

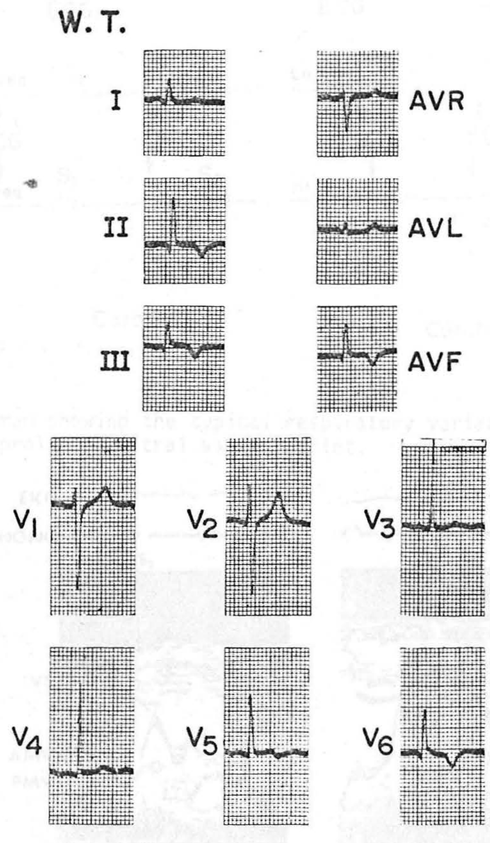


from Lobstein

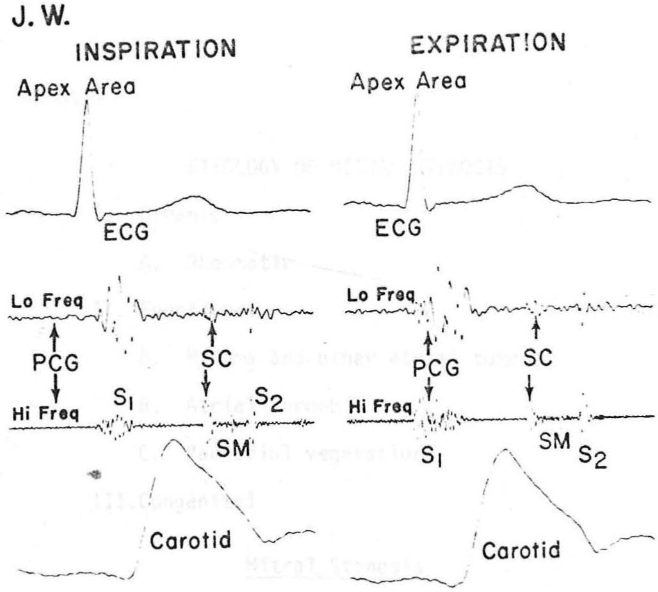
Phonocardiogram showing a typical mitral click and late systolic murmur.



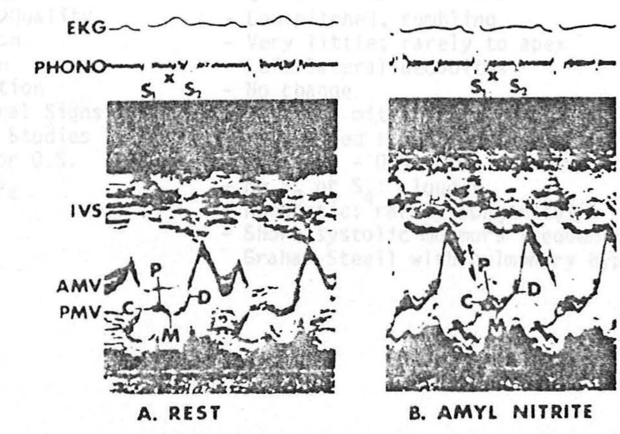
Phonocardiograms showing the effect of postural changes. On standing the systolic click (SC) moves closer to the first heart sound (S_1), and the systolic murmur (SM) increases in duration. On standing, the murmur increases markedly in amplitude and assumes a "whooping" quality. A Valsalva maneuver results in lengthening of the murmur.



ECG from a patient with click murmur syndrome showing inverted T wave in II, III, and AVF and V_6 and slight ST elevation suggesting inferior ischemia. Coronary angiograms were normal



A phonocardiogram showing the typical respiratory variation often seen in patients with prolapsed mitral valve leaflet.



A Recording from a patient with the late systolic pattern of mitral valve prolapse at rest. As in figure 1, the earliest vibrations of the auscultatory phenomena (X) occur during the P-M interval. One-second time lines have been used for illustrative purposes. Again fainter echoes may be seen synchronous with and posterior to the labeled dominant mitral valve echo. Labels as in figure 1. B: Recording from the same patient during amyl nitrite inhalation. The onset of prolapse (P) occurs earlier in systole due to a decrease in the C-P interval. Time lines and paper speed same as in A.

from Winkle, Goodman & Popp

8. Mitral Stenosis

ETIOLOGY OF MITRAL STENOSIS

I. Organic

   A. Rheumatic

II. Functional

   A. Myxoma and other atrial tumors

   B. Atrial thrombi

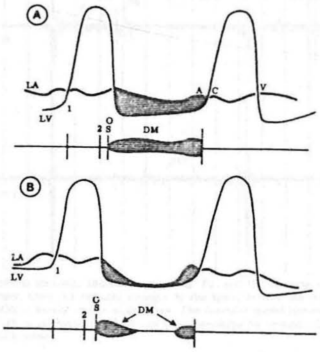
   C. Bacterial vegetations

III. Congenital

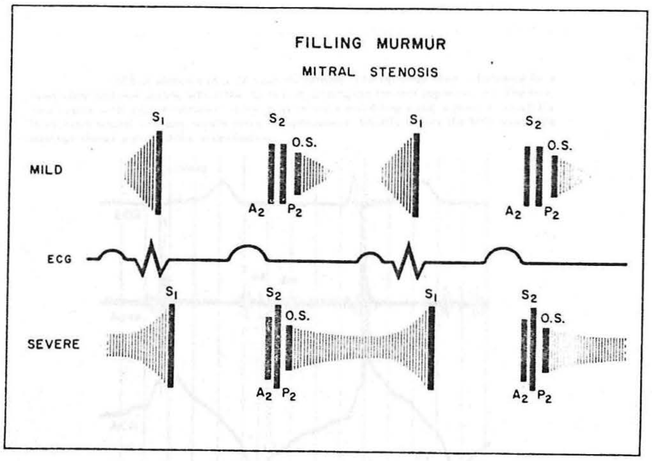
<u>Mitral Stenosis</u>

1. Timing	- Diastolic
2. Duration	- Early and late to holodiastolic, begins with O.S.
3. Intensity	- 1 - 4+
4. Pitch & Quality	- Low pitched, rumbling
5. Radiation	- Very little; rarely to apex
6. Position	- Left lateral decubitus
7. Respiration	- No change
8. Peripheral Signs	- Clubbed, mitral faces
9. Special Studies	- Leg raised ↑, Valsalva ↓
10. Clicks or O.S.	- O.S. (S_2 - O.S. time of value)
11. S_3 and S_4	- No S_3 or S_4: loud S_1
12. Causes	- Rheumatic; rarely congenital
13. Other	- Short systolic murmurs frequent; Graham-Steell with pulmonary hypertension

Schematic representation of the duration of the diastolic murmur of mitral stenosis, and its relationship to the intracardiac pressures. The diastolic murmur persists as long as there is a significant diastolic-pressure gradient across the mitral valve and is, therefore, longer in more severe grades of stenosis. A, severe stenosis. B, mild stenosis.

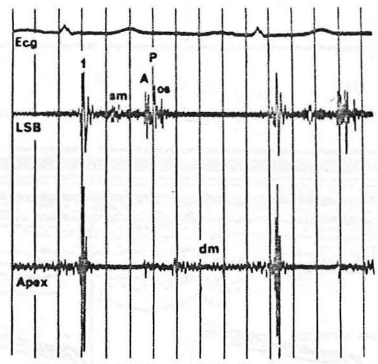


from Tavel



In mild mitral stenosis, the diastolic gradient across the valve is limited to the two phases of rapid ventricular filling in early diastole and presystole, and the rumble occurs during either or both of these periods. As the stenotic process becomes severe, a large gradient exists across the valve during all of the diastolic filling period. Therefore, all other things being equal, the longer the significant gradient, the longer the rumble and the more severe the stenosis. As left atrial pressure becomes higher, the time from the aortic closure sound to the opening snap shortens. In severe mitral stenosis, secondary pulmonary hypertension results in a louder pulmonary closure sound and the splitting becomes narrow.

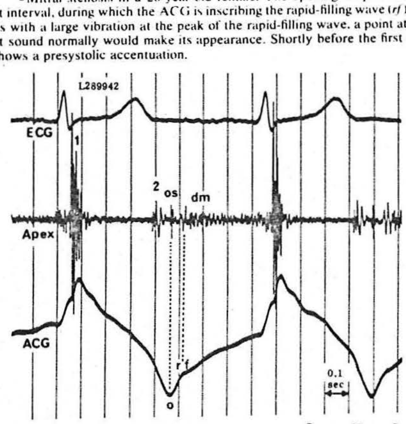
from AHA



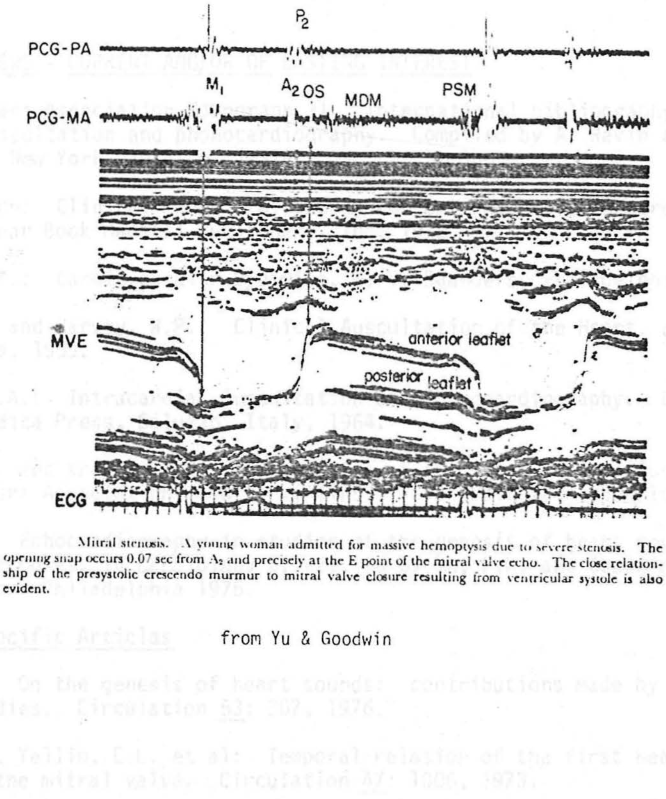
– Mitral stenosis. Identification of A2, P2, and the opening snap (*OS*) at the left sternal border. Only A2 radiates strongly to the apex, despite the rather large P2 at the LSB. The OS is barely visible at the apex. The diastolic mitral rumble begins a short time after the OS, a relationship which can be appreciated by comparing the two sound tracings with each other.

from Tavel

- Mitral stenosis in a 28-year-old female. The opening snap is followed by a brief silent interval, during which the ACG is inscribing the rapid-filling wave (*rf*). The murmur begins with a large vibration at the peak of the rapid-filling wave, a point at which the third heart sound normally would make its appearance. Shortly before the first sound, the murmur shows a presystolic accentuation.



from Tavel



Mitral stenosis. A young woman admitted for massive hemoptysis due to severe stenosis. The opening snap occurs 0.07 sec from A_2 and precisely at the E point of the mitral valve echo. The close relationship of the presystolic crescendo murmur to mitral valve closure resulting from ventricular systole is also evident.

from Yu & Goodwin

REFERENCES

A. GENERAL REVIEWS - CURRENT AND/OR OF LASTING INTEREST

1. American Heart Association Monograph 31. International bibliography of cardiovascular auscultation and phonocardiography. Compiled by A. Ravin and F.K. Frame. AHA New York, 1971. p. 1-318.

2. Tavel, Morton: Clinical Phonocardiography and External Pulse Recording. 2nd Edition. Year Book Medical Publishers, Inc. 1972.

3. Rushmer, R.F.: Cardiovascular Dynamics. W. V. Saunders, Philadelphia 1976 p.436

4. Levin. S.A. and Harvey, W.P.: Clinical Auscultation of the Heart. W. B. Saunders, Philadelphia, 1959.

5. Feruglio, G.A.: Intracardiac Auscultation and Phonocardiography. Edizioni Mineriva Medica Press, Saluzzo, Italy, 1964.

6. Leonard J.J. and Kroetz, F.W.: Examination of the Heart Part 4: Auscultation. American Heart Association, New York, 1965. (Available from your local AHA).

7. Craige, E.: Echocardiography in studies of the genesis of heart sounds and murmurs in Progress in Cardiology p.1-21, ed by Yu, P.N. and Goodwin, J.F. Lea & Febiger, Philadelphia 1975.

B. Selected Specific Articles

8. Craige, E.: On the genesis of heart sounds: contributions made by echocardiographic studies. Circulation 53: 207, 1976.

9. Laniado, S., Yellin, E.L. et al: Temporal relation of the first heart sound to closure of the mitral valve. Circulation 47: 1006, 1973.

10. MacCanon, D.M., Bruce, D.W. et al: Mass excursion parameters of first heart sound energy. J. Appl. Physiol. 27: 649, 1969.

11. Ross, R.S. and Criley, J.M.: Cineangiocardiographic studies of the origin of cardiovascular physical signs. Circulation 30:255, 1964.

12: Waider, W. and Craige, E.: First heart sound and ejection sounds. Echocardiographic and phonocardiographic correlation with valvular events. Am. J. Cardiol. 35:346, 1975.

13. Burggraf, G. W. and Craige, E.: The first heart sound in complete heart block Phono-echocardiographic correlations. Circulation 50:17, 1974.

14. Shaver, J.A., Nadolny, R.A. et al: Sound pressure correlates of the second heart sound An intracardiac sound study. Circulation 49:316, 1974.

15. Chandbabatna, P.A.N., Lopez, J.M. and Cohen, L.S.: Echocardiographic observations on the mechanism of production of the second heart sound. Circulation 51:292, 1975.

16. Shaver, J.A. and O'Toole, J.D.: The second heart sound: Newer concepts. Modern Concepts of Cardiovascular Disease 66:7-16, 1977.

17. Adolph, R.J.: Second heart sounds: The role of altered electromechanical events. AHA Monograph Number 46, p. 45, 1975.

18. Curtiss, E.I. et al: Newer concepts in physiologic splitting of the second heart sound. AHA Monograph Number 46, p. 68, 1975.

19. Shaver, J.A. Griff, F.W. and Leonard, J.J.: Ejection sounds of left-sided origin. AHA Monograph Number 46, p. 27, 1975.

20. Martin. C.E. et al: Ejection sounds of right-sided origin. AHA Monograph Number 46, p. 35, 1975.

21. Fontana, M.E. et al: Functional anatomy of mitral valve prolapse. AHA Monograph Number 46, p. 126, 1975.

22. Lobstein, H.P. Mullins, C.B. et al: Electrocardiographic abnormalities and coronary arteriograms in the mitral click-murmur syndrome. N. Eng. J. Med. 289:127-131, 1973.

23. Leatham, Aubrey: Prognosis of isolated slight mitral regurgitation with a late systolic murmur: 9 to 22-year follow-up. AHA Monograph Number 46, p. 133, 1975.

24. Winkler, R.A., Goodman, D.J. and Popp, R.L.: Simultaneous echocardiographic-phonocardiographic recordings at rest and during amyl nitrite administration in patients with mitral valve prolapse. Circulation 51:522, 1975.

25. Gibson, T.C. et al: The A wave of the apexcardiogram and left ventricular diastolic stiffness. Circulation 49:441, 1974.

26. Craige, Ernest: The fourth heart sound. AHA Monograph Number 46, p. 74, 1975.

27. Kontos, H.A. Shapiro, W. and Kemp. V.A.: Observations on the atrial sound in hypertension. Circulation 28:877-884, 1963.

28. Shah, P.M. and Jackson, D.: Third heart sound and summation gallop. AHA Monograph Number 46, p. 79, 1975.

29. Tavel, M.E.: Innocent murmurs. AHA Monograph Number 46, p. 85, 1975.

30. Craige, E.: Letters to the editor. Am Heart J. 89:128, 1975.

31. Millward, D.K. et al: Echocardiographic studies to explain opening snaps in presence of nonstenotic mitral valves. Am J. of Cardiol. 31:64, 1973.

32. Criley, J.M. et al: Mitral stenosis: mechanico-acoustical events. AHA Monograph Number 46, p. 149, 1975.

33. Paley, H.W.: Left ventricular outflow tract obstruction: Heart sounds and murmurs. AHA Monograph Number 46, p. 107, 1975.

34. Craige, Ernest: The Austin Flint murmur. AHA Monograph Number 46, p. 160, 1975.

35. Reddy, P.S. et al: Syndrome of acute aortic regurgitation. AHA Monograph Number 46, p. 166, 1975.

36. Harvey, W.P.: Innocent vs significant murmurs. Current Problems in Cardiology 1: No. 8 p. 7-50, 1976.

37. Ronan, J.A.: Effect of vasoactive drugs and maneuvers on heart murmurs. AHA Monograph Number 46, p. 183, 1975.

38. Myers, J.D.: The mechanisms and significances of continuous murmurs. AHA Monograph Number 46, p. 201, 1975.

39. Shapiro, W. et al: Intermittent disappearance of the murmur of patent ductus arteriosus. Circulation 22:226, 1960.

40. Shapiro, W.: Unusual experiences with precordial continous murmurs. Am. J. Cardiol. 7:511-516, 1961.

41. Leatham, Aubrey: Eisenmenger's syndrome. AHA Monograph Number 46, p. 193, 1975.

42. Leatham, Aubrey: The spectrum of ventricular septal defect. AHA Monograph Number 46, p. 135, 1975.