CARDIAC TAMPONADE

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CARDIAC TAMPONADE

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I. Introduction

A. General

1. Wood, P.: Diseases of the Heart and Circulation. Ed. 2. Philadelphia: J. B. Lippincott Co., 1956.
2. Hurst, J. W. and Logue, R. B. (eds.): The Heart: Arteries and Veins. New York: McGraw-Hill Book Co., 1966.
3. Friedberg, C. K.: Diseases of the Heart. Vol. I, ed. 3. Philadelphia and London: W. B. Saunders Co., 1966.
4. Burwell, C. S.: Diseases of the pericardium. In: Oxford Medicine. pp. 251-284. New York: Oxford University Press, 1940.
5. Spodick, D. H.: Acute Pericarditis. New York: Grune and Stratton, 1959.
6. Spodick, D. H.: Chronic and Constrictive Pericarditis. New York: Grune and Stratton, 1964.
7. McIntosh, H. D.: Pericarditis. Disease-a-Month April, 1964.
8. Shabetai, R., Fowler, N. O. and Fenton, J. C.: Restrictive cardiac disease. Pericarditis and the myocardiopathies. Am. Heart J. 69:271, 1965.
9. Spodick, D. H.: Acute cardiac tamponade: pathologic physiology, diagnosis and management. Prog. Cardiovas. Dis. 10:64, 1967.

Cardiac compressive or restrictive disorder is a general term used to describe a variety of pathological conditions with a similar cardiodynamic abnormality. All of these conditions tend to hinder the filling of the cardiac chambers from their respective venous reservoirs. This resistance to filling may arise in the pericardium, epicardium, myocardium, or endocardium.

B. Definition

1. Loc cit
3. Loc cit
9. Loc cit

Cardiac tamponade is defined as the decompensated phase of cardiac compression caused by an unchecked rise in intrapericardial pressure due to fluid accumulation. The terms pericardial effusion and cardiac tamponade are not synonymous. A very small pericardial effusion which occurs acutely may cause cardiac tamponade, and a very large pericardial effusion which occurs slowly may not result in cardiac tamponade. With the resistance to filling caused by the elevated pericardial fluid pressure, there is a decrease in cardiac output. Compensatory mechanisms tend to maintain

systemic perfusion pressure and peripheral oxygenation. It is only when these have failed that cardiac tamponade exists. This condition is illustrated in Figure 1.

II. Pathophysiologic Mechanisms

A. Causative mechanisms

1. Hindrance to cardiac filling

10. Cohnheim, J.: Lecture on General Pathology. Section I: The Pathology of the Circulation. London: The New Sydenham Society, 1889.
11. Beck, C. S.: Acute and chronic compression of the heart. Am. Heart J. 14:515, 1937.
12. Beck, C. S.: Heart: extrinsic lesions. In: Medical Physics, ed. by O. Glasser. Vol. 1, p. 570. Chicago: Year Book Publishers, 1950.
13. Starling, E. H.: Some points in the pathology of heart disease. Lancet 1:652, 1897.
14. Kuno, Y.: The mechanical effect of fluid in the pericardium on the function of the heart. J. Physiol. 51:221, 1917.
15. Nerlich, W. E.: Determinants of impairment of cardiac filling during progressive pericardial effusion. Circulation 3:377, 1951.
16. Metcalfe, J., Woodbury, J. W., Richards, V. and Burwell, C. S.: Studies in experimental pericardial tamponade; effects on intravascular pressures and cardiac output. Circulation 5:518, 1952.
17. Isaacs, J. P., Berglund, E. and Sarnoff, S. J.: Ventricular function: pathologic physiology of acute cardiac tamponade studied by means of ventricular function curves. Am. Heart J. 48:66, 1954.
18. Martin, J. W. and Schenk, W. G., Jr.: Pericardial tamponade: newer dynamic concepts. Am. J. Surg. 99:782, 1960.
19. Kuhn, L. A.: Cardiodynamics of pericardial disease. J. Mt. Sinai Hosp. 13:382, 1964.
20. Craig, R. J., Whalen, R. E., Behar, V. S., Thompson, H. K., Jr. and McIntosh, H. D.: Ventricular volume changes in acute pericardial tamponade. Circulation 34:III-80, 1966.

It has been abundantly shown that injection of fluid in the pericardium raises pericardial pressures, decreases cardiac output, and causes a fall in arterial blood pressure. Cohnheim suggested that the increased pericardial pressure caused a blockage to the

entrance of blood from the systemic veins and thereby diminished the amount of blood flowing into the heart. This concept has been further supported by Beck. Starling felt that the increased pericardial pressure caused a hindrance to diastolic expansion of the heart. The experimental study of the Sarnoff group and the recent data of McIntosh et al prove this latter concept to be true. This is illustrated in Figure 2.

2. Decrease in cardiac contractility

17. Loc cit
20. Loc cit
21. Ferguson, R., Bristow, D., Mintz, F. and Rapaport, E.: Thermodilution used to assess the effects of pericardial tamponade on ventricular function and volumes. Clin. Res. 11:100, 1963.
22. O'Rourke, R. A., Fischer, D. P., Escobar, E. E., Bishop, V. S. and Rapaport, E.: Effect of acute pericardial tamponade on coronary blood flow. Am. J. Physiol. 212:549, 1967.

The Sarnoff group could not demonstrate any shift of the ventricular function curve during cardiac tamponade, and, therefore, concluded that no change in cardiac contractility was involved in the pathophysiology of this condition. Ferguson et al have suggested that a decrease in ventricular contractility occurs during cardiac tamponade and O'Rourke and his co-workers have found a decrease in coronary flow during tamponade which they feel may account for the decreased performance of the left ventricle. McIntosh and his group also feel that a decrease in contractility of the ventricle occurs during cardiac tamponade.

B. Compensatory mechanisms

5. Loc cit
9. Loc cit

Spodick presents an analysis of the compensatory mechanisms which are brought into play during cardiac tamponade. There are several theoretical shortcomings to his analysis. Table I is my scheme of the compensatory mechanisms during cardiac tamponade.

COMPENSATORY MECHANISM IN CARDIAC TAMPONADE

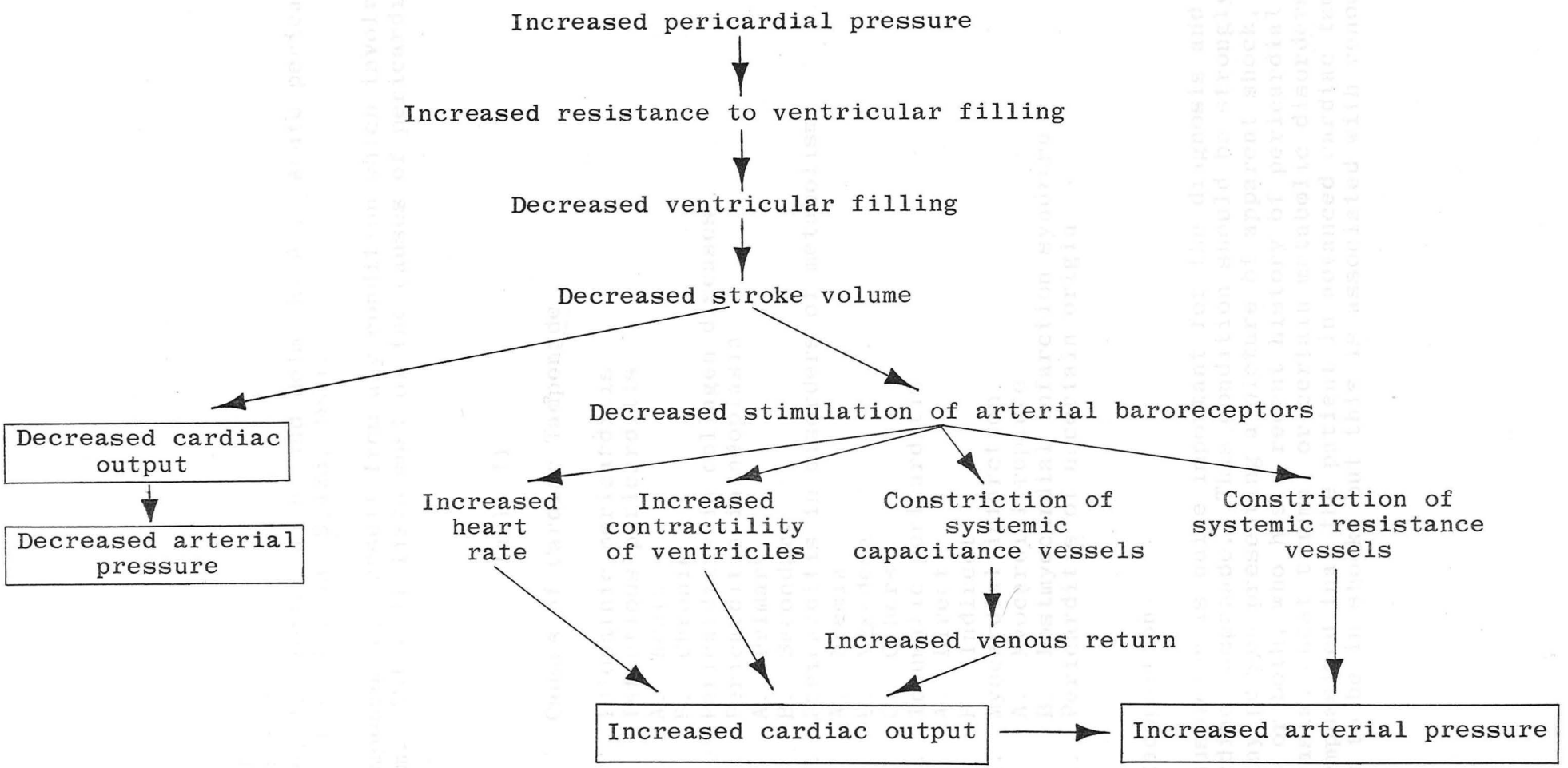

Table I

III. Etiology

1. Loc cit
9. Loc cit
23. McGuire, J., Kotte, J. H. and Helm, R. A.: Acute pericarditis. Circulation 9:425, 1954.

Cardiac tamponade can result from any condition which involves the pericardium. Table II lists most of the causes of pericardial tamponade.

Table II

Causes of Cardiac Tamponade

I. Idiopathic pericarditis
II. Infectious pericarditis
  A. Acute
  B. Chronic
III. Pericarditis in collagen diseases
IV. Pericarditis in neoplasia
  A. Primary
  B. Secondary
V. Pericarditis in disorders of metabolism
  A. Uremia
  B. Myxedema
  C. Others
VI. Traumatic pericarditis
  A. Direct
  B. Indirect
VII. Myocardial infarction
  A. Myocardial rupture
  B. Postmyocardial infarction syndrome
VIII. Pericarditis of uncertain origin

V. Clinical Recognition

Clinical suspicion is quite important for the diagnosis and therapy of cardiac tamponade. This condition should be strongly suspected in any person presenting a picture of apparent shock, heart failure, or both, who has a recent history of pericardial disease, neoplasia, chest trauma, or certain metabolic disorders. It should be emphasized that the patient in advanced cardiac tamponade appears to be in shock, but this is associated with venous hypertension.

A. Symptoms

9. Loc cit
24. Williams, C. and Soutter, L.: Pericardial tamponade: diagnosis and treatment. Arch. Int. Med. 94:571, 1954.
25. Soloff, L. A. and Zatuchni, J.: The definitive diagnosis of effusive or constrictive pericarditis. Am. J. Med. Sci. 234:687, 1957.
26. Ray, C. T.: Problems in differentiating pericardial effusion from generalized dilatation of the heart. Bull. Tulane Univ. Med. Fac. 17:165, 1958.
27. Burch, G. E. and Phillips, J. H.: Methods in the diagnostic differentiation of myocardial dilatation from pericardial effusion. Am. Heart J. 64:266, 1962.

Table III gives the symptoms of cardiac tamponade.

Table III

Symptoms of Cardiac Tamponade

1. Chest pain
2. Dyspnea
   Orthopnea is frequently present but not due to pulmonary vascular congestion
   Paroxysmal nocturnal dyspnea does not occur
3. Cough, hoarseness, and hiccups
4. Right upper quadrant pain

B. Signs

1. Examination of the chest

a. Inspection, palpation, and percussion

9. Loc cit
26. Loc cit
27. Loc cit
28. Durant, T. M.: The recognition of pericardial disease. Mod. Concepts Cardiovas. Dis. 27:455, 1958.
29. Dressler, W.: Percussion of the sternum. I. Aid to differentiation of pericardial effusion and cardiac dilatation. J.A.M.A. 173:761, 1960.

Suggestive evidence of pericardial effusion can be obtained by inspection, palpation, and percussion of the chest. A visual

diminution or absence of the apical impulse, and percussion of cardiac dullness well outside of the palpable apical impulse may be noted. Also the presence of clear lung fields with marked cardiomegaly is suggestive. The detection of an area of dullness, bronchial breathing, and bronchophony at the base of the left lung (Ewart's sign) is a strong clue for the diagnosis of pericardial effusion; however, this same sign may be present in congestive heart failure.

b. Auscultation

9. Loc cit
26. Loc cit
27. Loc cit
28. Loc cit
30. Harvey, W. P. and Mandanis, J. P.: Auscultatory findings in pericardial effusion and in ventricular aneurysm. Circulation 18:1034, 1958.
31. Harvey, W. P.: Auscultatory findings in diseases of the pericardium. Am. J. Cardiol. 7:15, 1961.
32. Phillips, J. H., Jr. and Burch, G. E.: Editorial: selected clues in cardiac auscultation. Am. Heart J. 63:1, 1962.
33. McKusick, V. A.: Cardiovascular Sound in Health and Disease. Baltimore: Williams and Wilkins Co., 1958.
34. Lange, R. L., Botticelli, J. T., Tsagaris, T. J., Walker, J. A., Gani, M. and Bustamante, R. A.: Diagnostic signs in compressive cardiac disorders: constrictive pericarditis, pericardial effusion, and tamponade. Circulation 33:763, 1966.

Both heart sounds are frequently muffled or distant and splitting of the second sound is difficult to distinguish. Also most patients with inflammatory effusions have a pericardial friction rub, regardless of the amount of pericardial fluid. While it is true that an abnormal early diastolic sound is usually inaudible, in occasional patients a third heart sound may be heard and recorded. This is probably caused by a rather sudden end of the first rapid filling phase of the ventricle as is frequently found in constrictive pericarditis and called a "pericardial knock". Also a fourth heart sound (atrial sound) can rarely be heard. Probably the third and fourth heart sounds are not frequently noted because of the damping effect of the pericardial fluid. One has a better chance of eliciting these findings with phonocardiography.

2. Examination of the peripheral vessels

a. Kussmaul's sign

9. Loc cit
26. Loc cit
27. Loc cit
28. Loc cit
34. Loc cit
35. Kussmaul, A.: Über schwielige Mediastino-perikarditis und den paradoxen Puls. Berlin klin. Wchnschr. 10:433, 445, 461, 1873.
36. Hitzig, W. M.: On mechanisms of inspiratory filling of the cervical veins and pulsus paradoxus in venous hypotension. J. Mt. Sinai Hosp. 8:625, 1942.

Kussmaul's sign is a regular inspiratory increase in venous pressure during ordinary or quiet breathing which may be observed in the neck veins or recorded in the right atrium. This phenomenon is "paradoxical" in the sense that the venous and right atrial pressures usually decrease during normal inspiration. This sign is frequently noted in pericardial tamponade unless the venous pressure is so high that the variations with respiration are difficult to elicit. It should also be noted that Kussmaul's venous signs occur in other conditions such as constrictive pericarditis and right ventricular failure.

b. Friedreich's sign

34. Loc cit
37. Friedreich, N.: Zur Diagnose der Herzbeutelverwachsungen. Virchow Arch. Path. Anat. 29:296, 1864.

Friedreich's sign is an early diastolic pressure dip observed in the neck veins or recorded in the right atrium. This neck vein sign has rarely been reported in pericardial effusion but is frequently noted in constrictive pericarditis, restrictive myocardiopathies, and severe right heart failure. Cardiac catheterization studies in patients with constrictive pericarditis or restrictive myocardiopathy demonstrate an early diastolic dip in right atrial and right ventricular pressures. This finding can also be observed in patients with pericardial effusion.

c. Pulsus paradoxus

1) General

9. Loc cit
34. Loc cit
35. Loc cit

38. Gauchat, H. W. and Katz, L. N.: Observations on pulsus paradoxus (with special reference to pericardial effusion). I. Clinical. Arch. Int. Med. 33:350, 1924.
39. Editorial: Paradox of the paradoxical pulse. New England J. Med. 242:990, 1950.
40. Sharp, J. T., Bunnell, I. L., Holland, J. F., Griffith, G. T. and Greene, D. G.: Hemodynamics during induced cardiac tamponade in man. Am. J. Med. 29:640, 1960.

By common consent the term paradoxical pulse or "pulsus paradoxus" refers to a phenomenon in which the arterial pulse is markedly diminished, or even abolished, during ordinary or quiet inspiration and reappears promptly during expiration. In 1873 Kussmaul first suggested that the phenomenon be termed paradoxical, ".... partly because of the striking disproportion between the action of the heart and the arterial pulse, and partly because the pulse, although apparently irregular, shows in reality a regular waxing and waning". He said nothing about a reversal of the normal response of the pulse to the phases of respiration. Clearly, the "paradox" involved the uniform and unchanged precordial impulse while the arterial pulse waxed and waned with respiration. A better term for the phenomenon now called "pulsus paradoxus" might be "exaggerated respiratory pulse variation".

Some of the various causes of pulsus paradoxus are shown in Table IV.

Table IV

Causes of Pulsus Paradoxus

1. Pericardial effusion
2. Constrictive pericarditis
3. Congestive heart failure
4. Obstructive and restrictive respiratory diseases
5. Exaggerated respiratory excursions

Pulsus paradoxus can best be quantitated by use of a sphygmomanometer. The sphygmomanometer cuff is inflated above systolic pressure and then slowly deflated until the first beats are heard. It will be obvious that most of the beats are not being heard and those that are, "come through" during expiration. The cuff is then deflated until all beats are heard - or until all beats disappear, as occurs when the pulsus occupies the entire pulse pressure down to the diastolic pressure. The difference in millimeters of mercury between the first intermittently-heard beats and the final

appearance or disappearance of all the beats is recorded as the amount of pulsus paradoxus. A 10 mm Hg pulsus paradoxus can be found in normal subjects. Many feel that a value above 10 mm Hg is pathological, but others require a 20 mm Hg difference to be significant.

2) Pathophysiologic mechanisms

8. Loc cit
9. Loc cit
27. Loc cit
36. Loc cit.
40. Loc cit
41. Katz, L. N. and Gauchat, H. W.: Observations on pulsus paradoxus (with special reference to pericardial effusion). II. Experimental. Arch. Int. Med. 33:371, 1924.
42. Dornhorst, A. C., Howard, P. and Leathart, A. I.: Pulsus paradoxus. Lancet 1:746, 1952.
43. Golinko, R. J., Kaplan, N. and Rudolph, A. M.: The mechanism of pulsus paradoxus during acute pericardial tamponade. J. Clin. Invest. 42:249, 1963.
44. Shabetai, R., Fowler, N. O., Braunstein, J. R. and Gueron, M.: Transmural ventricular pressures and pulsus paradoxus in experimental cardiac tamponade. Dis. Chest 39:557, 1961.
45. Shabetai, R., Fowler, N. O. and Gueron, M.: The effects of respiration on aortic pressure and flow. Am. Heart J. 65:525, 1963.
46. Shabetai, R., Fowler, N. O., Fenton, J. C. and Masangkay, M.: Pulsus paradoxus. J. Clin. Invest. 44:1882, 1965.
47. Guntheroth, W. G., Morgan, B. C. and Mullins, G. L.: Effect of respiration on venous return and stroke volume in cardiac tamponade. Mechanism of pulsus paradoxus. Circulation Res. 20:381, 1967.
48. Dock, W.: Inspiratory traction on the pericardium. Arch. Int. Med. 108:837, 1961.

The pathophysiologic mechanism or mechanisms of pulsus paradoxus have interested both cardiovascular physiologists and cardiologists for many years. The normal fall in arterial blood pressure during inspiration has been shown to be caused by a decrease in left ventricular stroke output. During inspiration there is a decreased inflow into the left heart in spite of an increased inflow and thereby output of the right heart. This increased right heart output is "sponged up" by the increased vascular capacitance of the pulmonary vessels. Three theories have been offered to explain the exaggerated drop in left ventricular stroke output seen during pericardial effusion. These are 1) the "sponge effect" is

accentuated because right heart inflow and thereby output does not go up during inspiration because the intrathoracic pressure drop is not transmitted to the right heart chambers; 2) the decreased left ventricular stroke output during inspiration is increased because right heart filling increases as usual and tends to prevent left heart filling; and 3) right heart inflow is decreased because the descent of the diaphragm during inspiration increases resistance to filling of the right heart chamber. The second theory fits best with the experimental facts.

V. Diagnostic Methods

A. Venous pressure and circulation time

4. Loc cit
9. Loc cit
27. Loc cit
34. Loc cit
36. Loc cit
49. Esser, H. K. and Berliner, K.: Duplicate measurements of circulation time made with the saccharin method. Ann. Int. Med. 19:64, 1943.
50. Bellet, S., Nadler, C. S. and Steiger, W. A.: Circulation time (arm to tongue time) in large pericardial effusions, an aid in differential diagnosis between large pericardial effusions and cardiac dilatation. Ann. Int. Med. 34:856, 1951.

The venous pressure is elevated in pericardial effusion. Also one may detect an inspiratory increase in venous pressure. In uncomplicated cardiac tamponade the circulation time is usually normal. However, a prolongation can occur with marked tamponade which principally reflects a decrease in the blood flow velocity (decreased cardiac output). In the absence of underlying cardiomegaly or heart failure, moderate prolongation of the arm to tongue circulation time beyond 16 sec may occur, with return to normal after a slight decrease in the amount of pericardial fluid.

B. Electrocardiography

1. General

9. Loc cit
26. Loc cit
27. Loc cit
51. Hull, E.: The electrocardiogram in pericarditis. Am. J. Cardiol. 7:21, 1961.

52. Toney, J. C. and Kolmen, S. N.: Cardiac tamponade: fluid and pressure effects on electrocardiographic changes. Proc. Soc. Exper. Biol. Med. 121:642, 1966.

The voltage of the electrocardiogram is frequently decreased in pericardial effusion but this is a nonspecific finding. Experimental studies have shown that when this occurs it depends primarily on the hemodynamic abnormality (decreased arterial pressure and venous pressure) rather than on mere insulation by the pericardial fluid. Also electrocardiographic findings consistent with acute pericarditis prior to or concurrent with evidence of cardiac tamponade are very helpful, especially if they show the expected evolution.

2. Electrical alternans

53. Hering, H. E.: Experimentelle Studien an Saugethienen über das Elektrocardiogramm II. Z. exp. Path. Ther. 7:363, 1909.
54. Littmann, D. and Spodick, D. H.: Total electrical alternation in pericardial disease. Circulation 17:912, 1958.
55. Spodick, D. H.: Electric alternation of the heart. Its relation to the kinetics and physiology of the heart during cardiac tamponade. Am. J. Cardiol. 10:155, 1962.
56. Littmann, D.: Alternation of the heart. Circulation 27: 280, 1963.

A rather uncommon finding - simultaneous electrical alternans of both the P waves and QRS complexes - is virtually pathognomonic of cardiac tamponade. Ventricular alternans (only the QRS is seen to alternate) is much more commonly found in cardiac tamponade but is much less specific. The exact mechanism of electrical alternans during cardiac tamponade is not clear, but two theories have been proposed. One suggests that it is due to anatomic oscillation and the other that it is due to a conduction aberration.

3. Ice water test

57. Friedman, B. and McClure, H. H.: A simple bloodless and painless presumptive test for pericardial fluid and thickening. Am. J. Med. Sci. 244:321, 1962.

Normal subjects and cardiac patients with or without enlarged hearts show transient changes in the T vector of the electrocardiogram after drinking ice water. These changes are usually a decreased positivity in leads II, III, and aVf; and increased positivity in aVl and V_1-V_4. This effect of ice water is markedly de-

creased or absent in patients with pericardial effusion or thickening. This is a harmless and simple presumptive test for the presence of pericardial effusion.

C. Echocardiography

9. Loc cit
58. Edler, I.: Diagnostic use of ultrasound in heart disease. Acta Med. Scand. Suppl. 308:32, 1955.
59. Effert, S., Erken, H. G. and Grossbrockehoff, T.: Echo method in cardiological diagnosis. Germ. Med. Month. 2: 325, 1957.
60. Feigenbaum, H., Waldhausen, J. A. and Hyde, L. P.: Ultrasound diagnostic of pericardial effusion. J.A.M.A. 191: 711, 1965.
61. Moss, A. J. and Bruhn, F.: The echocardiogram. New England J. Med. 274:380, 1966.
62. Feigenbaum, H., Zaky, A. and Waldhausen, J. A.: Use of reflected ultrasound in detecting pericardial effusion. Am. J. Cardiol. 19:84, 1967.
63. Christensen, E. E. and Bonte, F. J.: The relative accuracy of echocardiography, intravenous carbon dioxide, and blood pool scanning in detecting pericardial effusions in dogs. In press.

It was first shown that reflected ultrasound could be used to detect pericardial fluid anterior to the anterior heart wall. This method did not prove very useful since the anterior heart wall could rarely be identified normally; thus to exclude the presence of a pericardial effusion, one relied on negative findings. Feigenbaum et al have used reflected ultrasound to detect pericardial fluid in the vicinity of the posterior wall of the heart. Recently Christensen and Bonte have shown in dog experiments that echocardiography of the anterior wall is a more accurate and sensitive method of determining pericardial effusion than gas cardiography with carbon dioxide and radioisotope scanning of the cardiac chambers. The echocardiogram appears to be a simple, innocuous, and relatively accurate method for the detection of pericardial effusion.

D. Phonocardiography

6. Loc cit
9. Loc cit
33. Loc cit
64. McKusick, V. A. and Harvey, A. M.: Diseases of the pericardium. Adv. Int. Med. 7:157, 1955.
65. Cohen, L. S. and Braunwald, E.: Personal communications.

The findings here are the same as those discussed in the section on auscultation. This is probably a more sensitive way to pick up a "pericardial knock" and a fourth heart sound (atrial sound). This is shown in Figure 3. However, the pericardial fluid still has a damping effect when the phonocardiogram is obtained from the precordium. It is possible that these findings might be picked up much more frequently by intracardiac phonocardiography.

E. Chest radiography

9. Loc cit
27. Loc cit
66. Arendt, J.: Radiological differentiation between pericardial effusion and cardiac dilatation. Radiology 50:44, 1948.
67. Torrance, D. J.: Demonstration of subepicardial fat as an aid in the diagnosis of pericardial effusion or thickening. Am. J. Roentgenol. 74:850, 1955.
68. Mellins, H. Z., Kottmeier, P. and Kiely, B.: Radiologic signs of pericardial effusion: an experimental study. Radiology 73:9, 1959.
69. Steinberg, I.: Roentgenography of pericardial disease. Am. J. Cardiol. 7:33, 1961.

Conventional x-ray views of the heart demonstrate an enlarged cardiopericardial shadow in most patients with pericardial effusion. Cardiomegaly with clear lung fields is very suggestive. When the patient is erect, the shadow appears bottle-shaped, and when recumbent it appears globular with a widening of the base of the heart. Fluoroscopic examination reveals decreased pulsations of the cardiac borders. However, these findings are not diagnostic of pericardial effusion since they can also be due to cardiac dilatation.

F. Radioisotope scanning of cardiac chambers

9. Loc cit
27. Loc cit
70. Wagner, H. N., Jr., McAfee, J. G. and Mozley, J. M.: Diagnosis of pericardial effusion by radioisotope scanning. Arch. Int. Med. 108:679, 1961.
71. O'Meallie, L. P., Love, W. D. and Burch, G. E.: Differentiation of massive pericardial effusion from cardiac dilatation using I^{131} albumin. Am. Heart J. 62:453, 1961.
72. Bonte, F. J., Andrews, G. J., Elmendorf, E. A., Presley, N. L. and Krohmer, J. S.: Radioisotope scanning in the detection of pericardial effusions. South. Med. J. 55:577, 1962.

73. Johnson, P. M.: Some diagnostic applications of combined radioisotope scanning of adjacent organs. J.A.M.A. 194: 455, 1965.
74. Bonte, F. J. and Curry, T. S., III: The radioisotope blood pool scan. Am. J. Roentgenol. 96:690, 1966.
75. Miercort, R. D. and Brown, D. W.: Radioisotope scintillation scanning in the evaluation of pericardial effusion. J.A.M.A. 200:168, 1967.

This method utilizes precordial photoscanning following the intravenous injection of I^{131}-labeled human serum albumin (RISA). The cardiopericardial shadow obtained by a chest roentgenogram is then compared with the scan of radioactivity recorded from the RISA which has entered the intracardiac blood pool. The presence or absence of pericardial effusion is based on the following points: 1) a significant discrepancy between the areas and transverse diameters of the cardiac blood pool scan and those of the radiographic cardiopericardial shadow, and 2) areas of decreased radioactivity separating the radioactive concentration of the cardiac blood pool from those of the lung and liver. Bonte and his group have recently introduced the use of technetium (Tc^{99m}) labeled human serum albumin instead of RISA.

G. Gas cardiography with carbon dioxide

9. Loc cit
27. Loc cit
76. Scatliff, J. H., Kummer, A. J. and Janzen, A. H.: The diagnosis of pericardial effusion with intracardiac carbon dioxide. Radiology 73:871, 1959.
77. Durant, T.: Negative (gas) contrast angiocardiography. Am. Heart J. 61:1, 1961.

The thickness of the pericardium or pericardial sac can be assessed by negative-contrast roentgenography using the intracardiac injection of carbon dioxide. With the patient in the left lateral decubitus, 50 to 100 ml of carbon dioxide injected into the right atrium will outline the right lateral limits of the chamber. Radiographs demonstrate the aerated lungs, the opaque right atrial wall or "band", the carbon dioxide gas "bubble", and the blood level. The "band" includes the pleura, pericardium, and right atrial wall and normally measures 5 mm or less. When the band is wider than 10 mm, thickening or an effusion is present, and when the band is wider than 20 mm, an effusion is present.

H. Angiocardiographic study

9. Loc cit

78. Williams, R. G. and Steinberg, I.: Value of angiocardiography in establishing the diagnosis of pericarditis with effusion. Am. J. Roentgenol. 61:41, 1949.
79. Jorgens, J., Kundel, R. and Lieber, A.: The cinefluorographic approach to the diagnosis of pericardial effusion. Am. J. Roentgenol. 87:911, 1962.
80. Dinsmore, R. E., Miller, A. R., Potsaid, M. S. and Shawdon, H. H.: Cineangiographic patterns in pericardial disease. Radiology 86:425, 1966.
81. Shuford, W. H., Sybers, R. G., Acker, J. J. and Weens, H. S.: A comparison of carbon dioxide and radiopaque angiocardiographic methods in the diagnosis of pericardial effusion. Radiology 86:1064, 1966.

Angiocardiography is quite useful for demonstrating the presence of pericardial effusion. By opacifying the chambers of the heart, thickness of the heart wall can be estimated. The thickness of the wall of the normal right atrium is 2 to 3 mm, the right ventricle 3 to 5 mm, and the left ventricle about 10 mm. A heart wall thickness greater than these widths, especially if it surrounds the heart in the frontal view, is due to pericardial effusion, pericardial thickening, or an extracardiac mass.

I. Cardiac catheterization studies

1. Distance between right atrial chamber and right heart border

1. Loc cit
82. Wood, P.: Diagnosis of pericardial effusion by means of cardiac catheterization. Brit. Heart J. 13:574, 1951.

Wood introduced a method of diagnosing pericardial fluid by passing a half looped catheter with the tip directed laterally into the right atrium. Normally when the catheter tip lies against the right atrial wall, no opacity can be seen between it and the right lung field. When a pericardial effusion is present, a significant band of opacity appears between the catheter tip and the right lung field.

2. Pressures in cardiac chambers

83. Isaacs, J. P., Carter, M. N., II and Haller, J. A., Jr.: Experimental pericarditis: the pathologic physiology of constrictive pericarditis. Bull. Johns Hopkins Hosp. 90: 239, 1952.

84. Wilson, R. H., Hoseth, W., Sadoff, C. and Dempsey, M. E.: Pathologic physiology and diagnostic significance of the pressure pulse tracings in the heart in patients with constrictive pericarditis and pericardial effusion. Am. Heart J. 48:671, 1954.
85. Cohen, L. S. and Braunwald, E.: Personal communications.

Cardiac catheterization studies reveal similar findings in all the disorders (pericardial, epicardial, myocardial, and endocardial) that cause cardiac compressive or restrictive disorder. The right ventricular pressure curve, particularly in constrictive pericarditis, consists of an early diastolic dip followed by a diastolic plateau. The level of the diastolic plateau is at a pressure roughly equal to the mean atrial pressure and peripheral venous pressure, and usually at a pressure in excess of one-third of the ventricular systolic pressure. The right atrial pressure curve usually has an "m" shape in each cardiac cycle. Some of these findings are shown in Figure 4. It is thus clear that cardiac catheterization studies cannot distinguish pericardial fluid causing compression from other disorders which cause a similar cardiodynamic abnormality.

VI. Therapy

The definitive therapy for cardiac tamponade is to remove the pericardial fluid. However, it may be necessary to maintain the circulation by supportive methods until the pericardial fluid can be removed. Such measures are indicated when the patient's condition is perilous and 1) the diagnosis is not adequately established, 2) the equipment for removal of the pericardial effusion is not available, 3) the attempted removal of pericardial effusion is unsuccessful, and 4) the tamponade is associated with significant blood loss.

A. Supportive

1. Intravenous fluids

9. Loc cit
86. Beck, C. S.: Further observations on stab wounds of the heart. Ann. Surg. 115:698, 1942.
87. Cooper, F. W., Stead, E. A. and Warren, J. V.: The beneficial effect of intravenous infusions in acute pericardial tamponade. Ann. Surg. 120:822, 1944.

The intravenous infusion of blood or dextran expands the venous blood volume, raises venous pressure, and thereby increases caridac

filling. The effectiveness of this therapy can be judged by the effect on arterial blood pressure.

2. Beta-adrenergic stimulation

9. Loc cit
14. Loc cit
17. Loc cit
88. Binion, J. T., Morgan, W. J., Jr., Welch, G. H. and Sarnoff, S. J.: Effect of sympathomimetic drugs in acute experimental cardiac tamponade. Circulation Res. 4:705, 1956.
89. Krasnow, N., Rolett, E. L., Yurchak, P. M., Hood, W. R., Jr. and Gorlin, R.: Isoproterenol and cardiovascular performance. Am. J. Med. 37:514, 1964.

In 1917 Kuno found that adrenalin administered during cardiac tamponade increased the arterial pressure and decreased the venous pressure. Binion et al demonstrated that Aramine and Wyamine by augmenting cardiac contractility induce the heart to do more stroke work without an increase in end-diastolic fiber length. However, these drugs also cause increased peripheral vascular resistance and would tend to decrease systolic emptying. An ideal drug would appear to be isoproterenol which stimulates only the beta-adrenergic receptors. This would increase cardiac contractility and decrease peripheral vascular resistance, and thereby augment the stroke volume delivered from any given level of end-diastolic filling. To my knowledge isoproterenol has not been used in cardiac tamponade.

3. Digitalis

9. Loc cit
27. Loc cit
90. McMichael, J. and Sharpey-Schafer, E. P.: Action of intravenous digoxin in man. Quart. J. Med. 37:123, 1945.

Digitalis would also appear to be a useful agent in cardiac tamponade since it increases cardiac contractility. Clinically this has not proved to be so and is due to the fact that digitalis probably decreases venous return in this condition.

B. Definitive (removal of pericardial effusion)

1. Pericardial tap (pericardicentesis)

9. Loc cit
91. Kotte, J. H. and McQuire, J.: Pericardial paracentesis. Mod. Concepts Cardiovas. Dis. 20:102, 1951.

92. Bishop, L. H., Jr., Estes, E. H., Jr. and McIntosh, H. D.: The electrocardiogram as a safeguard in pericardiocentesis. J.A.M.A. 162:264, 1956.
93. Neill, J. R., Hurst, J. W. and Renfold, E. L. J.: A pericardiocentesis electrode. New England J. Med. 264:711, 1961.
94. Kilpatrick, Z. M. and Chapman, C. B.: On pericardiocentesis. Am. J. Cardiol. 16:722, 1965.

Pericardial fluid is usually drained by a percutaneous needle puncture (pericardicentesis). The immediate indication for this procedure is rapidly advancing cardiac tamponade as demonstrated by a shock-like syndrome, and a rising venous pressure above 130 mm H_2O. The safest and most successful approach is through the subxiphoid-subcortical area. Safeguards include electrocardiographic monitoring of the needle position and if blood is withdrawn to determine if it was drawn from the heart or pericardial cavity.

2. Surgical drainage and biopsy (pericardiotomy)

94. Loc cit
95. Barr, J. F.: The use of pericardial biopsy in establishing etiologic diagnosis in acute pericarditis. Arch. Int. Med. 96:693, 1955.
96. Weinberg, M., Fell, E. H. and Lynfield, J.: Diagnostic biopsy of the pericardium and myocardium. Arch. Surg. 76:825, 1958.
97. Schwartz, M. J., Nay, H. R. and Fitzpatrick, H. F.: Pericardial biopsy. Arch. Int. Med. 112:917, 1963.
98. Webb, W. and Sugg, W. L.: Personal communications.

It has been proposed by Kilpatrick and Chapman that the choice between pericardicentesis and pericardiotomy is as follows.

> "In cases where the minutes count and time is of the essence, indirect pericardial puncture is indicated; in cases where the primary question is one of etiologic diagnosis and there is no immediate threat to life, a direct approach for the purpose of obtaining a biopsy specimen as well as a sample of fluid is preferable."

Webb and Sugg have recently performed 17 pericardicenteses under local anesthesia at Parkland Memorial Hospital. In this group there were no deaths. One patient had a septic wound which drained for seven weeks, 2 patients had a pneumothorax, and 2 patients had a pleural effusion. A definitive tissue diagnosis was made in 9 of the cases and a diagnosis of nonspecific pericarditis was made in 8.

Fig 1

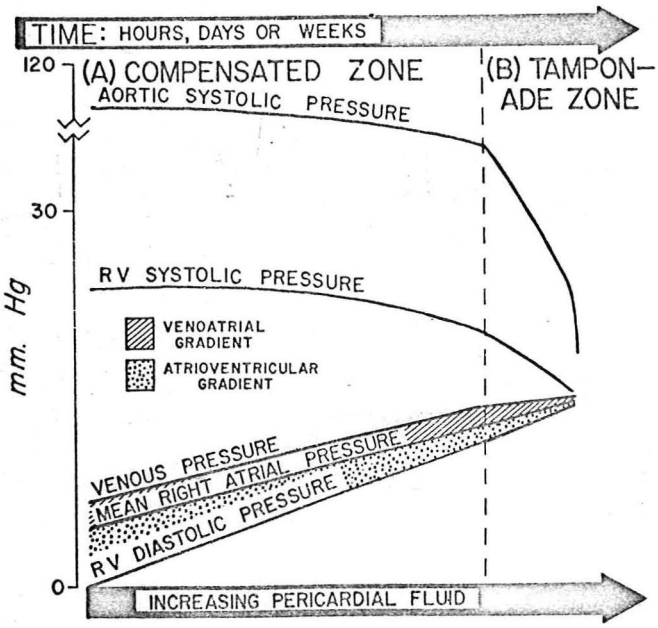

Fig 2

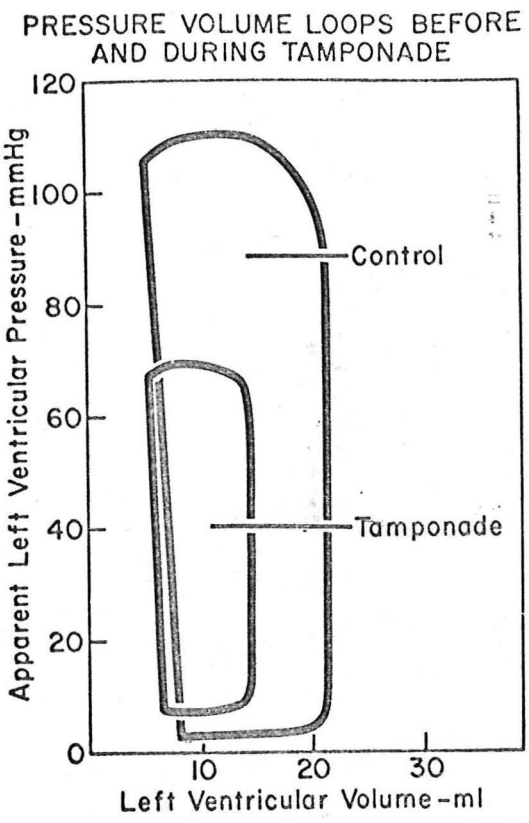

Fig 3

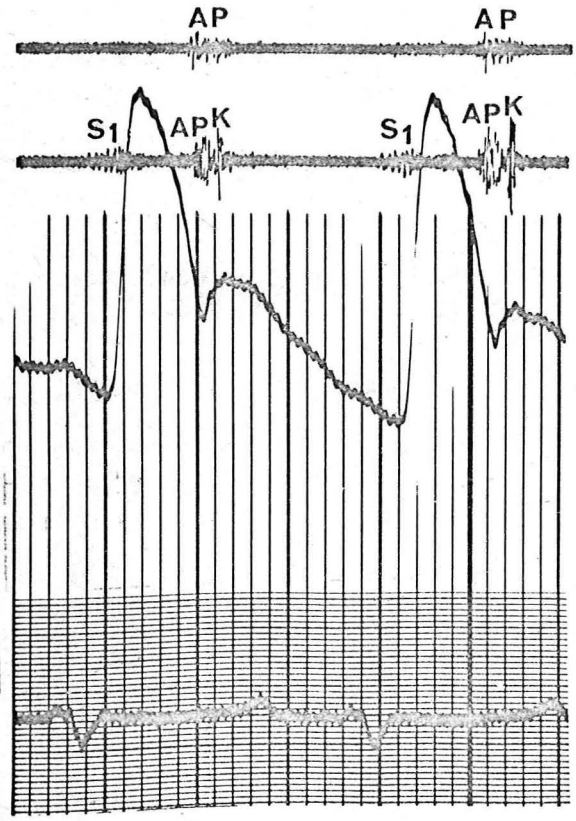

Fig 4