Shock in Myocardial Infarction

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Preface

The subject of this book is shock in myocardial infarction and although the major pathogenic factor frequently is myocardial failure, the title cardiogenic shock has been avoided in order to direct the material toward the clinical syndrome as it presents to the practicing physician. The easily remedied factors making up the clinical syndrome are missed if one concentrates solely on the cardiogenic aspects.

Mortality in the shock syndrome associated with acute myocardial infarction has been reduced by the recognition and treatment of complicating factors such as arrhythmias, intravascular volume depletion, respiratory insufficiency and bacteremia. For the patient with shock due solely to loss of functioning left ventricular myocardium, research to date has added little to life. Pharmacological and mechanical means of cardiovascular support have been developed out of an understanding of the basic pathophysiology of the disease. Although the prolongation of existence in this manner adds little to life it does give time to evaluate the next step. As described in the last chapter of this book infarctectomy and revascularization are leading to meaningful survival in a few patients and these patients are being identified through careful physiologic and anatomic assessment. Hope lies in that the final chapter of this book has not been written and that long term mechanical circulatory assistance will become a reality of the present. While awaiting the development of a mechanical heart we must continue to refine our understanding of the ventricular dysfunction in myocardial infarction and develop means of quantifying and limiting the extent of

infarction. We must also seek ways to prevent the occurrence and recurrence of this tragic event.

The studies reported in our chapters represent results of research done at Cook County Hospital and the University of Illinois College of Medicine between 1962 and 1971 and supported by National Heart and Lung Institute Grant HE-08834. We wish to thank the staff, fellows, residents, technicians and secretaries who made this work possible, frequently under the most trying circumstances. We particularly wish to thank Raymond Pietras, M.D., Jeb Boswell, M.D., Edward Winslow, M.D., Ziad Sinno, M.D., Kenneth Rosen, M.D., William Towne, M.D., Sarah Johnson, M.D., Ruben Chaquimia, M.D., Charalambos I. Stavrakos, M.D., Bun Su Co, M.D., Cheng Yee Teng, M.D ., Anthony Cruz, M.D., Mr. Charles Buchino, Dorothy Gore, Virginia Cummings, Ruby Randall, Margaret Tautkus and Patricia Berg. Our Most grateful appreciation goes to Dr. Joh R. Tobin, Jr. who organized and directed the Department of Adult Cardiology from its inception in 1959 until 1968.

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The Coronary Arteries and Myocardium in Acute Myocardial Infarction and Shock

Cardiogenic shock is initiated by an impairment in myocardial function and usually is associated with severe anatomic lesions of the heart. Acute myocardial infarction (AMI) and cardiovascular surgery are common clinical settings in which cardiogenic shock occurs. Noncardiogenic shock may be caused by hemorrhage, trauma, fluid loss, sepsis, anaphylaxis, anesthesia, and stimulation of neural reflex mechanisms (neurogenic). Although noncardiogenic shock is initiated by factors involving the peripheral vascular system, secondary myocardial involvement frequently occurs when shock is severe or prolonged. Under these conditions functional and anatomic derangements of the heart often develop. Anatomic features of the cardiac lesions in cardiogenic and noncardiogenic shock are described in this chapter.

CARDIOGENIC SHOCK FROM ACUTE MYOCARDIAL INFARCTION

Cardiogenic shock (Table 1-1) has become the leading cause of death in patients hospitalized for AMI.[1,2] Since the advent of coronary care units, the frequency of cardiogenic shock in patients with fatal AMI has at least doubled (40 percent to about 80 percent).[1,2] Necropsy studies have shown important differences in the hearts of patients with AMI and cardiogenic shock compared to patients with AMI dying of other complications, such as arrhythmias or emboli. The major differ-

Table 1-1
Power Failure Syndrome (Cardiogenic Shock)

Definition: Inability of myocardium to maintain the level of cardiac output necessary for adequate organ perfusion.

Features
1. Evidence of acute myocardial infarction (history, ECG, elevated serum enzyme levels).
2. Shock (systolic blood pressure <90 mm Hg) with tachycardia and signs of ↑catecholamine output.
3. Evidence of underperfusion of at least one organ system:
   a. Brain (disorientation, seizures)
   b. Bowel (abdominal pain)
   c. Kidneys (oliguria)
   d. Lungs (cyanosis, $\downarrow pO_2$)
4. Hypotension and organ-perfusion inadequacy responding only transiently or not at all to therapy, including vasopressors.

ences are the *amount of myocardium damaged (Fig. 1-1) and the frequency of coronary arterial thrombi (Figs. 1-2 to 1-7).*

Several studies[2–10] of AMI have compared the location and size of the infarct to the presence or absence of cardiogenic shock. The location of myocardial necrosis bears no relationship to the occurrence of shock.[4,6,7,9] Shock occurs with approximately equal frequency in patients with anterior as compared to those with posterior wall infarcts[6,7] The *size* of the infarct, in contrast, does correlate with the occurrence of shock.[3–8] Rosenberg and Malach,[3] Walston et al.,[6] and Harnaryan et al.[5] found shock to be more frequent in patients with larger compared to those with smaller acute infarcts. Others,[2,9,10] however, found no such correlation. Malach and Rosenberg[4] and McQuay et al.[9] noted a higher frequency of previous infarcts (myocardial scars) in patients with cardiogenic shock. This observation suggested that the total amount of damaged myocardium was more important than the size of the acute infarct in determining the occurrence of cardiogenic shock. Page et al.[7] attributed the conflicting data in the previous studies to the lack of quantitation of the extent of both recent and old myocardial damage. To determine the extent of myocardial necrosis and fibrosis, these workers studied 20 patients with fatal AMI and shock, 14 others with fatal AMI without shock, and 20 with fatal shock (noncardiac) without AMI. Of the 20 patients with AMI and shock, 19 had lost 40 percent to 70 percent of left ventricular myocardium and 1 had lost 35 percent; 13 had combined recent and old infarcts, and 7 had recent infarcts only. Of the 14 patients with AMI and no shock, 12 had lost 30 percent or less of left

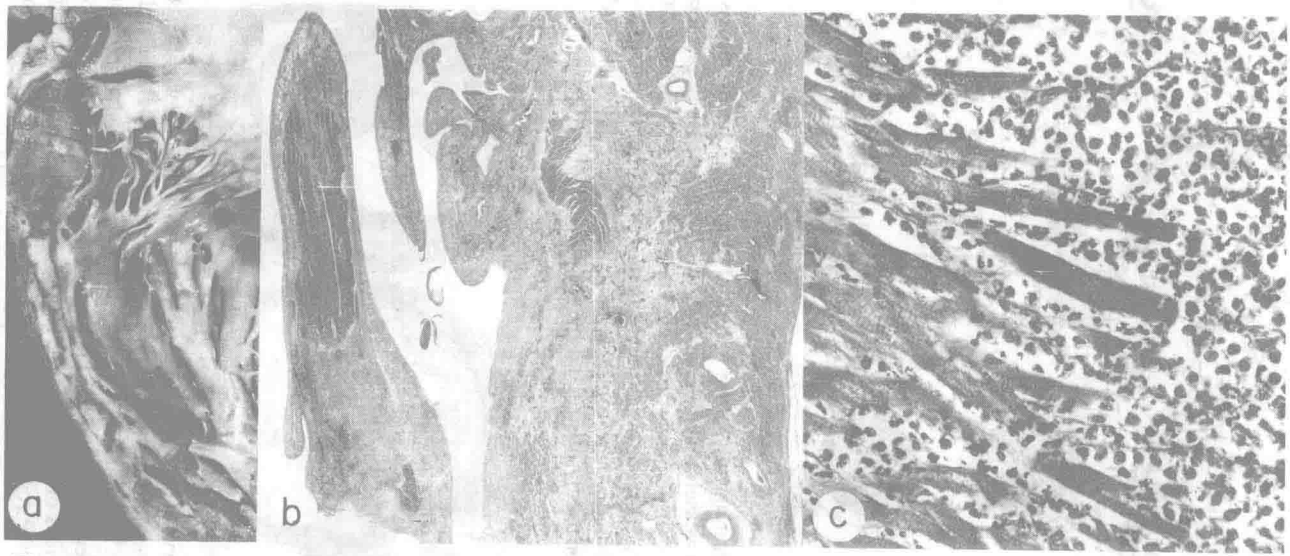

Fig. 1-1. Massive myocardial infarct in a 71-year-old man (A69-232) who developed congestive cardiac failure 1 month before death. Serial electrocardiograms showed severe ST depressions in V4-6, inverted P waves in V2-3, and changes of progressive myocardial infarction. About 3 weeks before death a systolic murmur was noted and severe mitral regurgitation was documented by left ventricular angiography. After initial improvement, cardiac failure progressively worsened, and the patient died in cardiogenic shock. At necropsy, the anterolateral and posterior walls of the left ventricle, including both papillary muscles (a and b), were necrotic. The necrotic muscle cells exhibited either hypereosinophilic dense, hemogeneous cytoplasm or cytoplasm with a preserved pattern of cross-striations (c). The right, left anterior descending, and left circumflex coronary arteries were severely (more than 75 percent) narrowed by old arteriosclerotic plaques. In addition, the lumen of the proximal left circumflex artery was completely occluded by a recent thrombus superimposed on an old plaque. Hematoxylin and eosin stains, original magnifications, × 6.4 (b) and ×250 (c).

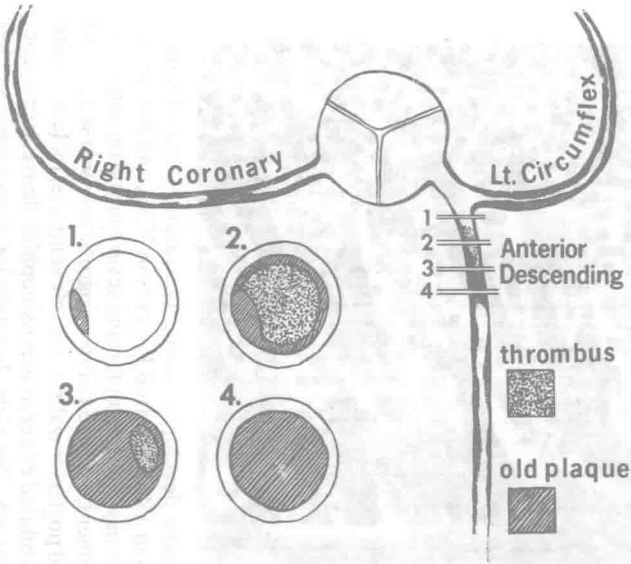

Fig. 1-2. Diagram illustrating the diffuse nature of coronary atherosclerosis and the usual status of a vessel at and distal to a thrombus. At level 2 in the anterior descending artery the lumen is obstructed primarily by a thrombus. At level 3, however, the major percent of narrowing is the result of old atherosclerotic plaquing and just distal to the thrombus, the lumen is severely narrowed (more than 75 percent) or totally obstructed by old plaque only.

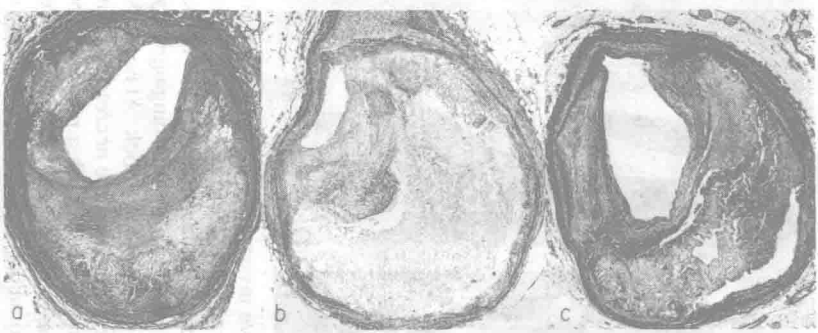

Fig. 1-3. Major coronary arteries in a 63-year-old woman (A69-262) who died 8 days after the onset of acute transmural myocardial infarction. She died suddenly, presumably of an arrhythmia. She never had hypotension. Each of the extramural coronary arteries contained old plaques and their lumens were quite narrowed. No hemorrhages or thrombi were found. (a) Left anterior descending, proximal 1 cm, (b) left circumflex, proximal 1 cm, (c) right, about 5 cm from the aortic ostium. Movat stains; each original magnification × 16.

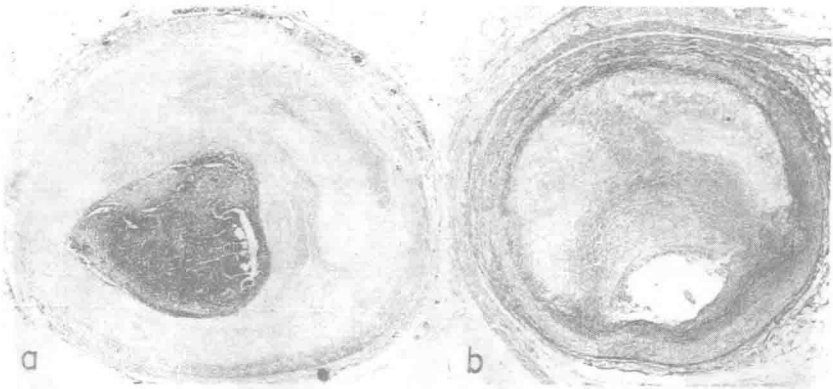

Fig. 1-4. Left circumflex coronary artery in a 57-year-old man (GT #70A-269) who died 4 days after onset of acute myocardial infarction. His course was characterized by shock, severe congestive cardiac failure, and heart block. (a) Proximal, (b) distal. The proximal artery contains a thrombus. The artery just distal to the thrombus is more than 75 percent narrowed by old plaque. Hematoxylin and eosin stain (a), elastic van Gieson (b); original magnifications ×16.

ventricular muscle, 1 had lost 35 percent, and 1, 40 percent; 6 had combined recent and old infarcts and 8 had recent infarcts only. The patients with cardiogenic shock, in contrast to those with AMI without shock, had microscopic-sized foci of necrotic myocardial cells at the edges of the infarcts and at other sites in both left and right ventricles. Similar, widespread, microscopic-sized foci of necrosis also were observed in the hearts of the 20 patients dying of shock not due to AMI. Page et al. concluded that cardiogenic shock in AMI is associated with extensive left ventricular myocardial damage due to recent or recent and healed infarction, and that additional widespread acute myocardial damage occurs secondary to the shock.

A second important feature of cardiogenic shock in AMI is its association with a *high frequency of recent coronary thrombosis* (Figs. 1-2 to 1-7). Of 37 patients with fatal AMI studied by Walston and associates,[6] 24 had cardiogenic shock and 13 did not. No differences between the two groups of patients were observed in the extent of coronary arteriosclerosis, which was extensive and severe in all but 2. The frequency of coronary arterial thrombi in the two groups, however, differed greatly (Table 1-2): thrombi were found in 17 (71 percent) of 24 patients with (Figs. 1-4 to 1-7) and in only 2 (15 percent) of 13 patients without (Fig. 1-3) the "power failure syndrome" (cardiogenic shock) (Table 1-1). Of the 37 patients, 19 had coronary arterial thrombi and 17

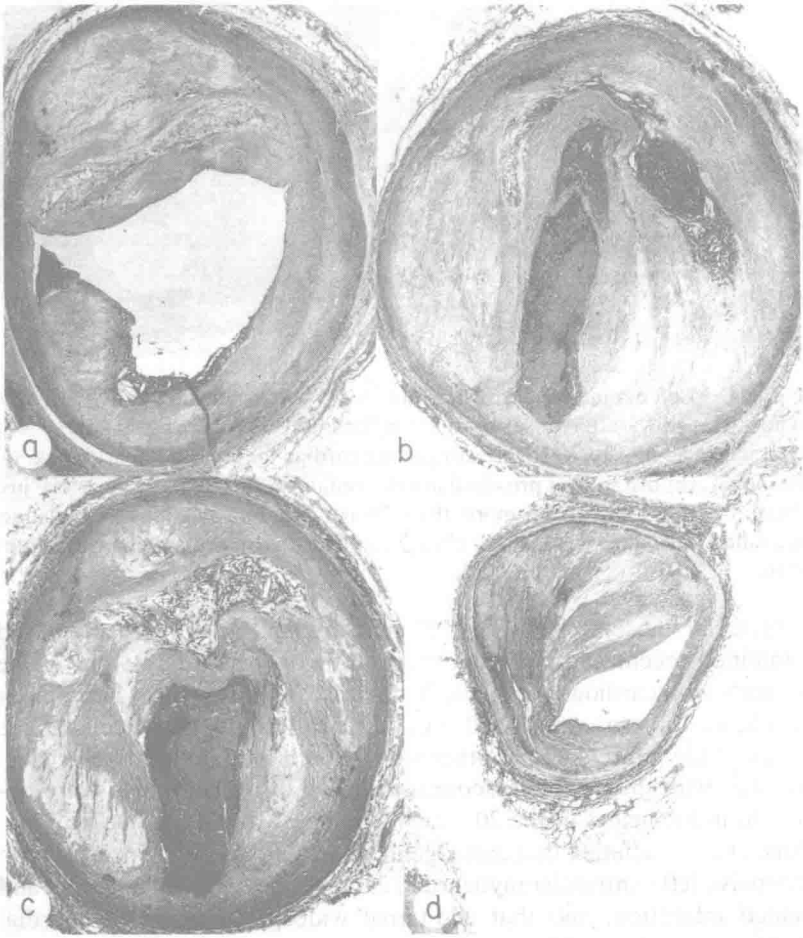

Fig. 1-5. Left circumflex coronary artery in a 43-year-old man (GT #70A-581) who developed severe chest pain 20 hours before death. Electrocardiogram showed an acute anterior myocardial infarct, right bundle branch block, left axis deviation, and subsequently, left bundle branch block. A pacemaker was inserted. Nevertheless, the patient experienced progressive cardiac failure and shock. At necropsy, a large lateral infarct was present. Each of the three major coronary arteries were more than 75 percent narrowed by old arteriosclerotic plaques. In addition, the lumen of the proximal left circumflex coronary artery was occluded by recent thrombus superimposed on old plaque (b and c). The thrombus appeared to have formed at the first site of severe luminal narrowing since the lumen of the first portion of the vessel was narrowed less than 50 percent (a). The lumen of the artery distal to the thrombus was very narrow (d). Hematoxylin and eosin stains; each original magnification × 20.

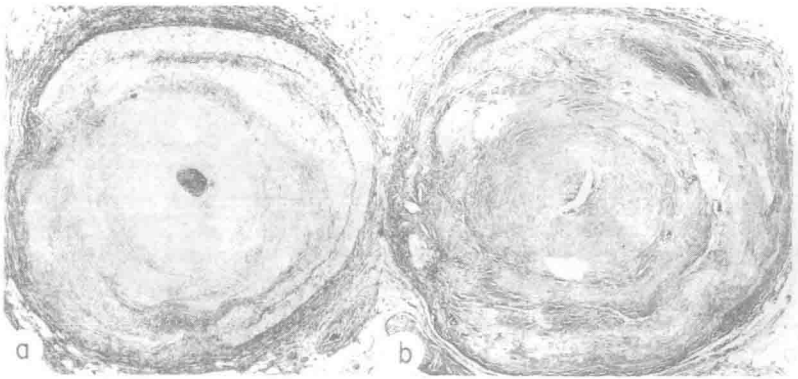

Fig. 1-6. Anterior descending coronary artery 6–7 cm from aortic ostium in a 59-year-old woman (A68-285) who had the onset of acute transmural myocardial infarction 48 hours before death. She began to have intermittent chest pain 2 months earlier and had had systemic hypertension for 10 years. Her final 2 days were characterized by shock, pulmonary edema, and bradycardia. At necrospy, a "massive" anterior wall infarct, which was aneurysmally dilated, was present. The lumens of all three major coronary arteries were more than 75 percent narrowed by old plaques. In addition, a small occluding thrombus was present in the left anterior descending vessel (a). The percent of narrowing caused by the thrombus is small, however, compared to the percent of narrowing resulting from old atherosclerotic plaques. The luman at the site of (a), and distal to (b), the thrombus is already severely narrowed by old plaque. Movat stain (a); hematoxylin and eosin stain (b), each original magnification ×25.

(90 percent) of them had the power failure syndrome; of the 18 patients without thrombi, 7 (39 percent) had the power failure syndrome. Of their 20 patients with cardiogenic shock, 5 had acute coronary occlusions from hemorrhages into old arteriosclerotic plaques whereas occlusions due to this mechanism occurred in only 1 of the 13 patients without shock. Thus, 22 (88 percent) of the 24 patients with cardiogenic shock had acute coronary occlusions due to thrombosis or intramural hemorrhage, whereas only 3 (23 percent) of 13 patients without shock had an acute occlusion. Kurland and associates[10] also studied the association of acute coronary occlusion and cardiogenic shock in fatal AMI. These authors, however, did not specify whether the acute coronary occlusions were due to thrombi or to hemorrhages into old arteriosclerotic plaques or to both. Of 46 patients with cardiogenic shock, 31 (67 percent) had acute coronary occlusions; of 81 patients without shock, 39 (48 percent) had acute coronary occlusions. Although it was less dramatic than in the study of Walston et al., the difference between the two