ACUTE CORONARY OCCLUSION:

Hemodynamic Alterations and Possible Role of Assisted Circulation

Parkland Memorial Hospital

Medical Grand Rounds

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Case I [redacted] [redacted]

History: The patient was a 54-year-old [redacted] female who was admitted with the complaint of chest pain which occurred while riding on a bus. This pain was described as feeling like "a truck was sitting on my chest", and also it radiated into the left arm. The patient became nauseated and vomited. She was taken from the bus and brought to [redacted]. During the past 2 years the patient has noted chest pain which occurred while carrying groceries or getting excited and which could be relieved by rest in about 15 minutes. During the past year she has been treated for hypertension with reserpine. There was no history of dyspnea, orthopnea, swelling of lower extremities, or diabetes mellitus.

Physical examination: The patient was a moderately obese white female in acute distress with chest pain and intermittent bouts of vomiting. Pulse was 100 and blood pressure 160/100. The fundi were normal and no cervical venous distention was noted. The lungs were clear to percussion and auscultation. The heart was slightly enlarged and the rhythm was normal sinus. No murmurs or gallop rhythm were heard. Abdominal examination was negative, and there was no edema of the lower extremities.

Laboratory: On admission the chest films showed cardiomegaly and clear lung fields. The electrocardiogram revealed an acute anterior myocardial infarction. The circulation time was 15 seconds and the venous pressure 15.5 cm H_2O.

Diagnoses:
1) Arteriosclerotic heart disease with acute myocardial infarction
2) Probable essential hypertension

Hospital course: The chest pain was relieved by oxygen administration and morphine sulfate and the patient seemed to be getting along well. However, the blood pressure slowly fell from 160/100 to 100 systolic and aramine was started. Blood pressure continued to fall. Levophed was started and systolic pressure was brought to 130. Patient developed acute pulmonary edema and was digitalized. Blood pressure again fell despite increasing levophed, pulmonary edema became worse, and the patient expired 7 hours after admission.

Case II [redacted] [redacted]

History: The patient was a 72-year-old [redacted] female whose chief complaint was substernal chest pain for one day. She was apparently in good health until she awoke at 2 am on the morning prior to admission with severe substernal chest pain which radiated into both shoulders and upper arms. This pain remained severe for 4 to 5 hours and then gradually subsided leaving the patient quite weak. The patient was brought to [redacted] because of continued weakness and slight chest pain. There was no history of previous chest pain, dyspnea, orthopnea, edema, hypertension, or diabetes mellitus.

Physical examination: The patient was a weak [redacted] female in no acute distress. Pulse was 130 and blood pressure 70/50. The fundi were normal. Cervical veins were not distended. Fine crepitant rales were heard in bases of both lungs. The heart was slightly enlarged to percussion. Rhythm was normal sinus with runs of premature ventricular contractions. No murmurs were heard, but a loud scratchy to-and-fro friction rub was heard. Liver and spleen were not palpable and there was no edema of the lower extremities.

Laboratory: Chest films showed left ventricular enlargement and normal lung fields. Electrocardiogram revealed acute anterior myocardial infarction. Sed rate, white blood cell count, and SGOT were all elevated.

Diagnoses:
1) Arteriosclerotic heart disease
2) Acute myocardial infarction

Hospital course: Shortly after admission the patient developed hypotension 70/50 and runs of ventricular tachycardia. Arterial pressure was increased to 126/90 with aramine. Electrocardiogram revealed extension of the infarcted area. The patient developed orthopnea and cervical venous distention. She was digitalized. Patient improved and was able to maintain her arterial pressure. Her condition remained fairly stable for about 16 days. At that time she developed atrial flutter with a 2:1 to 4:1 AV block. Digitalis was discontinued for 24 hours and 80 mg of potassium were given. Heart rate increased and digitalis was reinstituted. The patient's rhythm returned to normal sinus. The patient again became hypotensive and aramine was started. Aramine dosage was increased, and arterial pressure continued to fall. She expired 18 days after admission.

I. Introduction

The care of patients with acute coronary occlusion is a problem of increasing importance. The salvage rate in the treatment of this condition has been greatly increased since the institution of intensive care units. This is principally due to better methods of recognition and treatment of acute arrhythmias. However, there are still cases in which the failure of the heart as a muscular pump continues to be a serious problem. The purpose of this grand rounds is to present the hemodynamic findings that occur in acute coronary occlusion, discuss how this condition may lead to progressive, unrelenting failure of the left ventricle as an effective pump, and finally to present a new approach to therapy which involves mechanical assistance to the circulation.

II. Hemodynamic Alterations During Acute Coronary Occlusion

A. Clinical studies of acute coronary occlusion

1. Hemodynamic data obtained during acute coronary occlusion

(1) Fishberg, A. M., Hitzig, W. M. and King, F. H.: Circulatory dynamics in myocardial infarction. Arch. Int. Med. 54:6, 1934.

In this study measurements were made in patients with acute coronary occlusion of arterial pressure, venous pressure and circulation time. It was suggested that when shock was present it was due to peripheral circulatory collapse. However, since cardiac output was not measured in these patients, it is difficult to accept their conclusions.

(2) Grishman, A. and Master, A. M.: Cardiac output in coronary occlusion studies by the Wezler-Boeger physical method. Proc. Soc. Exper. Biol. and Med. 48:207, 1941.

(3) Starr, I. and Wood, F. C.: Studies with the ballistocardiograph in acute cardiac infarction and chronic angina pectoris. Am. Heart J. 25:81, 1943.

These two early studies of patients during acute coronary occlusion both showed a subnormal cardiac output. However, one might question the reliability of the methods used.

(4) Freis, E. D., Schnaper, H. W., Johnson, R. L. and Schreiner, G. E.: Hemodynamic alterations in acute myocardial infarction. I. Cardiac output, mean arterial pressure, total peripheral resistance, "central" and total blood volumes, venous pressure and average circulation time. J. Clin. Invest. 31:131, 1952.

(5) Smith, W. W., Wikler, N. S. and Fox, A. C.: Hemodynamic studies of patients with myocardial infarction. Circulation 9:352, 1954.

(6) Gilbert, R. P., Goldberg, M. and Griffin, J.: Circulatory changes in acute myocardial infarction. Circulation 9:847, 1954.

(7) Gammill, J. F., Appelgarth, J. J., Reed, C. E., Fernald, J. D. and Antenucci, A. J.: Hemodynamic changes following acute myocardial infarction using the dye injection method for cardiac output determination. Ann. Int. Med. 43:100, 1955.

(8) Lee, G. de J.: Total and peripheral blood flow in acute myocardial infarction. Brit. Heart J. 19:117, 1957.

(9) Broch, O. J., Humerfelt, S., Haarstad, J. and Myhre, J. R.: Hemodynamic studies in acute myocardial infarction. Am. Heart J. 57:522, 1959.

(10) Murphy, G. W., Glick, G., Schreiner, B. F. and Yu, P. N.: Cardiac output in acute myocardial infarction. Am. J. Cardiol. 11:587, 1963.

(11) MacKenzie, G. J., Taylor, S. H., Flenley, D. C., McDonald, A. H., Staunton, H. P. and Donald, K. W.: Circulatory and respiratory studies in myocardial infarction and cardiogenic shock. Lancet 2:825, 1964.

(12) Malmcrona, R. and Varnauskas, E.: Haemodynamics in acute myocardial infarction. Acta med. scand. 175:1, 1964.

(13) Malmcrona, R.: Haemodynamics in myocardial infarction. Acta med. scand. Suppl. 417:1-54, 1964.

These groups have all measured cardiac output by the indicator dilution technique in patients with acute myocardial infarction. In general they found a normal or slightly decreased cardiac output and stroke volume in those patients who had normal arterial pressures and a decrease in cardiac output and stroke volume in those with decreased arterial pressure. Further, Gilbert et al. found that the lowering of the cardiac index was roughly proportional to the clinical severity of the attack.

(14) Gubner, R. and Crawford, J. H.: Roentgenkymographic studies of myocardial infarction. Am. Heart J. 18:8, 1939.

(15) Sussman, M. D., Dack, S. and Master, A. M.: The roentgenkymogram in myocardial infarction. I. The abnormalities in left ventricular contraction. Am. Heart J. 19:453, 1940.

(16) Master, A. M., Gubner, R., Dack, S. and Jaffe, H. L.: The diagnosis of coronary occlusion and myocardial infarction by fluoroscopic examination. Am. Heart J. 20:475, 1940.

These studies demonstrated the stretching of the ischemic area in acute coronary occlusion in man by fluoroscopic and roentgenkymographic methods. The importance of these findings will be evident later when we discuss ventricular dynamics during acute coronary occlusion.

2. Investigations concerning medical treatment of left ventricular failure in acute coronary occlusion

We will not attempt to discuss medical treatment of acute coronary occlusion in this grand rounds. As demonstrated by the two cases summarized in this protocol, heroic medical measures are at times not sufficient to restore an adequate circulatory state.

B. Experimental studies of acute coronary occlusion in animals

1. Hemodynamic data obtained during acute coronary occlusion

(17) Porter, W. T.: Further researches on the closure of the coronary arteries. J. Exp. Med. 1:46, 1896.

(18) Gross, L., Mendlowitz, M. and Schauer, G.: Hemodynamic studies in experimental coronary occlusion. I. Open chest experiments. Am. Heart J. 13:647, 1937.

(19) Mendlowitz, M., Schauer, G. and Gross, L.: Hemodynamic studies in experimental coronary occlusion. II. Closed chest experiments. Am. Heart J. 13:664, 1937.

(20) Gross, L., Schauer, G. and Mendlowitz, M.: Hemodynamic studies in experimental coronary occlusion. V. Changes in arterial blood pressure. Am. Heart J. 16:278, 1938.

(21) Kupfer, S.: Can experimental shock be induced by coronary occlusion? Proc. Soc. Exper. Biol. and Med. 76: 134, 1951.

(22) Selzer, A. and Taylor, G. W.: The hypotensive state following acute myocardial infarction. II. Experimental studies. Am. Heart J. 44:12, 1952.

(23) Wégria, R., Frank, C. W., Misrahy, G. A., Wang, H., Miller, R. and Case, R. B.: Immediate hemodynamic effects of acute coronary artery occlusion. Am. J. Physiol. 177:123, 1954.

(24) Bing, R. J., Castellanas, A., Gradel, E., Lupton, C. and Siegel, A.: Experimental myocardial infarction: circulatory, biochemical and pathologic changes. Am. J. M. Sc. 232:533, 1956.

(25) Agress, C. M., Glassner, H. F., Binder, M. J. and Fields, J.: Hemodynamic measurements in experimental coronary shock. J. Appl. Physiol. 10:469, 1957.

(26) Hammer, J. and Pisa, Z.: Haemodynamic changes after embolization of the coronary bed in closed chest dogs. Rev. Czechoslovak Med. 7:171, 1961.

(27) Rushmer, R. F., Watson, N., Harding, D. and Baker, D.: Effects of acute coronary occlusion on performance of right and left ventricles in intact unanesthetized dogs. Am. Heart J. 66:522, 1963.

In 1896 Porter clearly demonstrated that ligation of the left anterior descending coronary artery in open chest dogs can cause a fall in arterial pressure and a decrease in cardiac output. In similar experiments Gross, Mendlowitz and Schauer found a drop in cardiac output but little change in arterial pressure. Wégria et al. produced acute coronary occlusion and also found an immediate drop in cardiac output and a decrease in arterial blood pressure, both of which rose to or toward control levels in most experiments. In 1963 Rushmer and his group produced acute coronary occlusion in unanesthetized close chest dogs while measuring the performance of the left ventricle by quite accurate and dynamic techniques. The principal findings were a reduction in peak ejection velocity, peak flow acceleration, and stroke volume of the left ventricle.

2. The role of cardiac and peripheral circulatory factors in the shock of acute coronary occlusion

(1) Loc. cit.

(28) Boyer, N. H.: Cardiogenic shock. New England J. Med. 230:226, 1944.

(29) Agress, C. M. and Binder, M. J.: Cardiogenic shock. Am. Heart J. 54:458, 1957.

(30) Guzman, S. V., Swenson, E. and Mitchell, R.: Mechanism of cardiogenic shock. Circulation Res. 10:746, 1962.

(31) Levy, M. N. and Frankel, A. L.: Vasomotor responses to acute coronary occlusion in the dog. Am. J. Physiol. 172:427, 1963.

(32) Costantin, L.: Extracardiac factors contributing to hypotension during coronary occlusion. Am. J. Cardiol. 11: 205, 1963.

It has been argued by some that decreased arteriolar and venous tone are responsible for the shock of acute coronary occlusion. The most recent work on this point is that of Costantin. He demonstrated a reflex with probable stretch receptors in the ventricle which cause a withdrawal of sympathetic tone to both the heart and periphery. The quantative role of this reflex in acute coronary occlusion is not clear.

3. Evaluation of ventricular performance after decreased coronary flow or acute coronary occlusion

(33) Case, R. B., Berglund, E. and Sarnoff, S. J.: Ventricular function. II. Quantitative relationship between coronary flow and ventricular function with observations on unilateral failure. Circulation Res. 2:319, 1954.

(34) Stone, H. L., Bishop, V. S. and Guyton, A. C.: Cardiac function after embolization of coronaries with microspheres. Am. J. Physiol. 204:16, 1963.

Case, Berglund and Sarnoff demonstrated a decreased ventricular function curve when left coronary artery flow was decreased. This maneuver caused a generalized myocardial ischemia and is not comparable to acute coronary occlusion of one artery. Stone, Bishop and Guyton produced acute coronary occlusion by embolization with microspheres. The ventricular function curve was decreased by this intervention.

4. Ventricular dynamics after acute coronary occlusion

(35) Porter, W. T.: On the results of ligation of the coronary arteries. J. Physiol. 15:121, 1893.

(36) Orias, O.: The dynamic changes in the ventricles following ligation of the ramus descendens anterior. Am. J. Physiol. 100:629, 1932.

(37) Tennant, R. and Wiggers, C. J.: The effect of coronary occlusion on myocardial contraction. Am. J. Physiol. 112:351, 1935.

(38) Wiggers, C. J.: The functional consequences of coronary occlusion. Ann. Int. Med. 23:158, 1945.

(39) Wiggers, C. J.: Reminiscences and Adventures in Circulation Research. New York: Grune and Stratton, 1958, pp. 357-359.

(40) Prinzmetal, M., Schwartz, L. L., Corday, E., Spritzler, R., Bergman, H. C. and Kruger, H. E.: Studies on the coronary circulation. VI. Loss of myocardial contractility after coronary artery occlusion. Ann. Int. Med. 31: 429, 1949.

(41) Sayen, J. J., Sheldon, W. F., Peirce, G. and Kuo, P. T.: Polarographic oxygen, the epicardial electrocardiogram and muscle contraction in experimental acute regional ischemia of the left ventricle. Circulation Res. 6:779, 1958.

(42) Sayen, J. J., Peirce, G., Katcher, A. H. and Sheldon, W. F.: Correlation of intramyocardial electrocardiograms with polarographic oxygen and contractility in the nonischemic and regionally ischemic left ventricle. Circulation Res. 9:1268, 1961.

Tennant and Wiggers occluded the coronary artery and demonstrated that a marked systolic expansion of the ischemic area replaces shortening. This not only reduces the total myocardial force available for raising intraventricular pressure but some of this pressure is spent in stretching the ischemic area and is thus lost for expelling blood into the aorta. That such bulging does occur after acute coronary occlusion has also been demonstrated by Prinzmetal et al. and Sayen et al.

(42) Mullins, C. B., Payne, R. M., Harris, M. D. and Mitchell, J. H.: Left ventricular dynamics after acute coronary occlusion. In preparation.

Volume and dimension changes of the left ventricle of dogs have been examined by a biplane cinefluorographic technique before and after tying the left anterior descending coronary artery. The changes in contraction patterns have been demonstrated by this method.

5. Ventricular performance after artificially induced ventricular aneurysm

(44) Tyson, K., Mandelbaum, I. and Sh[illegible] H. B., Jr.: Experimental production and study of left ventricular aneurysms. J. Thor. and Cardiovas. Surg. 44:731, 1962.

In this study in dogs the effect of artificially constructed left ventricular aneurysms on left ventricular function was examined. It was demonstrated that patent, paradoxically pulsating aneurysm of the left ventricle depressed left ventricular function curves (decreased contractility of the left ventricle).

III. Assisted Circulation in Acute Coronary Occlusion

A. General

(45) Galletti, P. M. and Brecher, G. A.: Heart-Lung Bypass: Principles and Techniques of Extracorporeal Circulation. New York: Grune and Stratton, 1962.

(46) Galletti, P. M.: Physiological basis for assisted circulation. J. Mt. Sinai Hosp. 23:178, 1965.

(47) Schenk, W. G., Delin, N. A., Camp, F. A., McDonald, K. E., Pollock, L., Gage, A. A. and Chardack, W. M.: Assisted circulation. An experimental evaluation of counterpulsation and left ventricular bypass. Arch. Surg. 88:327, 1964.

The development of "assisted circulation" may play an important role in the future treatment of cases of acute coronary occlusion which do not respond to heroic medical measures. The aim of mechanical assistance to the left ventricle is to decrease its energy requirements (oxygen consumption) and to provide better perfusion pressure and flow to the vital organs, particularly the coronary arteries.

B. Physiological principles of assisted circulation

1. Left ventricular oxygen consumption

(46) Loc. cit.

(47) Loc. cit.

(48) Sarnoff, S. J., Braunwald, E., Welch, G. H., Jr., Case, R. B., Stainsby, W. N. and Macruz, R.: Hemodynamic determinants of oxygen consumption of the heart with special reference to the time-tension index. Am. J. Physiol. 192:148, 1958.

(49) Welch, G. H., Jr., Sarnoff, S. J., Braunwald, E., Stainsby, W. N., Case, R. B. and Macruz, R.: The influence of cardiac output, aortic pressure, and heart rate on myocardial oxygen utilization. Surg. Forum 8:294, 1958.

These authors show that increasing ventricular work by augmenting cardiac output caused only a slight rise in myocardial oxygen consumption, while increasing ventricular work by elevating aortic pressure caused a marked increase in oxygen consumption. The primary determinant was found to be the total tension developed by the myocardium as indicated by the area beneath the systolic pressure curve (time-tension index).

2. Dynamics of coronary flow

(50) Gregg, D. E.: The Coronary Circulation in Health and Disease. Philadelphia: Lea and Febiger, 1950.

(51) Menno, A. D. and Schenk, W. G., Jr.: Dynamics of coronary arterial flow. Surgery 50:82, 1961.

(52) Gregg, D. E.: Physiology of the coronary circulation. Circulation 27:1128, 1963.

The majority of coronary flow occurs during diastole. Recently it has been shown that some flow also occurs during systole, but the importance of this is still being debated.

C. Types of assisted circulation

(46) Loc. cit.

(47) Loc. cit.

A limitation inherent in all methods of assisted circulation is damage to the red cells produced by the mechanical pump which is much more marked when an oxygenator is used. For this reason greater interest has been shown for those methods which do not require oxygenation of the blood. For our purposes assisted circulation can be classified into the following types.

Types of Assisted Circulation

1. Veno-arterial pumping without oxygenation
2. Left heart bypass
   a. Total bypass
   b. Partial bypass without diastolic augmentation
   c. Partial bypass with diastolic augmentation
3. Synchronized arterial counterpulsation

1. Veno-arterial pumping without oxygenation

(53) Dickson, J. F., III, Hamer, N. A. J. and Dow, J. W.: Veno-arterial pumping for relief of intractable cardiac failure in man. Arch. Surg. 78:418, 1959.

(54) Hamer, N. A. J., Dickson, J. F. and Dow, J. W.: Effect of prolonged veno-arterial pumping on circulation of dog. J. Thor. Surg. 37:190, 1959.

(55) Wyman, M. G., Weil, M. H. and Blankenhorn, J. H.: Partial cardiopulmonary bypass utilizing selective veno-arterial perfusion. J. Clin. Invest. 38:1056, 1959.

Veno-arterial blood consists simply of removing blood from the great veins and pumping it into the arterial system. This method is easy to perform and has been used in both animal studies and in man.

2. Left heart bypass

(56) Dennis, C., Carlens, E., Senning, Å., Hall, D. P., Moreno, J. R., Cappelletti, R. R. and Wesolowski, S. A.: Clinical use of a cannula for left heart bypass without thoracotomy. Ann. Surg. 156:623, 1962.

(57) Dennis, C., Hall, D. P., Moreno, J. R. and Senning, Å.: Left atrial cannulation without thoracotomy for total left heart bypass. Acta chir. scand. 123:264, 1962.

(58) Hall, D. P., Moreno, J. R., Dennis, C. and Senning, Å.: Left heart bypass as a means of support for a failing heart. Bull. de la Société Internat. de Chirurgia 21: 607, 1962.

(59) Dennis, C., Hall, D., Moreno, J. R. and Senning, Å.: Reduction of oxygen utilization of the heart by left heart bypass. Circulation Res. 10:298, 1962.

(60) Hall, D. P., Moreno, J. R., Dennis, C. and Senning, Å.: Experimental study of prolonged left heart bypass without thoracotomy. Ann. Surg. 156:190, 1962.

In this method the left atrium is cannulated transseptally through the jugular vein in the neck. Oxygenated blood is removed from the left atrium and pumped into the arterial system. If the total cardiac output is diverted by this means, then one has a total bypass of the left ventricle. If only a portion of the cardiac output is diverted, then the bypass is only partial. If the return of oxygenated blood is not synchronized with cardiac activity, diastolic augmentation is not achieved. If, however, the return is synchronized with cardiac activity and blood is returned only during diastole, then diastolic augmentation is achieved.

3. Synchronized arterial counterpulsation

(61) Jacobey, J. A., Taylor, W. J., Smith, G. T., Gorlin, R. and Harken, D. E.: A new therapeutic approach to acute coronary occlusion. Surg. Forum 12:225, 1961.

(62) Clauss, R. H., Birtwell, W. C., Albertal, G., Lunzer, S., Taylor, W. J., Fosbog, A. M. and Harken, D. E.: Assisted circulation. I. The arterial counterpulsator. J. Thor. and Cardiovas. Surg. 41:447, 1961.

(63) Lefemine, A. A., Low, H. B. C., Cohen, M. L. and Harken, D. E.: Assisted circulation. II. The effect of heart rate on synchronized arterial counterpulsation. Am. Heart J. 64:779, 1962.

(64) Lefemine, A. A., Low, H. B. C., Cohen, M. L., Lunzer, S. and Harken, D. E.: Assisted circulation. III. Effect of synchronized counterpulsation on myocardial oxygen consumption and coronary flow. Am. Heart J. 64:789, 1962.

(65) Jacobey, J. A., Taylor, W. J., Smith, G. T., Gorlin, R. and Harken, D. E.: A new therapeutic approach to acute coronary occlusion. I. Production of standardized coronary occlusion with microspheres. Am. J. Cardiol. 9:60, 1962.

(66) Jacobey, J. A., Taylor, W. J., Smith, G. T., Gorlin, R. and Harken, D. E.: A new therapeutic approach to acute coronary occlusion. II. Opening dormant coronary collateral channels by counterpulsation. Am. J. Cardiol. 11: 218, 1963.

(67) Watkins, D. H. and Duchesne, E. R.: Postsystolic myocardial augmentation: experimental development and clinical results in six cases. Proc. Soc. Exper. Biol. and Med. 107:659, 1961.

(68) Watkins, D. H. and Duchesne, E. R.: Postsystolic myocardial augmentation: physiological considerations and techniques. J. Appl. Physiol. 17:61, 1962.

(69) Birtwell, W. C., Soroff, H. S., Wall, M., Bisberg, A., Levine, H. J. and Deterling, R. A.: Assisted circulation. I. An improved method for counterpulsation. Trans. Am. Soc. Artif. Int. Organs 8:35, 1962.

(70) Soroff, H. S., Levine, H. J., Sachs, B. F., Birtwell, W. C. and Deterling, R. A., Jr.: Assisted circulation. II. Effects of counterpulsation on left ventricular oxygen consumption and hemodynamics. Circulation 27:722, 1963.

In this technique the femoral arteries are cannulated and connected to a blind "assisting" ventricle. By synchronizing with cardiac activity (EKG) the "assisting" ventricle is phased to remove blood rapidly from the arterial system of the patient during ventricular systole and to return this removed blood during ventricular diastole. This results in (1) the left ventricle ejects against a lower aortic resistance, the time-tension index is decreased and the oxygen consumption of the left ventricle is reduced, and (2) the aortic diastolic pressure is increased which should improve coronary flow, since the latter occurs almost entirely during diastole. This procedure, however, causes some degree of hemolysis and the cannulations required are rather heroic (at least to an Internist).

(71) Dennis, C., Moreno, J. R., Hall, D. P., Grosz, C., Ross, S. M., Wesolowski, S. A. and Senning, A.: Studies on external counterpulsation as a potential measure for acute left heart failure. Trans. Am. Soc. Artif. Int. Organs 9:186, 1963.

(72) Osborn, J. J., Russi, M., Salel, A., Bramson, M. L. and Gerbode, F.: Circulatory assistance by external pulsed pressures. Am. J. Med. Electronics 3:87, 1964.

Recently a method of synchronized counterpulsation has been developed which does not require arterial cannulation. In this

method a "G-suit" is placed over the lower extremities or lower half of the body. By synchronizing with the EKG, negative pressure is applied during systole and positive pressure during diastole. A reversal of the arterial pulse pressure and a reduction of the time-tension index has been achieved by this method in dogs. Experiments on man are supposedly in progress.

D. Evaluation of the types of assisted circulation

(46) Loc. cit.

(47) Loc. cit.

(73) Salisbury, P. F., Bor, N., Lewin, R. J. and Rieben, P. A.: Effects of partial and total heart-lung bypass on the heart. J. Appl. Physiol. 14:458, 1959.

(74) Salisbury, P. F., Cross, C. E., Rieben, P. A. and Lewin, R. J.: Comparison of two types of mechanical assistance in experimental heart failure. Circulation Res. 8:431, 1960.

(75) Salgado, C. R., Lüthy, E., Franch, R. H. and Galletti, P. M.: Influence of assisted circulation upon the size of cardiac cavities. Trans. Am. Soc. Artif. Int. Organs 9:202, 1963.

IV. Conclusion

The two cases presented at the beginning of this protocol are examples of progressive, unrelenting failure of the left ventricle as an effective pump in spite of heroic medical measures. In such cases the loss of effective muscle power of the infarcted area plus the ballooning action of this area combine to produce a situation which is not manageable by pharmacologic means. An approach which may in the future offer some help in this clinical situation is mechanical assistance to the circulation. The two most physiological approaches that have been developed are left heart bypass with diastolic augmentation and synchronized arterial counterpulsation.