MYOCARDIAL INFARCTION:

Complications and Current Management

PARKLAND MEMORIAL HOSPITAL
MEDICAL GRAND ROUNDS

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MYOCARDIAL INFARCTION:
Complications and Current Management

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I THE CORONARY CARE UNIT

500,000 persons in the United States will die this year of an acute myocardial infarction[1]. Greater than one-half of them will die before reaching the hospital. For patients reaching the hospital, some form of Coronary Care Unit may be available. These units had their inception in 1962[2-4].

1. Cardiovascular Disease in the United States. Facts and Figures. American Heart Association, New York, 1965.

2. Day, H.W.: An Intensive Coronary Care Area. Dis. Chest, 44:423, 1963.

3. Brown, K.W., MacMillan, R.L., Forbath, N., Mel'Grano, F., and Scott, J.W.: Coronary Unit: An intensive - care center for acute myocardial infarction. Lancet, 2:349, 1963.

4. Meltzer, L.: Concepts and Systems for Intensive Coronary Care. Acad. Med., New Jersey Bull., 10:304, 1964.

It was anticipated that the Coronary Care Unit would make major inroads into the mortality rate of patients admitted to the hospital. A summary of mortality of patients hospitalized over the past 25 years with a myocardial infarction revealed an average mortality of 33.1%[5].

TABLE I

MORTALITY IN ACUTE MYOCARDIAL INFARCTION
(Pre-Coronary Care Unit)

SERIES (REF.NO.)	DATE	TOTAL CASES	PER CENT MORTALITY
6	1937	300	29
7	1941	208	33
8	1942	128	47
9	1945	1247	52
10	1947	572	22
11	1950	127	20
12	1954	461	23
13	1954	1318	34
14	1955	397	26
15	1957	1530	33
16	1957	543	36
17	1958	187	24
18	1960	153	35
19	1961	44	27
20	1962	331	15
21	1962	250	52
22	1962	520	34
23	1963	667	33
24	1964	30	27
25	1964	100	31
26	1964	500	24
27	1964	63	30
28	1964	1331	30
TOTAL		11,007	33.1%

5. Stephen, S.A.: Discussion of Presentation by Snow, P.J.D.: Treatment of Acute Myocardial Infarction with Propranolol. Am. J. Cardiol., 18:460, 1966.

6. Master, A.M., Dack, S., and Jaffe, H.L.: Disturbances of Rate and Rhythm in Acute Coronary Artery Thrombosis. Ann. Int. Med., 11:735, 1938.

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11. Goldman, M.J.: Cardiac Arrhythmias in Myocardial Infarction. Am. Heart J., 39:884, 1950.

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13. Russek, H.I., and Zohman, B.L.: Chances for Survival in Acute Myocardial Infarction. J.A.M.A., 156:765, 1954.

14. Ball, C.O.T. et al: The Functional Circulatory Consequences of Myocardial Infarction. Circulation, 11:749, 1955.

15. Biorck, G., Blomqvist, G., and Sievers, K.: Studies on Myocardial Infarction in Malmo 1935 to 1954. 1. Morbidity and mortality in a Hospital Material. Acta med. scandinav., 159:253, 1957.

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17. Johnson, C.C., and Miner, P.F.: The Occurrence of Arrhythmias in Acute Myocardial Infarctions. Dis. Chest, 33:414, 1958.

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19. Begg, T.B.: Prophylactic Quinidine after Myocardial Infarction. Brit. Heart J., 23:415, 1961.

20. Dreifus, L.S., Oslick, T., and Likoff, W.: Cardiac Arrhythmias: Therapy following acute myocardial infarction. Geriatrics, 17:569, 1962.

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23. Wahlbert, F.: A Study of Acute Myocardial Infarction at the Seraphimer Hospital during 1950-1959. Am. Heart J., 65:749, 1963.

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27. Robinson, J.S., Sloman, G., and McRae, C.: Continuous Electrocardiographic Monitoring in the Early Stages After Acute Myocardial Infarction. M.J. Australia, 1:427, 1964.

28. Pell, S., and D'Alonzo, C.A.: Immediate Mortality and Five Year Survival of Employed Men with a First Myocardial Infarction. New England J. Med., 270:915, 1964.

Killip[29] compared the results in 100 patients admitted to the Coronary Care Unit of New York Hospital with those of 100 patients admitted to regular care during the same period. The two groups were felt to be reasonably comparable.

1. Age distribution was similar.
2. Sex ratio were identical.
3. The incidence of shock was the same.
4. Admission to CCU was on bed - availability basis, not on severity of illness.

Results of study: Mortality

Coronary Care Unit - 32%
Regular Care - 30%

29. Killip. T., and Kimball, J.T.: Treatment of Myocardial Infarction in a Coronary Care Unit: A two year experience with 250 patients. Am. J. Cardiol., 20:457, 1967.

Certain changes were instituted:

1. Immediate defibrillation by nurse.
2. Specific indications for antiarrhythmic drugs.
3. Periodic training sessions.

In the next 150 patients admitted to the CCU, the mortality dropped to 24%. There was no change in the incidence of patients dying in shock, but there was a decrease in the number of patients dying of ventricular arrhythmias.

The results of a USPHS study substantiate the fact that the CCU has decreased the mortality rate in patients who would otherwise have succumbed to ventricular arrhythmias[30].

TABLE II

USPHS COOPERATIVE STUDY OF CCU
(10 HOSPITALS)

NO. PATIENTS	2842
MORTALITY	710 (25%)
PATIENTS DEVELOPING V.F.	284
NO. SUCCESSFUL RESUSCITATION	131 (46%)

30. Killip, T., and Kimball, J.T.: A Survey of the Coronary Care Unit, Concept and Results. Progress in Cardiovascular Disease, 11:45, 1968.

In two series in which mortality rates were analysed according to severity of infarction, the following data were obtained[30,31].

TABLE III

MORTALITY IN MYOCARDIAL INFARCTION

SERIES	MILD INFARCTION (NO FAILURE OR HYPOTENSION)	SEVERE INFARCTION (FAILURE OR HYPOTENSION)	CARDIOGENIC SHOCK
Killip[30]	6%	21%	81%
Lawrie[31]	4%	15%	83%

31. Lawrie, D.M., Greenwood, T.W., Goddard, M., Harvey, A.C., Donald,K.W., Julian, D.G., and Oliver, M.F.: A Coronary Care Unit in the Routine Management of Myocardial Infarction. Lancet, 2:110, 1967.

Two conclusions concerning Coronary Care Units may be drawn.

1. Inroads have been made in the therapy of ventricular arrhythmias following myocardial infarction.
2. No gains have been made in the salvage of the patient with cardiogenic shock.

II ARRHYTHMIAS

Incidence:

TABLE IV

INCIDENCE OF ARRHYTHMIAS IN MYOCARDIAL INFARCTION

	DAY[2]	BROWN[3]	JULIAN[25]	MELTZER[32]	LOWN[33]
NO. PATIENTS	119	100	100	141	130
ANY ARRHYTHMIAS	75%	61%	95%	82%	88%
VPC's	41%	40%	67%	45%	71%
VENTRICULAR TACHYCARDIA	27%	11%	6%	14%	29%
VENTRICULAR FIBRILLATION	8%	----	10%	10%	0

32. Meltzer, L.E., and Kitchell, J.B.: The Incidence of Arrhythmias Associated With Acute Myocardial Infarction. Progress in Cardiovascular Diseases, 9:50, 1966.

33. Lown, B., Fakhro, A.M., Hood, W.B. Jr., and Thorn, G.W.: The Coronary Care Unit: New perspectives and directions. J.A.M.A., 199:188, 1967.

Lown has shifted his emphasis from resuscitation and therapy of cardiac arrest to the identification and prompt treatment of so - called minor rhythm disorders which may lead to electrical disorganization of the heart beat. Lidocaine is used if ventricular premature contractions:

1. Occur early in the diastolic period near the T wave.
2. Occur in salvos of two or more.
3. Have a multiform configuration.
4. Occur at a frequency greater than five per minute.

A. ECTOPIC

1. Lidocaine:

The electrophysiological action on cardiac muscle is to elevate the diastolic threshold to stimulation without affecting the absolute refractory period[36].

The dosage schedule employed by Kimball and Killip for the control of ventricular premature contractions is[43]

1. One mg. per kilogram intravenously over a one minute period. If after three minutes the arrhythmia is not controlled, then
2. Additional one mg. per kilogram doses every 3 to 5 minutes until the ventricular premature contractions disappear or a total dose of 5 mg. per kilogram is given over a 20 minute period.
3. Maintenance - one mg. per minute by intravenous drip.

34. Carden, N.L., and Steinhaus, J.E.: Lidocaine in Cardiac Resuscitation from Ventricular Fibrillation. Circulation Research, 4;680, 1956.

35. Weiss, W.A.: Intravenous Use of Lidocaine for Ventricular Arrhythmias. Anesth. Analg. (Cleveland), 39:369, 1960.

36. Harrison, D.C., Sprouse, J.H., and Morrow, A.G.: Antiarrhythmic Properties of Lidocaine and Procainamide. Circulation, 28:486, 1963.

37. Gianelli, R., von der Groben, J.V., Spivack, A.P., and Harrison,D.C.: Effect of Lidocaine on Ventricular Arrhythmias in Patients with Coronary Heart Disease. New England J. Med., 277:1215, 1967.

38. Nelson, D.H., and Harrison, D.C.: A Comparison of the Negative Inotropic Effects of Procaine amide, lidocaine, and quinidine. Physiologist, 8:241, 1965.

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41. Schumacker, R.R., Lieberson, A.D., Childress, R.H., and Williams, J.F. Jr.: Hemodynamic Effects of Lidocaine in Patients with Heart Disease. Circulation, 37:965, 1968.

42. Lieberman, N.A., Harris, R.S., Katz, R.I., Lipschutz, H.M., Dolgin, M., and Fisher, V.J.: The Effects of Lidocaine on the Electrical and Mechanical Activity of the Heart. Am. J. Cardiol., 22:357, 1968.

43. Kimball, J.T., and Killip, T.: Aggressive Treatment of Arrhythmias in Acute Myocardial Infarction: Procedures and results. Progress in Cardiovascular Disease, 10:483, 1968.

2. Dilantin

Introduced in 1938[44]. Cardiovascular effects noted in 1939[45,46]. First reported laboratory study dealing with ventricular arrhythmias was 1950[47]. Since 1965, increased cardiologic interest in this agent[49-53].

a. Actions and Uses:

1. Locus of action is upon heart, not central nervous system.
2. Decreases ventricular automaticity.

3. Augments atrio-ventricular conduction; cf lidocaine. procaine amide, propranolol, potassium.
4. Because of #2 and #3, it has a special role in therapy of digitalis intoxication[55-62].
5. Causes only mild depression of left ventricular contractility[63-70].

b. Administration:

Dosage schedule recommended by Bigger et al[71]:

Day 1 - 1000 mg. in divided doses
Day 2 - 500 mg. in divided doses
Day 3 - 400 mg. in divided doses
Maintenance - 300-400 mg. in divided doses

This schedule produces plasma levels of between 15-20 microgram per cent.

c. Elimination:

1. Is parahydroxylated in the liver, and is excreted as a glucuronic acid conjugate[72].
2. Phenobarbital decreases dilantin's pharmacologic effect[73]. (Increases dilantin's parahydroxylation.)
3. Coumadin and INH augment dilantin's pharmacologic effect[73].

44. Merritt, H.H., and Putnam, T.J.: Sodium Diphenylhydantoinate in the Treatment of Convulsive Disorders. J.A.M.A., 111:1068, 1938.

45. Williams, D.: Treatment of Epilepsy with Sodium Diphenylhydantoinate Lancet, 2:678, 1939.

46. Finkleman, I., and Arieff, A.J.: Untoward Effects of Phenytoin Sodium in Epilepsy. J.A.M.A., 118:1209, 1942.

47. Harris, A.S., and Kokernot, R.H.: Effects of Diphenylhydantoin Sodium (Dilantin Sodium) and Phenobarbital Sodium Upon Ectopic Ventricular Tachycardia in Acute Myocardial Infarction. Am. J. Physiol., 163:505, 1950.

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49. Conn, R.D.: Diphenylhydantoin Sodium in Cardiac Arrhythmias. New Eng. J. Med., 272:277, 1965.

50. Bernstein, H., Gold, H., Lang, T.W., Pappelbaum, S., Bayika, V., and Corday, E.: Sodium Diphenylhydantoin in the Treatment of Recurrent Cardiac Arrhythmias. J.A.M.A., 191:695, 1965.

51. Parrow, A.: Use of Anticonvulsive Drugs in the Treatment of Recurrent Cardiac Arrhythmias. Acta. Med. Scand., 180:413, 1966.

52. Bashour, F.A., Jones, R.D., Edmondson, R.: Ventricular Tachycardia in Acute Myocardial Infarction: Preliminary report on the prophylactic use of Dilantin. Clin. Res., 13:399, 1966.

53. Rosen, M., Lisak, R., and Rubin, I.L.: Diphenylhydantoin in Cardiac Arrhythmias. Am. J. Cardiol., 20:674, 1967.

54. Hockman, C., Mauck, H.P., and Chu, N.: ECG Changes Resulting from Cerebral Stimulation. III Action of Diphenylhydantoin on Arrhythmias. Am. Heart J., 74:256, 1967.

55. Helfant, R.H., Scherlag, B.J., and Damato, A.N.: The Electrophysiological Properties of Diphenylhydantoin Sodium as Compared to Procaine Amide in the Normal and Digitalis-Intoxicated Heart. Circulation, 36:108, 1967.

56. Helfant, R.H., Scherlag, B.J., and Damato, A.N.: Protection from Digitalis Toxicity with the Prophylactic Use of Diphenylhydantoin Sodium. Circulation, 36:119, 1967.

57. Rosati, R.A., Alexander, J.A., Schall, S.F., and Wallace, A.G.: Influence of Diphenylhydantoin on Electrophysiological Properties of the Canine Heart. Circulation Research, 21:757, 1967.

58. Helfant, R.H., Lau, S.H., Cohen, S.I., and Damato, A.N.: Effects of Diphenylhydantoin on Atrioventricular Conduction in Man. Circulation, 36:686, 1967.

59. Helfant, R.H., Scherlag, B.J., and Damato, A.N.: Use of Diphenylhydantoin Sodium to Dissociate the Effects of Procaine Amide on Automaticity and Conduction in the Normal and Arrhythmic Heart. Am. J. Cardiol., 20:820, 1967.

60. Scherlag, B.J., Helfant, R.H., and Damato, A.N.: The Contrasting Effects of Diphenlyhydantoin and Procaine Amide on A-V Conduction in the Digitalis-Intoxicated and the Normal Heart. Am. Heart J., 75:200, 1968.

61. Helfant, R.H., Scherlag, B.J., and Damato, A.N.: Diphenylhydantoin Prevention of Arrhythmias in the Digitalis-Sensitized Dog After Direct-Current Cardioversion. Circulation, 37:424, 1968.

62. Helfant, R.H., Scherlag, B.J., and Damato, A.M.: Electrophysiological Effects of Direct Current Countershock Before and After Ouabain Sensitization and After Diphenylhydantoin Desensitization in the Dog. Circulation Research, 22:615, 1968.

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64. Vasko, J.S., Elkins, R.C., Fogarty, T.J., and Morrow, A.G.: Effects of Diphenylhydantoin on Cardiac Performance and Peripheral Vascular Resistance. Surgical Forum, 17:189, 1966.

65. Gupta, D.N., Unal, M.O., Bashour, F.A., and Webb, W.R.: Effects of Diphenylhydantoin (Dilantin) on Peripheral and Coronary Circulation and Myocardial Contractility in the Experimental Animal. Dis. Chest, 51:248, 1967.

66. Louis, S., Kutt, H., and McDowell, F.: The Cardiocirculatory Changes Caused by Intravenous Dilantin and its Solvent. Am. Heart J., 74:523, 1967.

67. Lieberson, A.D., Schumacher, R.R., Childress, R.H., Boyd, D.S., and Williams, J.F. Jr.: Effect of Diphenylhydantoin on Left Ventricular Function in Patients with Heart Disease. Circulation, 36:692, 1967.

68. Mierzwiak, D.S., Mitchell, J.H., and Shapiro, W.: The Effect of Diphenylhydantoin (Dilantin) and Quinidine on Left Ventricular Function in Dogs. Am. Heart J., 74:780, 1967.

69. Mierzwiak, D.S., Shapiro, W., McNalley, M.C., and Mitchell, J.H.: Cardiac Effects of Diphenylhydantoin (Dilantin) in Man. Am. J. Cardiol., 21:20, 1968.

70. Nayler, W.G., McInnes, I., Swann, J.B., Race, D., Carson, V., and Lowe, T.E.: Some Effects of Diphenylhydantoin and Propranolol on the Cardiovascular System. Am. Heart J., 75:83, 1968.

71. Bigger, J.T., Schmidt, D.H., and Kutt, H.: Relationship Between the Plasma Level of Diphenylhydantoin and its Cardiac Antiarrhythmic Effects. Circulation, 38:363, 1968.

72. Maynert, E.W.: Metabotic Fate of Diphenylhydantoin in the Dog, Rat, and Man. J. Pharmacol. Exp. Ther., 130:275, 1960.

73. Cucinell, S.A., Koster, R., Conney, A.H., and Burns, J.J.: Stimulatory Effect of Phenobarbital on the Metabolism of Diphenylhydantoin. J. Pharmacol. Exp. Ther., 141:157, 1963.

74. Mercer, E.N., and Osborne, J.A.: The Current Status of Diphenylhydantoin in Heart Disease. Ann. Int. Med., 67:1084, 1967.

75. Unger, A.H., and Sklaroff, H.J.: Fatalities Following Intravenous Use of Sodium Diphenylhydantoin for Cardiac Arrhythmias: A report of two cases. J.A.M.A., 200:159, 1967.

76. Gellerman, G.L., and Martinez, C.: Fatal Ventricular Fibrillation Following Intravenous Sodium Diphenylhydantoin Therapy, J.A.M.A., 200:161, 1967.

In summary, dilantin is effective in the therapy of ventricular arrhythmias and because of its lack of depression of atrio-ventricular conduction, is of particular value during the course of a myocardial infarction when first degree or second degree heart block may be present concomitant with ventricular ectopic rhythms.

B. BLOCK

THE COMPLICATIONS OF A MYOCARDIAL INFARCTION ARE, IN MANY CASES, PREDICTABLE AND ARE RELATED TO THE VESSEL INVOLVED[77,78].

1. In 55% of individuals, the sinus node artery arises from the right coronary artery.
2. In 90% of individuals, the atrio-ventricular nodal artery arises from the right coronary artery.

77. James, T.N.: Arrhythmias and Conductinn Disturbances in Acute Myocardial Infarction. Am. Heart J., 64:416, 1962.

78. James, T.N.: The Coronary Circulation and Conductinn System in Acute Myocardial Infarction. Prog. in Cardiovascular Diseases, 10:410, 1968.

1. Bradycardia - Rate less than 50 is ominous[79-80].

   a. Factors leading to bradycardia:

      1. Ischemia and products of ischemia[81-85].
      2. Pericarditis[86].
      3. von Bezold-Jarisch reflex[87].

79. Haden, R.F., Langsjoen, P.H., Rapoport, M.I., and McNerney, J.J.: The Significance of Sinus Bradycardia in Acute Myocardial Infarction. Dis. Chest, 44:168, 1963.

80. Thomas, M., and Woodgate, D.: Effect of Atropine on Bradycardia and Hypotension in Acute Myocardial Infarction. Brit. Heart J., 28:409, 1966.

81. James, T.N.; Myocardial Infarction and Atrial Arrhythmias. Circulation, 24:761, 1961.

82. Hoffman, B.F., and Cranefield, P.F.: Electrophysiology of the Heart. New York, McGraw-Hill Book Co., 1960.

83. James, T.N.: The Chronotropic Action of ATP and Related Compounds Studied by Direct Perfusion of the Sinus Node. J. Pharmacol. Exp. Ther. 149:2331, 1965.

84. Imai, S., Riley, A.L., and Berne, R.M.: Effect of Ischemia on Adenine Nucleotides in Cardiac and Skeletal Muscle. Circulation Research, 15:443, 1964.

85. Sherf, L., and James, T.N.: Chronotropic Action of Amino Acids. J. Pharmacol. Exp. Ther., 153:197, 1966.

86. James, T.N.: Pericarditis and the Sinus Node. Arch. Int. Med., 110:305, 1962.

87. Juhasz-Nagy, A., and Szentivanyi, M.: Localization of the Receptors of the Coronary Chemoreflex in the Dog. Arch. Int. Pharmacodyn., 131:39, 1961.

b. Consequences of Bradycardia:

1. Fall in cardiac output.
2. Predisposition to escape rhythms[88,89].

88. Han, J., Millet, D., Chizzonitti, B., and Moe, G.K.: Temporal Dispersion of Recovery of Excitability in Atrium and Ventricle as a Function of Heart Rate. Am. Heart J., 71:481, 1966.

89. Han, J., DeTraglia, J., Millet, D., and Moe, G.K.: Incidence of Ectopic Beats as a Function of Basic Rate in the Ventricle. Am. Heart J., 72:632, 1966.

2. Atrio - Ventricular Block

TABLE V

MORTALITY IN HEART BLOCK IN MYOCARDIAL INFARCTION[90]
(9 Series Compilation) 1938-1963

Pre-Pacemaking Treatment

NO. CASES	NO. DEATHS
173	93 (54%)

90. Gregory, J.J., and Grace, W.F.: The Management of Sinus Bradycardia, Nodal Rhythm, and Heart Block for the Prevention of Cardiac Arrest in Acute Myocardial Infarction. Progress in Cardio. Dis., 10:505, 1968.

TABLE VI

MORTALITY IN CATHETER PACEMAKER TREATMENT
IN HEART BLOCK IN MYOCARDIAL INFARCTION

SERIES-REF.NO.	NO. CASES	NO DEATHS
91	3	3
92	1	1
93	4	2
94	1	1
95	2	1
96	23	7
97	2	1
98	43	19
99	9	7
100	11	7
101	20	11
102	4	3
	123	63 (51%)

91. DeSanctis, R.W.: Short Term Use of Intravenous Electrode in Heart Block. J.A.M.A., 184:544, 1963.

92. Zucker, I.R., Parsonet, V., Gilbert, L., and Asa, M.: Dipolar Electrode in Heart Block. J.A.M.A., 184:549, 1963.

93. Samet, P., Jacobs, W., and Bernstein, W.H.: Electrode Catheter Pacemaker in the Treatment of Complete Heart Block in the Presence of Acute Myocardial Infarction. Am. J. Cardiol., 11:379, 1963.

94. Delman, A.J., Schwedel, J.B., and Escher, D.J.: An Intracardiac Pacemaker in Adams-Stokes Attacks in Acute Myocardial Infarction. J.A.M.A., 184:1040, 1963.

95. Levy, L., and Albert, H.M.: Therapy of Complete Heart Block Complicating Recent Myocardial Infarction. J.A.M.A., 187:617, 1964.

96. Bruce, R.A., Blackman, J.R., Cobb, L.A., and Dodge, H.T.: Treatment of Asystole or Heart Block During Acute Myocardial Infarction with Electrode Catheter Pacing. Am. Heart J., 69:460, 1965.

97. Winters, W.L., Jr., Tyson, R.R., and Soloff, L.A.: Cardiac Pacing: I. Clinical experience. Ann. Int. Med., 62:208, 1965.

98. Paulk, E.A., and Hurst, J.W.: Complete Heart Block in Acute Myocardial Infarction. A Clinical Evaluation of the Intracardiac Bipolar Catheter Pacemaker. Am. J. Cardiol., 17:695, 1966.

99. Epstein, E.F., Coulshed, C.S., McKendrick, C.J.,and Kearns, W.E.: Artificial Pacing by Electrode Catheter for Heart Block or Asystole Complicating Myocardial Infarction. Brit. Heart J., 28:546, 1966.

100. Harris, A., and Bluestone, R.: Treatment of Slow Rates Following Acute Myocardial Infarction. Brit. Heart J., 28:631, 1966.

101. Bilitch, M., Lau, F.Y.K., Cafferky, E.A., and Cosby, R.S.: Importance of Sino-Atrial Activity in Pacing With Acute Myocardial Infarction and A-V Block. Circulation, 34:III-56, 1966, Abstr.

102. Friedberg, C.K., Cohen, H., and Donoso, E.: Advanced Heart Block as a Complication of Acute Myocardial Infarction. Role of Pacemaker Therapy. Prog. in Cardiov. Dis., 10:466, 1968.

The data strongly suggest that the mortality of complete heart block associated with myocardial infarction does NOT seem to have been changed by the institution of pacemaking. Pre-existing and co-existing conditions often dictate the outcome of a patient with complete heart block. Congestive heart failure or shock at the time of insititution of pacing are associated with a 90% mortality(98).

I THE CORONARY CARE UNIT
II ARRHYTHMIAS
III CARDIOGENIC SHOCK
IV DISORDERS OF PAPILLARY MUSCLE
V VENTRICULAR SEPTAL DEFECT
VI VENTRICULAR ANEURYSM
VII CONGESTIVE HEART FAILURE

III CARDIOGENIC SHOCK

A. Definition:

The syndrome characterized by a persistent decline in arterial pressure associated with other signs of shock, such as alterations in the sensorium, cold clammy skin, tachycardia, and anemia or marked oliguria(103-106).

103. Heyer, H.E.: A Clinical Study of Shock During Acute Myocardial Infarction. Am. Heart J., 62:436, 1961.

104. Braunwald, E.: The Pathogenesis and Treatment of Shock in Myocardial Infarction. The Johns Hopkins Medical Journal, 121:421, 1967.

105. Braunwald, E.: Acute Myocardial Infarction with Shock. Circulation, Supplement Symposium on Coronary Heart Disease (Second Edition), 1968.

106. Weil, M.H., and Shubin, H.: Shock Following Acute Myocardial Infarction. Prog. in Cardiov. Dis., 11:1, 1968.

Cardiogenic shock is associated more frequently with occlusions involving the left coronary artery, as this artery supplies the greatest portion of the left ventricle.

TABLE VII

IMPORTANT PHYSIOLOGIC CHANGES IN CORONARY SHOCK

ART. PRESS. ↓↓	VENOUS PRESS. = or ↑
CARDIAC OUTPUT ↓	ART. pO_2 ↓
STROKE VOLUME ↓↓↓	PH ↓
SYSTEM. VASC. RESIST. ↑ or =	

The hallmark of myocardial infarction and shock is severe left ventricular failure. This is present even if the clinical signs of congestive heart failure are absent.

1. Digitalis

Controversy surrounds the use of digitalis in cardiogenic shock. Support for its use comes from studies demonstrating its powerful inotropic effects after coronary embolization[107,108].

107. Cronin, R.F.P., and Zsoter, T.: Hemodynamic Effect of Rapid Digitalizatinn in Experimental Cardiogenic Shock. Am. Heart J., 69:233, 1965.

108. Marano, A.J., Kline, H.J, Castero, J., and Kuhn, L.A.: Hemodynamic Effects of Ouabain in Experimental Acute Myocardial Infarction with Shock. Am. J. Cardiol., 17:327, 1966.

Other investigators feel that the dangers of arrhythmias due to digitalis overshadow the possible advantages[109,110].

109. Selzer, A.: The Use of Digitalis in Acute Myocardial Infarction. Prog. in Cardiov. Dis., 10:518, 1968.

110. Morris, J.J., Taft, C.V., Whalen, R.E., and McIntosh, H.D.: Digitalis Intoxication in Experimental Myocardial Infarction. Clin. Research, 15:216, 1967 (abstr.).

2. Isoproterenol (Prototype Beta-Adrenergic Stimulator)

a. Increases cardiac output.
b. Decreases peripheral resistance.
c. Dilates coronary vascular bed.
d. Increases myocardial oxygen requirements by increasing velocity of contraction.

111. Krasnow, N., Rolett, E.L., Yurchak, P.M., Hood, W.B. Jr., and Gorlin, R.: Isoproterenol and Cardiovascular Performance. Am. J. Med., 37:514, 1964.

112. Klocke, F.J., Kaiser, F.A., Ross, J. Jr., and Braunwald, E.: Mechanism of Increased Myocardial Oxygen Uptake Produced by Catecholamines. Am. J. Physiol., 209:913, 1965.

113. Eichna, L.W.: The Treatment of Cardiogenic Shock. Am. Heart J., 74:848, 1967.

114. Fearon, R.E.: Comparison of Norepinephrine and Isoproterenol in Experimental Coronary Shock. Am. Heart J., 75:634, 1968.

3. Glucagon

Glucagon was isolated in 1923 from crude extracts of pancreas. The similarities in action between epiniphrine and glucagon led to an investigation into the cardiac actions of glucagon (118-123).

115. Unger, R.H., and Eisentraut, A.M.: Glucagon. In, Hormones in the Blood, 2nd Edition, Academic Press, Londnn and New York.

116. Sutherland, E.W., and Cori, C.F.: Influence of Insulin Preparations on Glycogenolysis in Liver Slices. J. Biol. Chem., 172:737, 1948.

117. Sutherland, E.W., Robinson, G.A., and Butcher, R.W.: Some Aspects of the Biological Role of Adenosene. 3',5'-Mono-Phosphate (Cyclic AMP), Circulation, 37:279, 1968.

118. Farah, A., and Tuttle, R.: Studies on the Pharmacology of Glucagon. J. Pharmacol. Exp. Ther., 129:49, 1960.

119. Regan, T.J., Lehan, P.H., Henneman, D.H., Behar, A., and Hellems, H.K.: Myocardial Metabolic and Contractile Response to Glucagon and Epinephrine. J. Lab. Clin. Med., 63:638, 1964.

120. Whitehouse, F.W., and James, T.N.: Chronotropic Action of Glucagon on the Sinus Node. Proc. Soc. Exptl. Biol. Med., 122:823, 1966.

121. Lucchesi, B.R.: Cardiac Actions of Glucagon. Circulation Research, 22:777, 1968.

122. Glick, G., Parmley, W.W., Wechsler, A.S., and Sonnenblick, E.H.: Glucagon. Circulation Research, 22:789, 1968.

123. Parmley, W.H., Glick, G., and Sonnenblick, E.H.: Cardiovascular Effects of Glucagon in Man. New Eng. J. Med., 279:12, 1968.

The mechanism by which glucagon exerts a positive inotropic action upon the heart is still not known. In spite of many similarities to the action of epinephrine, beta-adrenergic blockade does not block the cardiac actions of glucagon. It is likely that the final mediating factor affecting the contractile force of cardiac muscle is calcium[124] and glucagon may act at the cellular level by potentiating the interactions of calcium, actin, and myosin.

124. Nayler, W.G.: Calcium Exchange in Cardiac Muscles: Basic mechanism of drug action . Am. Heart J., 73:379, 1967.

4. Post Extra Systolic Potentiation: Paired Pacing

"The interval between a contraction of the heart and the preceding beat is of such importance for the strength of the contraction that a study of this effect is a prime necessity."

Bowditch 1871[125]

"Although paired electrical stimulation has profound effects on the electrical, mechanical, and metabolic properties of the myocardium, the most propitious manner in which this technic may be used clinically has not yet been defined. Nonetheless, important steps will have been taken if the physiologist solves the riddle of the mechanism of post extra systolic potentiation and the clinical investigator finds a practical way of utilizing the profound changes induced by paired electrical stimulation in the treatment of disturbances of cardiac contraction or rhythms[126]".

125. Bowditch, H.P.: (as quoted by Braunwald et al, Ref. 126)

126. Braunwald, E., Sonnenblick, E.H., Ross, J. Jr., and Frommer, P.L.: Paired Electrical Stimulation of the Heart. A Physiologic Riddle and a Clinical Challenge. Circulation, 32:677, 1965.

a. Definition:

The beat following a ventricular premature contraction will be stronger than the beat which precedes it. The earlier the premature beat, the greater the strength of the following contraction. The augmentation of contraction of the post PVC beat can be separated from the augmentation due to increased filling of the ventricle[127-132].

127. Braunwald, N.S., Gay, W.A. Jr., Morrow, A.G., and Braunwald, E.: Sustained, Paired Electrical Stimuli. Am. J. Cardio., 14:385, 1964.

128. Cranefield, P.F., Scherlag, B.J., Yeh, B.K., and Hoffman, B.F.: Treatment of Acute Cardiac Failure by Maintained Postextrasystolic Potentiation. Bulletin of the New York Academy of Medicine, 40:903, 1964.

129. Braunwald, E., Ross, J. Jr., Frommer, P.L., Williams, J.F. Jr., Sonnenblick, E.H., and Gault, J.H.: Clinical Observations on Paired Electrical Stimulation of the Heart. Am. J. Med., 37:700, 1964.

130. Cranefield, P.F., and Hoffman, B.F.: The Physiologic Basis and Clinical Implications of Paired Pulse Stimulation of the Heart. Dis. Chest, 49:561, 1966.

131. Resnekov, L., Sowton, E., Lord, P., and Norman, J.: Haemodynamic and Clinical Effects of Paired Stimulation of the Heart. Brit. Heart J., 28:622, 1966.

132. Frommer, P.L., Robinson, B.F., and Braunwald, E.: Paired Electrical Stimulation. Am. J. Cardiol., 18:738, 1966.

Although of great theoretical interest, paired pacing has not to date been an effective mode of therapy in clinical situations of left ventricular failure.

5. Assisted Circulation

133. Sugg, W.L., Webb, W.R., and Cook, W.A.: Collective Review: Assisted Circulation. The Annals of Thoracic Surgery, 3:247, 1967.

IV DISORDERS OF PAPILLARY MUSCLE

A. Rupture

1. Death usually occurs within 24 hours[134].
2. Incidence in myocardial infarction approaches 1%[135].
3. Posterior papillary muscle ruptures more frequently than anterior muscle.
4. The first successful mitral valve replacement was in 1965[139], and the first series reported was in 1967[140].

134. Sanders, R.J., Neuberger, K.T., and Ravin, A.: Rupture of Papillary Muscles: Occurrence of rupture of the posterior muscle in posterior myocardial infarction. Dis. Chest, 31:316, 1957.

135. Cederquist, L., and Soderstrom, J.: Papillary Muscle Rupture in Myocardial Infarction. Acta Med. Scandinavica, 176:287, 1964.

136. Adicoff, A., Alexander, C.S., Ferguson, J.H., and Kelly, W.D.: Surgical Repair of Ruptured Papillary Muscle Complicating Posterior Myocardial Infarction. Am. J. Cardiol., 11:246, 1963.

137. Holloway, D.H. Jr., Whalen, R.E., and McIntosh, H.D.: Systolic Murmurs Developing After Myocardial Ischemia or Infarction. J.A.M.A., 191:888, 1965.

138. Horlick, L., Merriman, J.E., and Robinson, C.L.N.: A Case of Mitral Insufficiency Following Myocardial Infarction with Rupture of a Papillary Muscle. Canad. Med. Assoc. J., 94:192, 1966.

139. Austen, W.G., Sanders, C.A., Averell, J.H., and Friedlich, A.L.: Ruptured Papillary Muscle. Circulation, 32:597, 1965.

140. Cohen, L.S., Morrow, A.G., Braunwald, N.S., Roberts, W.C., and Braunwald, E.: Severe Mitral Regurgitation Following Acute Myocardial Infarctrion and Ruptured Papillary Muscle. Circulation, 36:87, 1967.

141. Cohen, L.S., Mason, D.T., and Braunwald, E.: Significance of an Atrial Gallop Sound in Mitral Regurgitation. Circulation, 35:112, 1967.

B. Dysfunction

The anatomic integrity of the papillary muscles is maintained, but the papillary muscles cannot maintain the requisite tension during systole. The mitral leaflets, therefore, prolapse, causing regurgitation[142-144]. This complication may occur in as many as 20% of patients with myocardial infarction[145].

142. Phillips, J.H., Burch, G.E., and DePasquale, N.P.: The Syndrome of Papillary Muscle Dysfunction. Ann. Int. Med., 59:508, 1963.

143. Burch, G.E., DePasquale, N.P., and Phillips, J.H.: Clinical Manifestations of Papillary Muscle Dysfunction. Arch. Int. Med., 112:112, 1963.

144. Bashour, F.A.: Mitral Regurgitation Following Myocardial Infarction: The syndrome of papillary mitral regurgitation. Dis. Chest, 48:113, 1965.

145. Heikkila, J.: Mitral Incompetence Complicating Myocardial Infarction. Brit. Heart J., 29:162, 1967.

146. James, T.N.: Anatomy of the Coronary Arteries in Health and Disease. Circulation, 32:1020, 1965.

V VENTRICULAR SEPTAL DEFECT

Perforation of the interventricular septum is difficult to differentiate clinically from papillary muscle rupture or dysfunction. Cardiac catheterization demonstrating a left to right shunt at the ventricular level establishes the diagnosis.(147-149)

147. Harrison, R.J., Shillingford, J.P., Allen, G.T., and Teare, D.: Perforation of Interventricular Septum after Myocardial Infarction . Brit. Med. J., 1:1066, 1961.

148. Honey, M., Belcher, J.R., Hasan, M., and Gibbons, J.R.P.: Successful Early Repair of Acquired Ventricular Septal Defect after Myocardial Infarction. Brit. Heart J., 29:453, 1967.

149. Jeresaty, R.M., Landry, A.B. Jr., and Stansel, H.C. Jr.: Post-infarction Interventricular Septal Defects. Am. Heart J., 74:543, 1967.

VI VENTRICULAR ANEURYSM

There are many gradations of abnormal contractility, ranging from lack of motion to paradoxical motion(150,151). There is also an increasing awareness of the disturbances in

1. AKINESIS - total lack of motion of a portion of left ventricular wall.

2. DYSKINESIS - paradoxical systolic expansion of part of the wall.
3. ASYNERESIS - diminished or inadequate motion of part of the wall.
4. ASYNCHRONY - disturbed temporal sequence of contraction.

150. Herman, M.V., Heinle, R.A., Klein, M.D., and Gorlin, R.: Localized Disorders in Myocardial Contraction. New Eng. J. Med., 277:333, 1967.

151. Cohen, L.S., Elliott, W.C., Klein, M.D., and Gorlin, R.: Coronary Heart Disease: Clinical, cinearteriographic and metabolic correlations. Am. J. Cardiol., 17:153, 1966.

The incidence of true cardiac aneurysms secondary to myocardial infarction ranges from 5-20% (152-163).

152. Abrams, D.L., Edelist, E., Luria, M.M., and Miller, A.J.: Ventricular Aneurysm. A Reappraisal Based on a Study of 65 Consecutive Autopsied Cases. Circulation, 27:164, 1963.

153. Wartman, W.B., and Hellerstein, H.K.: The Incidence of Heart Disease in 2,000 Consecutive Autopsies. Ann. Int. Med., 28:41, 1948.

154. Schlichter, J., Hellerstein , H.K., and Katz, L.H.: Aneurysm of the Heart: A correlative study of 102 proven cases. Medicine, 33:43, 1954.

155. Dressler, W., and Pfeiffer, R.: Cardiac Aneurysm: A report of ten cases. Ann. Int. Med., 14:100, 1940.

156. Parkinson, J., Bedord, D.E., and Thomson, W.A.R.: Cardiac Aneurysm. Quart. J. Med., 7:455, 1938.

157. Scherf, D., and Brooks, A.M.: The Murmurs of Cardiac Aneurysm. Am. J. M. Sc., 218:389, 1949.

158. Libman, E., and Sacks, B.: A Case Illustrating the Leucocytosis of Progressive Myocardial Necrosis Following Coronary Artery Thrombosis. Am. Heart J., 2:321, 1927.

159. Vakil, R.J.: Ventricular Aneurysm of the Heart. Preliminary report on some new clinical signs. Am. Heart J., 49:934, 1955.

160. Gorlin , R., Klein, M.D., and Sullivan, J.M.: Prospective Correlative Study of Ventricular Aneurysm. Am. J. Med., 42:512, 1957.

161. Klein, M.D., Herman, M.V., and Gorlin, R.: A Hemodynamic Study of Left Ventricular Aneurysm. Circulation, 35:614, 1967.

162. Nordenfelt, O.: The Electrocardiogram in Chronic Aneurysm of the Heart. Acta Med. Scandinav., 102:101, 1939.

163. Fearon, R.E., Cohen, L.S., O'Hara, J.M., and Goodyer, A.V.N.: Diastolic Murmurs Due to Two Sequelae of Atherosclerotic Coronary Artery Disease: Ventricular aneurysm and coronary artery stenosis. Am. Heart J., 76:252, 1968.

Ventricular aneurysms decrease left ventricular contractility by:

1. Quantitative reduction of viable myocardium.
2. The aneurysm acts as a second chamber into which the ventricle ejects.

Aneurysmectomy has been performed as a therapeutic surgical approach to the patient with a ventricular aneurysm.

Recently, operation has been advocated for removal of infarcted areas of ventricle, even in the absence of aneurysm(164). The rationale behind this procedure is the thesis that discontinuity of circular muscle leads to impaired contractile effort of the ventricle. Infarctectomy should still be considered an experimental approach.

164. Heimbecker, R.O., Lemire, G., and Chen, C.: Surgery for Massive Myocardial Infarction. Experimental Study of Emergency Infarctectomy. Circulation, 37:3, 1968.

VII CONGESTIVE HEART FAILURE

The Case Report represents a major complication of myocardial infarction.

F.P., #344351

F.P., a 46 year old male, was in good health until July 4, 1968, when he had the onset of substernal chest pain. The pain was intermittently present for the next three days, and upon arriving at the Emergency Room, his electrocardiogram revealed the changes of an acute anterior wall myocardial infarction. He had no history of hypertension, diabetes, or familial heart disease.

On physical examination his blood pressure was 120/86, pulse 120, respirations 24. He had no arcus or xanthelasma. There were bilateral rales one half way up both lung fields. The carotids were thready and the jugular venous pressure was normal. The first and second heart sounds were normal. There was a diastolic filling gallop. There were no murmurs.

The electrocardiogram revealed a massive acute anterior wall myocardial infarction with complete loss of R waves in leads V1-V6. The CBC was normal. The serum glutamic oxaloacetic transaminases were 399, 569, 195, 112, 90, and 47 over a six day period. Because of persistent tachycardia and evidence of pulmonary congestion, digitalis was administered on the 5th hospital day. His hospital course was otherwise uncomplicated and he was discharged on the 21st hospital day.

Two weeks later, he was readmitted in severe congestive heart failure. He remained in the hospital for two weeks and responded to diuretics and a low salt diet. A Grade 2/6 holosystolic murmur at the cardiac apex was heard for the first time. He was discharged on August 28, 1968.

On September 5, 1968, he was readmitted in severe congestive heart failure. He was diuresed of eight kilograms of edema fluid, and had a cardiac catheterization on September 16, 1968. The cardiac catheterization revealed marked pulmonary hypertension, 65/30 mm Hg., a mean left atrial pressure of 35 mm Hg., a left ventricular pressure of 100/40 mm Hg., and a cardiac index of 1.6 L/min/M^2. A left ventricular angiogram revealed minor mitral regurgitation and a totally akinetic anterior left ventricular wall.

The etiology of his unremitting heart failure was felt to be loss of a major portion of his left ventricular myocardium. In spite of maximal medical therapy, he remained in severe congestive heart failure, and it was decided that a heart transplant was the only procedure that offered him a chance for survival. This was performed on October 2, 1968. He did well for five days, but sustained circulatory failure late in the fifth post-operative day and expired. The exact cause of death is not yet obvious, but preliminary evidence points toward a rejection phenomenon.

There is a continuing need for very strict criteria in the selection of appropriate patients for heart transplants. This procedure must still be considered experimental. Nevertheless, a patient with severe, unremitting congestive heart failure due to massive infarction of the left ventricle deserves consideration for cardiac transplantation if all other medical therapy has been exhausted.