MEDICAL GRAND ROUNDS

Parkland Memorial Hospital

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VIGNETTES IN CARDIAC AUSCULTATION

PART II

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"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 II

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      3. Four cusps
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   With and without mitral regurgitation

III. Discrete Subvalvular Aortic Stenosis

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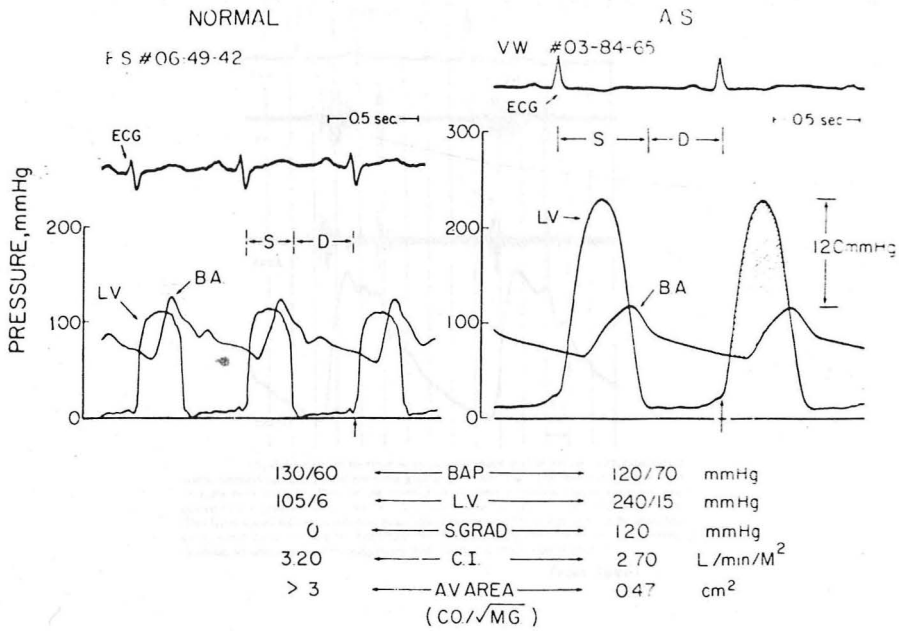
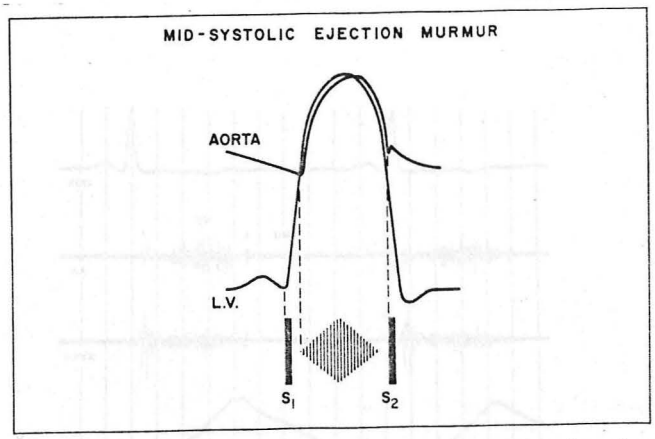
   A. Discrete
      1. Familial
      2. Nonfamilial
   B. Hypoplastic aorta (tunnel)
   C. Membrane (rare)

Aortic Stenosis

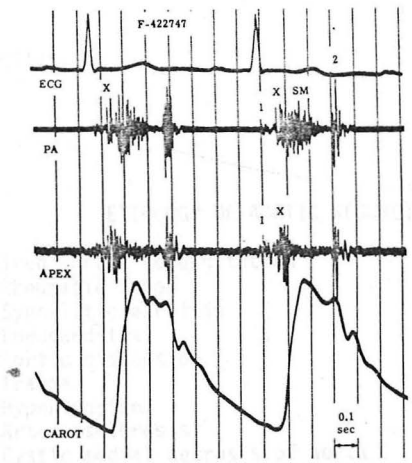
1.	Timing	- Systolic
2.	Duration	- Ejection diamond shaped ends with A_2
3.	Intensity	- 1 - 6+
4.	Pitch & Quality	- Rough, mid to high-pitched
5.	Radiation	- To cartoids,occasionally to apex
6.	Position	- Upright, leaning forward
7.	Respiration	- No change
8.	Peripheral Signs	- Slow upstroke, thrill in carotids; B.P. (½ pp)
9.	Special Studies	- Amyl nitrite ↑ murmur: Valsalva - little change
10.	Clicks or O.S.	- Ejection click
11.	S_3 and S_4	- S_4 (gradient > 60 mm Hg); single or paradoxically split S_2 with increasing severity
12.	Causes	- Biscuspid; rheumatic; valvular; "calcific"; HCVD
13.	Other	- A.I. Murmur often present





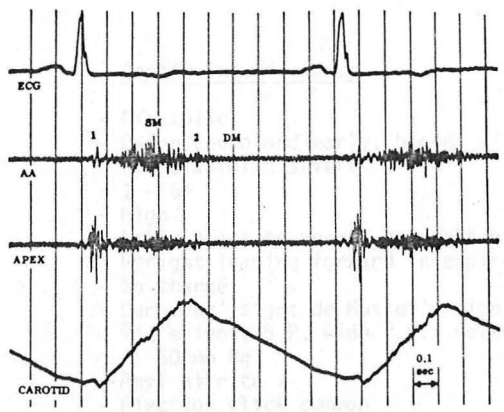
Mid-systolic ejection murmurs occur during the period of ventricular ejection. As a result, the onset of the murmur is separated from the first sound by the period of isometric contraction, and the murmur, which is crescendo-decrescendo in nature, stops before the respective semilunar valve closure.

from AHA



-Systolic ejection murmur in an asymptomatic 9-year-old boy with mild valvar aortic stenosis (aortic systolic pressure gradient, 20 mm. Hg). The murmur is short, with an early peak intensity. Despite the murmur's early timing, organic valvar disease is suggested by the presence of a prominent and late ejection sound (*X*), seen well at the apex. This latter sound follows a soft first heart sound by about 0.05 second and can be identified by its synchronization with the beginning rise of the carotid pulse. The pulse itself is steeply rising, an additional feature suggesting that the degree of stenosis is mild.

from Tavel



–Calcific aortic stenosis (severe) in a 56-year-old female. Systolic ejection murmur is present which is prolonged and has peak intensity in midsystole. The aortic second sound is soft, indicating immobility of the valve cusps. Carotid pulse shows characteristic slowing of upstroke (Chapter 7).

from Tavel

10. Aortic Regurgitation

ETIOLOGY OF AORTIC REGURGITATION

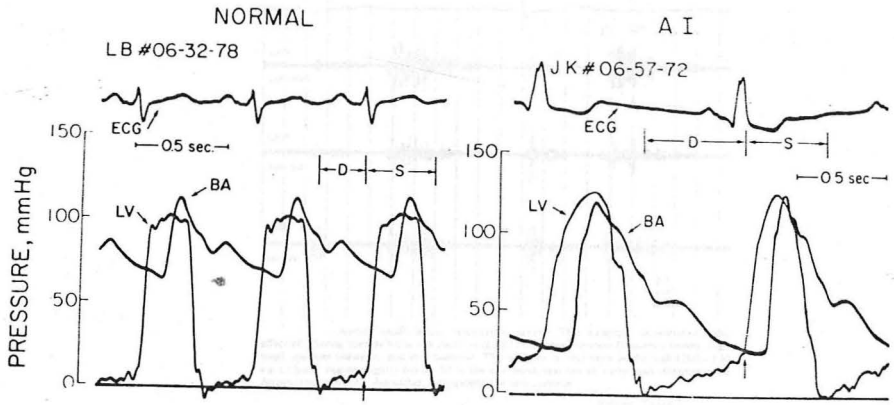
I. Acquired Aortic Regurgitation
   A. Rheumatic fever
   B. Syphilitic aortitis
   C. Endocarditis
   D. Aortic dissection
   E. Trauma
   F. Hypertension
   G. Arteriosclerosis
   H. Cystic medial necrosis of aorta

II. Congential
   A. Valvular aortic stenosis (bicuspid valve)
   B. Discrete subvalvular aortic stenosis

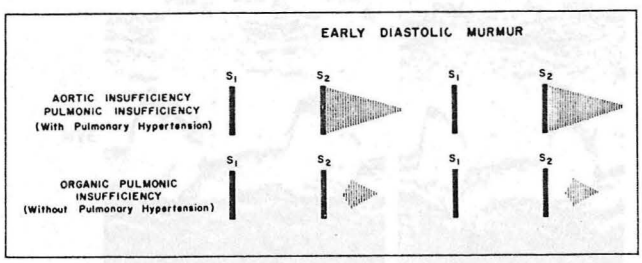
III. Associated Lesions
   A. Marfan's
   B. Rheumatoid spondylitis
   C. Rheumatoid arthritis (casual)
   D. Ruptured sinus of Valsalva
   E. Coarctation of aorta
   F. Ehlers-Danlos syndrome
   G. Reiter's syndrome and psoriasis
   H. Ventricular septal defect
   I. Osteogenesis imperfecta

Aortic Insufficiency

1.	Timing	- Diastolic
2.	Duration	- Decrescendo and early, begins with A_2 Holodiastolic severe
3.	Intensity	- 1 - 6+
4.	Pitch & Quality	- High
5.	Radiation	- Erb's point to apex - to right of sternum
6.	Position	- Upright leaning forward in expiration
7.	Respiration	- No change
8.	Peripheral Signs	- Duroziez' sign; de Musset's sign; Pulses Bisferiens; B.P. wide P.P.; severe-diastolic < 60 mm Hg
9.	Special Studies	- Amyl nitrite ↓
10.	Clicks or O.S.	- Ejection click common
11.	S_3 and S_4	- S_3 common, S_4 occasionally
12.	Causes	- Luetic; rheumatic; biscuspid valve, SBE, trauma, aortic dissection
13.	Other	- Austin Flint murmur (↓ with amyl nitrite)



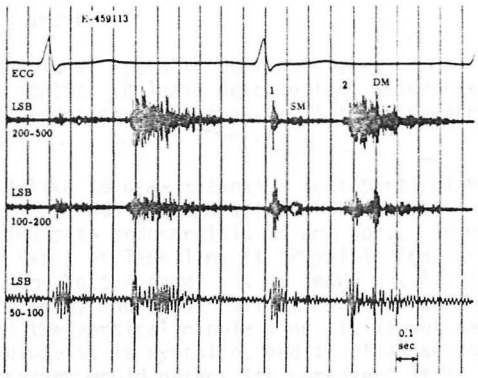
112/70	← BAP →	124/28	mmHg
42	← P.P →	96	mmHg
105/6	← L.V →	128/28	mmHg
3.20	← C.I. →	2.23	L/min/M^2



In aortic insufficiency or pulmonic insufficiency secondary to pulmonary hypertension, the murmur starts almost simultaneously with the second heart sound. Since the gradient between the aorta and the left ventricle is maximal almost instantaneously and then slowly decreases, the murmur also has a high-pitched, slow decrescendo character. insufficiency

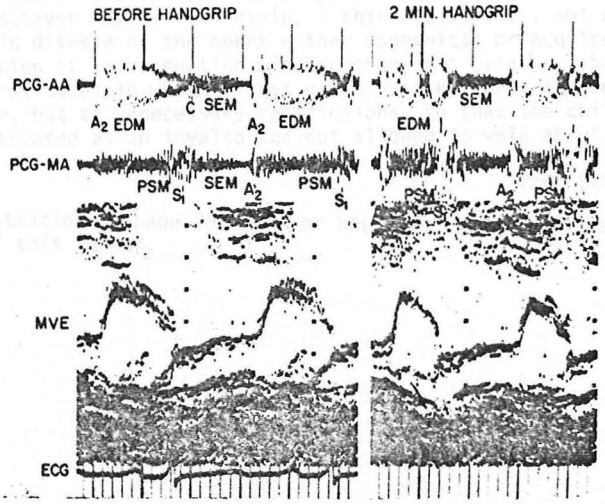
On the other hand, valvular pulmonic / without pulmonary hypertension is manifested by a murmur which starts later and has a rapid crescendo with a longer decrescendo. This murmur is lower-pitched than the usual early diastolic blowing murmurs, because the regurgitant flow is across the lower pressure system with only a small gradient.

from AHA



—Aortic insufficiency, moderately severe. This example demonstrates the effect of filtering sounds from one location (*LSB*) into three separate frequency bands, high (top), medium (middle), and low (bottom). The murmur is best seen in the high (200–500 c.p.s.) band, begins slightly before S2 in the low band, reaches an early peak intensity, and decrescendos rapidly thereafter, disappearing in late diastole.

from Tavel



Aortic regurgitation due to cystic medial degeneration. A loud musical early diastolic murmur (EDM) is seen in the aortic area (AA). At the mitral area a presystolic Austin Flint murmur (PSM) is visible. The anterior leaflet of the mitral valve (MVE) vibrates with the EDM. The valve is closing in late diastole coincident with the PSM. With handgrip (right) the Flint murmur is accentuated and again reaches its maximum intensity with the sharp closing movement of the valve.

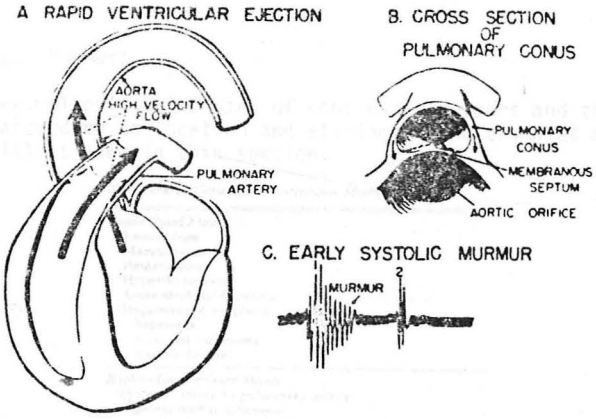
from Yu & Goodwin

11. Innocent Systolic Murmurs

An innocent or functional systolic murmur is very common in children and occassionally found in adults. A lucid description, which has held through time is the original description taken from Dr. Still's records, a murmur which bears his name -

> "And here I should like to draw attention to a particular bruit which has somewhat of a musical character, but is neither of sinister omen nor does it indicate endocarditis of any sort. In my own note-books I am in the habit of labelling it "Physiological bruit," but only for want of some better name. It is heard usually just below the level of the nipple, and about halfway between the left margin of the sternum and the vertical nipple line; it is not heard in the axilla nor behind; it is systolic, and is often so small that only a careful observer would detect it; moreover, it is sometimes very variable in audibility, being scarcely noticeable with some beats and easily heard with others; its characteristic feature is a twanging sound, very like that made by twanging a piece of tense string. This bruit is found mostly in children between the ages of two and six years; as a rule . . . the bruit is discovered only in the course of routine examination. It persists sometimes for many months; I have noted it as present in one case for two years. Whatever may be its origin, I think it is . . . not due to any organic disease of the heart either congenital or acquired; and I mention it in connection with endocarditis because I have seen several cases in which it has given rise not only to ground-less alarm, but to unnecessary restrictions, so that the child has been treated as an invalid and not allowed to walk about."
>
> from Still

With the illustration on page 29, Rushmer has given a lucid illustration and description of this murmur.



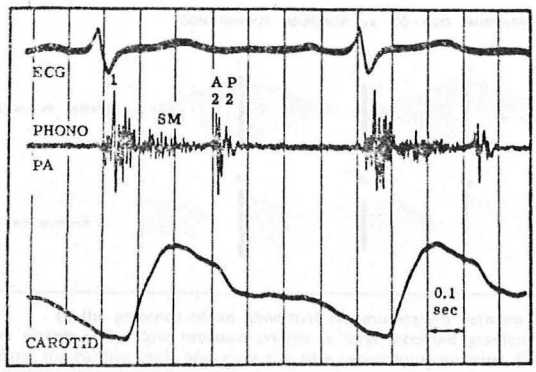
from Rushmer

Functional Systolic Murmurs

A. Under normal conditions, blood flows through the aorta and pulmonary arteries at sufficient velocity to produce turbulence during the rapid ejection phase of ventricular systole. Early systolic murmurs can be heard in many normal children at rest and in nearly any normal subject after exercise.

B. The right ventricular outflow tract has a roughly crescentric cross-sectional area, partly because the membranous portion of the interventricular septum bulges into the lumen. Bundles of myocardial fibers, encircling the conus region, tend to further diminish the cross-sectional area of this channel during systole. For these reasons turbulence is more likely to develop in the pulmonary artery than in the aorta Systolic murmurs in normal subjects usually have maximal intensity in the pulmonary area on the precordium.

C. An early systolic "functional" murmur may be regarded as an intensified fourth component of the first heart sound.



–Innocent systolic ejection murmur in a young healthy male with no heart disease. The murmur, recorded at the pulmonary area, is short, with an early peak intensity. (Courtesy of J.A.M.A. 202:119, 1967.)

from Tavel

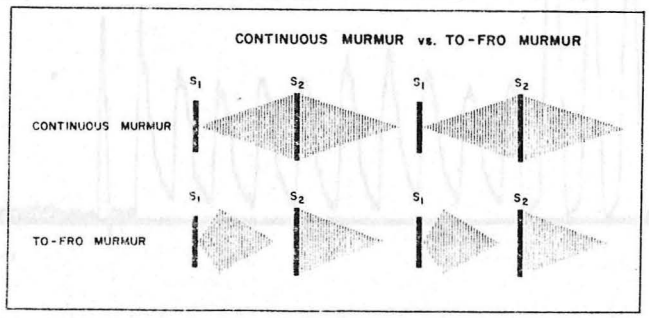
12. Continuous Murmurs

There are multpile etiologies of continuous murmurs and the same characteristics vary according to location and etiology. Two examples of continuous murmurs are illustrated in this section.

The Various Causes of Continuous Murmurs

Rapid Blood Flow
Venous hum
Mammary souffle
Hemangioma
Hyperthyroidism
Acute alcoholic hepatitis
Hyperemia of neoplasia
hepatoma
renal cell carcinoma
Paget's disease
High-to-Low Pressure Shunts
Systemic artery to pulmonary artery
patent ductus arteriosus
aorto-pulmonary window
truncus arteriosus
pulmonary atresia
anomalous left coronary artery
bronchiectasis
sequestration of lung
Systemic artery to right heart
ruptured sinus of Valsalva
coronary artery communication
Left-to-right atrial shunt
Lutembacher's syndrome
Mitral atresia + atrial septal defect
Veno-venous shunt
Anomalous pulmonary veins
Porta-systemic shunts
A-V fistula
systemic
pulmonary
Localized Arterial Obstruction
Coarctation of aorta
Pulmonary artery stenosis or obstruction
Carotid occlusion
Celiac, mesenteric occlusion
Renal occlusion
Femoral occlusion
Coronary occlusion

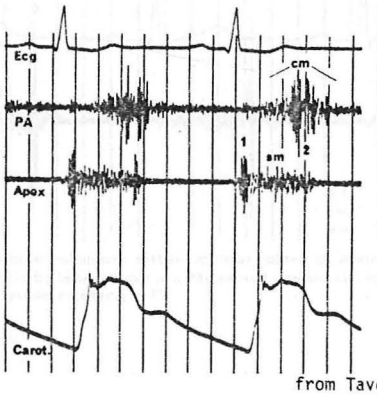
from AHA



In the presence of an abnormal communication between a high pressure system and a low pressure system, a large pressure gradient exists throughout the cardiac cycle and gives rise to a continuous murmur. The classical example of such a murmur is the murmur of patent ductus arteriosus. This type of murmur is at times confused with the to-fro murmur. The latter is a combination of an ejection murmur and a murmur of semilunar valve incompetence. The classical example of a to-fro murmur is the murmur of aortic stenosis and insufficiency. The continuous murmur builds up to a crescendo around the second heart sound, whereas the to-fro murmur can be seen to have two components. The mid-systolic ejection component decrescendos and disappears as it approaches the second heart sound.

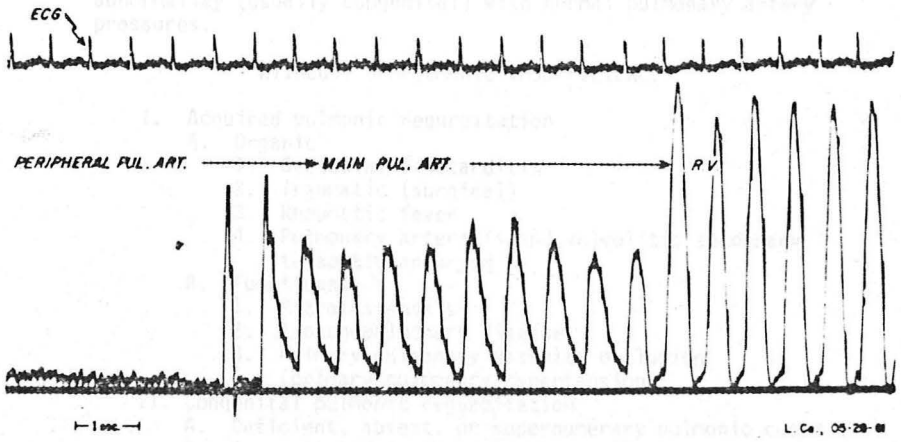
from AHA

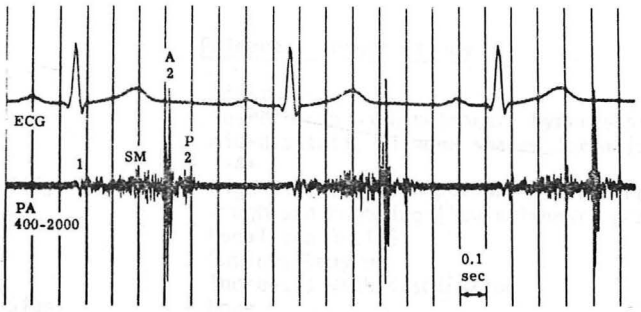
– Patent ductus arteriosus in an 18-year-old female. A continuous murmur (*cm*) is present at the pulmonary area which has its peak around the time of the second heart sound. A pansystolic murmur is also seen at the apex, possibly related to functional mitral insufficiency.



from Tavel

PERIPHERAL P.A. and PUL. VALVULAR STENOSIS





— Peripheral pulmonary systolic murmur caused by obstruction of the left main pulmonary artery by benign tumor in a 28-year-old woman. The murmur is long and crescendos in late systole, extending to P2.

from Tavel

13. Pulmonary Insufficiency

For practical purposes of auscultation two varieties of pulmonary insufficiency should be considered:

1) Pulmonary insufficiency secondary to severe pulmonary hypertension
2) Pulmonary insufficiency (organic) secondary to pulmonary valve abnormality (usually congenital) with normal pulmonary artery pressures.

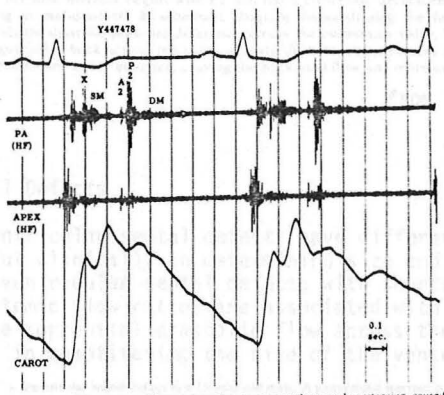
ETIOLOGY OF PULMONIC INSUFFICIENCY

I. Acquired pulmonic regurgitation
   A. Organic
      1. Bacterial endocarditis
      2. Traumatic (surgical)
      3. Rheumatic fever
      4. Pulmonary arteritis and valvulitis secondary to aortic aneurysm
   B. Functional
      1. Mitral stenosis
      2. Bronchopulmonary disease
      3. Primary pulmonary vascular occlusion (primary pulmonary hypertension)

II. Congenital pulmonic regurgitation
   A. Deficient, absent, or supernumerary pulmonic cusps
   B. Congenital dilatation of the pulmonary artery
   C. Pulmonary hypertension (severe, obstructive) in congenital heart disease

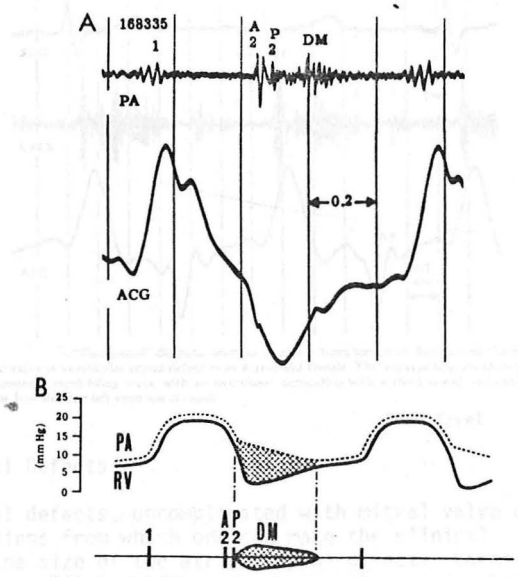
Pulmonary Insufficiency

1. Timing	- Diastolic
2. Duration	- Decrescendo with pulmonary hypertension; mid-diastolic diamond shaped (organic)
3. Intensity	- 1-4+
4. Pitch & Qualtiy	- High pitched (high pulmonary pressure); rough and rumbling (low pulmonary pressure)
5. Radiation	- Localized 3-LICS
6. Position	- Supine, legs up
7. Respiration	- Increases with inspiration
8. Peripheral Signs	- None
9. Special Studies	-Amyl nitrite may ↑ ; Valsalva may ↓
10. Clicks or O.S.	- Occasional ejection click
11. S_3 and S_4	- Right-sided S_3 and S_4 if ↑ P.A. pressure
12. Causes	- Most 2° to pulmonary hypertension; congenital
13. Other	



Pulmonary insufficiency secondary to severe pulmonary hypertension caused by atrial septal defect with increased pulmonary vascular resistance. A high-frequency decrescendo diastolic murmur is initiated by a large P2 at the pulmonary area. Note also the late ejection sound (X) at the pulmonary area and the fact that P2, which is separated widely from A2 and follows the dicrotic notch of the carotid pulse, radiates well to the apex.

from Tavel



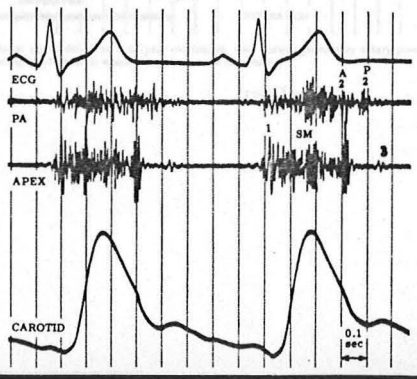
– Pulmonary insufficiency in the absence of pulmonary hypertension. **A**, tracing taken from a 22-year-old female with isolated congenital pulmonary insufficiency. A low-frequency diastolic murmur begins with P2 and has a crescendo-decrescendo configuration, stopping in mid-diastole. **B**, schematic diagram demonstrating the fact that the murmur parallels the diastolic pressure difference across the pulmonary valve. When sufficient blood regurgitates back across the valve in early diastole, the pressures in the right ventricle and pulmonary artery equalize, causing the backward flow and murmur to cease.

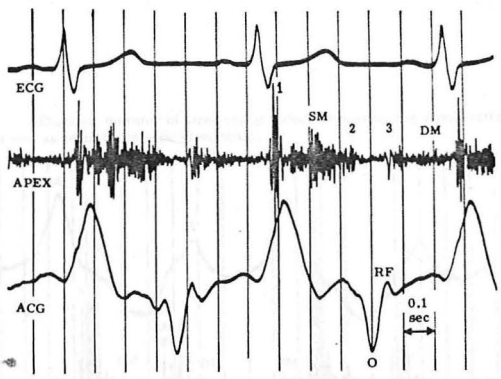
from Tavel

14. Ventricular Septal Defects

Large or small ventricular septal defects have different auscultatory findings that are useful clinically in determining size and perhaps indications for operation. Large ventricular septal defects with shunts greater than 2/1 or 2.5/1 pulmonary/systemic flow ratios are associated with a mid-diastolic rumble secondary to the torrential diastolic flow across the mitral valve. This finding is useful in quantitating the size of the ventricular septal defect.

– Ventricular septal defect in a 15-year-old male. A pansystolic murmur is seen in both tracings and obscures A2 at the pulmonary area. The murmur resembles an ejection murmur at the pulmonary area. The second sound is widely split at the pulmonary area, and a third heart sound is seen at the apex.



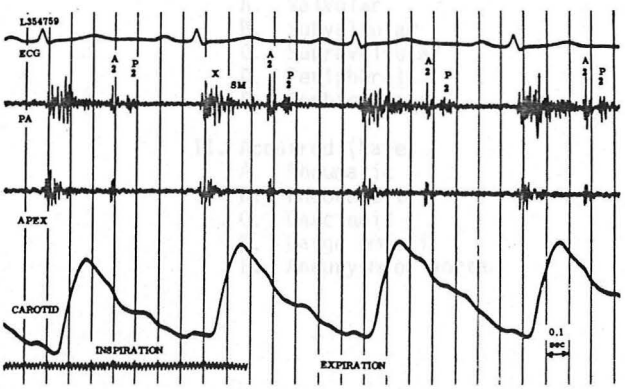


—"Functional" diastolic murmur resulting from torrential flow across the mitral valve in ventricular septal defect in an 8-year-old female. The apexcardiogram shows a prominent rapid-filling wave with an overshoot, coinciding with a third sound, indicating that flow into the left ventricle is rapid.

from Tavel

15. Atrial Septal Defects

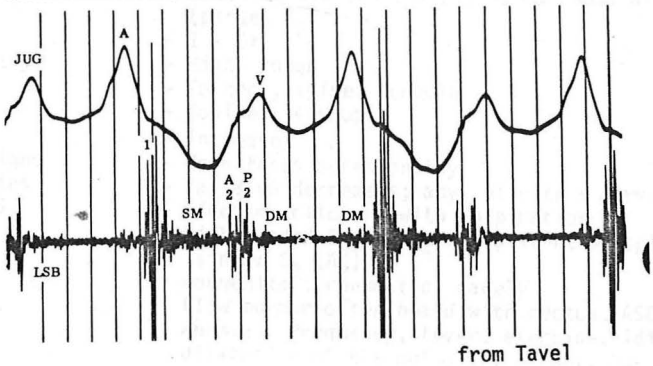
Atrial septal defects, uncomplicated with mitral valve disease, have classic auscultatory findings from which one can make the clinical diagnoses and quantitation of the size of the atrial septal defect. Large atrial septal defects greater than 2.5/1 pulmonary/systemic flow ratios have a mid-diastolic flow rumble due to the torrential flow across the tricuspid valve.



—Atrial septal defect in a 22-year-old female, with normal pulmonary artery pressure. The second sound shows wide splitting (0.07 second) which varies little with respiration.

from Tavel

. -Diastolic murmur in atrial septal defect, demonstrating atriosystolic accentuation as well as an early diastolic component.



from Tavel

16. Pulmonary Stenosis

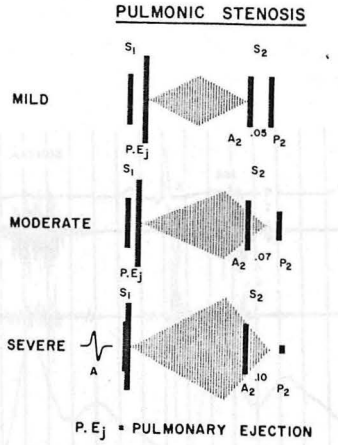
ETIOLOGY OF PULMONARY STENOSIS

I. Congenital
   A. Valvular
   B. Subvalvular
   C. Supravalvular
   D. Peripheral
   E. Combinations

II. Acquired (Rare)
   A. Rheumatic
   B. Endocarditis
   C. Carcinoid
   D. Large emboli
   E. Aneurysm of aorta

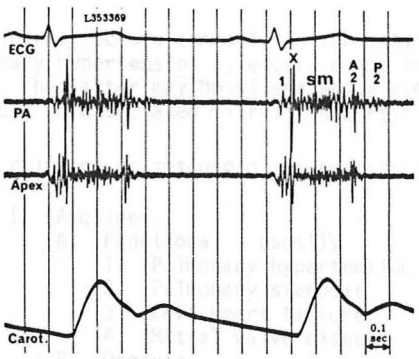
Pulmonary Stenosis

1. Timing	- Systolic
2. Duration	- Ejection diamond shaped; murmur ends with P_2 (passes A_2)
3. Intensity	- 1 - 6+
4. Pitch & Qualtiy	- High; rough
5. Radiation	- To neck, spine, scapula
6. Position	- Supine, legs up
7. Respiration	- Increases
8. Peripheral Signs	- Moon faces occasionally
9. Special Studies	- Valsalva decreases; amyl nitrite ↑ murmur
10. Clicks or O.S.	- Ejection click, ↓ with inspiration
11. S_3 and S_4	- Right-sided S_3 and S_4; persistent S_2 split or single S_2 (A_2)
12. Causes	- Congenital; rheumatic, rarely
13. Other	- Flow murmur often heard with pectus, ASD, anemia. Pregnancy, fever, exercise, idiopathic dilatation of the pulmonary artery; Co-existent peripheral P.S. occasionally



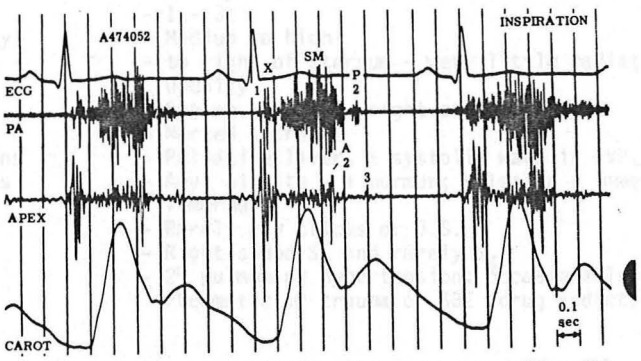
In valvular pulmonic stenosis with an intact ventricular septum, right ventricular systolic ejection becomes progressively longer with increasing obstruction to outflow. As a result, the murmur becomes louder and longer, enveloping the aortic closure sound. At the same time, pulmonic closure occurs later and splitting becomes wider, but it is more difficult to appreciate because the aortic closure sound is lost in the murmur and pulmonic closure becomes progressively fainter and lower-pitched. As the pulmonary diastolic pressure drops to 0, isometric contraction shortens to the point where the pulmonary ejection sound fuses with the first heart sound. In severe pulmonic stenosis, with concentric hypertrophy and decreasing right ventricular compliance, an atrial filling sound (A) appears.

from AHA



-Mild pulmonary stenosis in a 16-year-old male, featuring ejection sound (*X*), systolic ejection murmur, and late, soft P2. The ejection sound follows the first sound by 0.05 second, and is well recorded in the pulmonary area. The prolonged systolic murmur continues through A2.

from Tavel



-Severe pulmonary stenosis (right-ventricular systolic pressure 147 mm. Hg) in a 16-year-old male. Tracing at pulmonary area demonstrates an early systolic ejection sound (*X*) which decreases with inspiration (complex at right).

from Tavel

17. Tricuspid Regurgitation

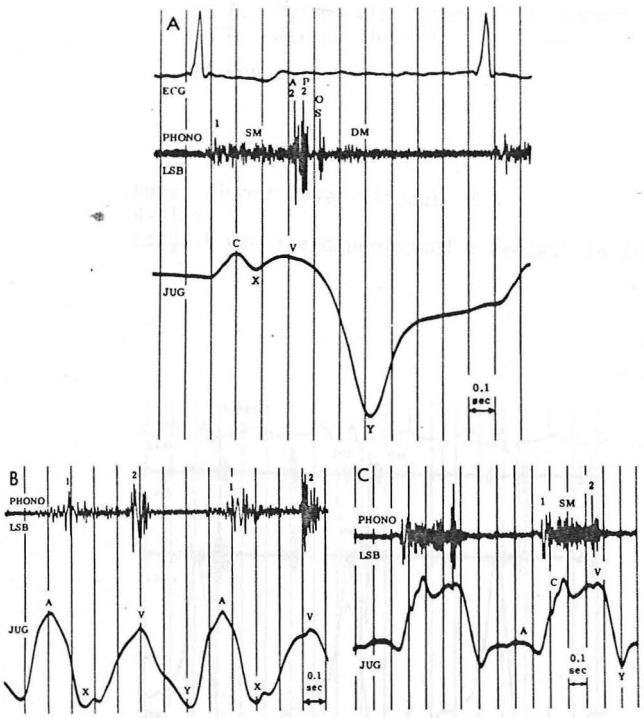
Tricuspid insufficiency occurs commonly in two settings in the adult: 1) secondary to pulmonary hypertension 2) endocarditis in the tricuspid valve in I.V. drug abusers. The latter may be silent while the former may be difficult to discern due to associated mitral and aortic valve murmurs.

ETIOLOGY OF TRICUSPID INSUFFICIENCY

I. Acquired
   A. Functional - usually
      1. Pulmonary hypertension
      2. Pulmonary stenosis
      3. Left heart failure
      4. Mitral valve disease
   B. Organic
      1. Rheumatic
      2. Trauma
      3. Endocarditis
      4. Carcinoid
      5. Ebstein's
   C. Congenital

Tricuspid Regurgitation

1.	Timing	- Systolic
2.	Duration	- Holosystolic or mid systolic
3.	Intensity	- 1 - 3+
4.	Pitch & Quality	- Medium to high
5.	Radiation	- to right of sternum - very little radiation usually
6.	Position	- Supine, legs up, right or mid-sternum
7.	Respiration	- Marked increase
8.	Peripheral Signs	- Pulsatile liver, ↑ systolic wave in JVP, edema
9.	Special Studies	- Amyl nitrite - ↑ murmur; Valsalva - immediately ↓ murmur
10.	Clicks or O.S.	- Rarely any clicks or O.S.
11.	S_3 and S_4	- Right-sided S_3 and rarely S_4
12.	Causes	- 2^0 pulmonary hypertension; occasionally rheumatic or trauma or SBE (drug addicts)
13.	Other	



-Jugular pulse tracings in tricuspid insufficiency. **A,** typical pattern in tricuspid insufficiency with atrial fibrillation. The *X* descent is completely obliterated by a long regurgitant *V* wave. **B,** tricuspid insufficiency with normal sinus rhythm. The *X* descent is well preserved but is not quite as deep as the *Y* descent. The *V* wave begins during the early part of ventricular systole. In normal sinus rhythm, the *X* descent should be deeper than the *Y* descent, and reversal of this relationship suggests tricuspid disease. **C,** frank tricuspid insufficiency (normal sinus rhythm). During all of ventricular systole, the pulse curve is deflected upward, demonstrating so-called ventricularization of the venous pulse.

from Tavel

18. Tricuspid Stenosis

Tricuspid stenosis is rare with the rapid disappearance of recurrent bouts of rheumatic carditis. When it does occur, it is almost always associated with mitral valve murmurs, resulting in difficulty in auscultating a separate valvular murmur over the tricuspid area. The jugular venous pulse changes with tricuspid stenosis are perhpas the best clue to the existance of the tricuspid stenosis.

ETIOLOGY OF TRICUSPID STENOSIS

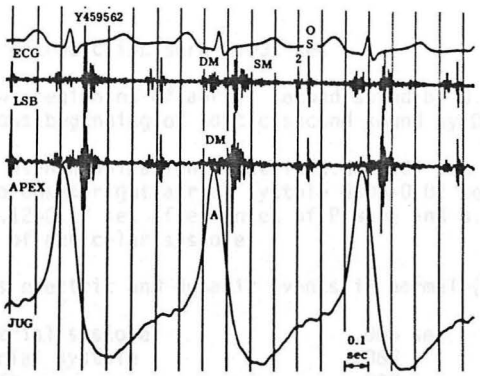
1. Organic
   A. Rheumatic, 3-5% of RHD
   B. Carcinoid syndrome

II. Functional - Rare
   A. Myxoma and other atrial tumors
   B. Atrial thrombi

III. Congenital

Tricuspid Stenosis

Rare, almost never without M.S.
4-LICS
Large A waves and prolonged y descent in JVP



-Tricuspid stenosis combined with mitral stenosis. At the left sternal border, a late diastolic (atriosystolic) tricuspid murmur is seen which *precedes* the apical presystolic accentuation of the mitral murmur, located in the bottom sound tracing. Note also the giant jugular *A* waves of tricuspid stenosis.

from Tavel

TIMING OF CARDIAC EVENTS

Normals - (GRAY)

QRS onset to: 1) Mitral closure - 0.059 sec (+ 0.010)
2) Tricuspid closure - 0.082 sec (+0.013)
3) Carotid upstroke - 0.121
(corrected for delay 0.089 sec ± 0.018)

Right Ventricular Systole

1) Tricuspid closure to pulmonary closure - 0.314 sec
(corrected for cycle length 0.349 ± 0.023)

Left Ventricular Systole

1) Mitral to aortic closure - 0.310 sec
(corrected for cycle length 0.345 ± 0.023)

Carotid Delay

Aortic closure to dicrotic incisura 0.032 sec

Third sound follows beginning of aortic second sound by 0.11-0.14 sec
Opening snap follows beginning of aortic second sound by 0.1 sec or less

Normal electrical asynchronization of atria 0.03-0.04 sec
Onset of P wave to onset right atrial systole 0.05-0.07 sec
Auricular sound 0.12-0.17 sec after onset of P wave and o.05-0.09 sec after onset of auricilar systole

Temporal relations electric and dynamic events in normal (BRAUNWALD)

Onset P - right atrial systole	.065 sec
Onset P - left atrial systole	.085
Onset right to left	.020
Onset Q - right ventricular systole	.065
Onset Q - left ventricular systole	.052
Onset left to right ventricular systole	.013
Onset Q - right ventricular ejection	.080
Onset Q - left ventricular ejection	.115
Onset right to left ventricular ejection	.035
Right ventricular isometric contraction	.016
Left ventricular isometric contraction	.061

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