

tion of patients with nontransmural infarction.

Several comments can be made regarding the study of Kossowsky et al.<sup>7</sup> First, the relatively benign prognosis (in terms of death from pump failure) in the 22 patients who escaped infarct extension is not unexpected. The clinical course of patients with myocardial infarction has been convincingly linked to the extent of ischemic damage to cardiac muscle,<sup>9</sup> and rises in enzymatic levels (a good indicator of infarct size) have been found to be higher in patients with transmural than nontransmural myocardial infarction.<sup>3,4</sup>

Secondly, Kossowsky et al<sup>7</sup> have identified nontransmural myocardial infarction as an intermediate step on the way to transmural myocardial infarction in some patients. It is already well recognized that the dividing line between transient ischemia with short-lived electrocardiographic changes and absent or minor enzymatic elevations,<sup>10</sup> and nontransmural infarction with longer-lasting electrocardiographic changes and definite enzymatic rises, is not always clear-cut, and a "stuttering" progression to infarction is commonly seen. It appears from the study of Kossowsky et al<sup>7</sup> that a similar gradual progression from nontransmural to transmural infarction also occurs rather frequently; however, it is disappointing that their complicated and uncomplicated groups could not be differentiated on clinical grounds at the onset of illness.

Thirdly, and finally, the article by Kossowsky et al<sup>7</sup> focuses upon an important subgroup of patients in which the efficacy of measures currently being evaluated for their ability to limit infarct size<sup>9</sup> should be investigated, *ie*, those with nontransmural infarction which may eventually become transmural. Although a means of delineating this group prospectively is not yet available, the high incidence of transmural extension in the study by Kossowsky et al<sup>7</sup> suggests that a controlled trial of therapy for intervention in all patients with nontransmural infarction might represent a profitable approach. One can only concur with Kossowsky and associates<sup>7</sup> that therapeutic manipulations may be more useful in the patient with a small nontransmural infarction than in the patient who has suffered massive transmural necrosis.

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## Atrial Fibrillation in Acute Myocardial Infarction

### Significance and Therapeutic Implications

In this issue of *Chest* (see page 8), the importance of atrial fibrillation that develops during the course of acute myocardial infarction was again emphasized.<sup>1</sup> Considerable controversy continues to exist regarding the prognostic significance of atrial fibrillation during myocardial infarction. A number of investigators, including Dr. Cristal and his associates, have shown that there is a greater overall early mortality in those patients who develop atrial fibrillation in this circumstance, especially in anterior infarction. Other authors<sup>1,2</sup> have shown that this complication is common but does not adversely affect mortality. A consideration of the possible mechanisms that produce atrial fibrillation in acute infarction and its hemodynamic consequences may provide a framework for resolving the controversy and for recommending appropriate therapy.

The first mechanism considered by Cristal et al was the development of atrial fibrillation as a consequence of left ventricular failure, with an acute

elevation of ventricular filling pressures. The occurrence was much higher in patients with heart failure and extensive anterior infarction, as was a poorer prognosis, than in patients with inferior infarction. Other possible mechanisms of atrial fibrillation include (1) atrial wall infarction, which also is more likely with the larger areas of myocardial necrosis; (2) the sudden release of localized stores of myocardial catecholamines in autonomic nerve endings; (3) the development of acute pericarditis; (4) the existence of intrinsic atrial muscular disease, with disorganization of the architecture of atrial muscle; (5) the presence of associated chronic pulmonary disease; (6) the occurrence of acute hypoxia; (7) the administration of drugs such as isoproterenol or digitalis; (8) the occurrence of hypokalemia due to diuretic therapy; or (9) a combination of these events with acute heart failure. The prognostic significance may then depend upon the subgroup of patients included in a given study and the etiology of atrial fibrillation in the larger number of patients.

In acute myocardial infarction in animal models and in man, the stroke volume is relatively fixed during the early stage. Loss of atrial function, with its influence on ventricular filling, may lead to severe hemodynamic depression and a lowered cardiac output. When this is coupled with extensive damage to the left ventricle, major circulatory derangements occur, and a higher mortality results. The prognostic factor, then, is the extent of myocardial damage and not the occurrence of atrial fibrillation independently. In inferior infarction the degree of myocardial loss is less extensive, and atrial fibrillation is not as devastating; however, in general, atrial fibrillation developing within 72 hours of an acute myocardial infarction results in a poor prognosis, much worse than that for patients who are resuscitated from major ventricular arrhythmias within the first 24 hours.

Treatment of atrial fibrillation during the course of acute myocardial infarction needs reexamination and requires knowledge of its etiology. The urgency for treatment depends upon the level of hemodynamic depression, the ventricular heart rate, the presence of other drugs, and the mechanism producing the atrial fibrillation. If the ventricular rate is rapid and the hemodynamic status is markedly depressed, direct-current cardioversion is appropriate early. If the etiologic mechanism is hypoxia, this should be corrected; if it is the administration of isoproterenol or digitalis, these medications should be discontinued; and if it is chronic pulmonary disease, this should be treated. If the atrial fibrillation is due to heart failure but the patient's hemodynamic status is relatively stable, treatment directed at the

heart failure with administration of diuretics and digitalis may be successful in slowing the ventricular rate or reverting the rhythm, or both. If atrial fibrillation persists after heart failure is treated, direct-current cardioversion is indicated before the patient is discharged.

The higher mortality associated with atrial fibrillation in acute infarction must ultimately result from extensive loss of myocardium, but prompt and judicious treatment may well reduce the overall mortality. It should be emphasized that the occurrence of atrial fibrillation should prompt the careful physician to look for an etiology such as subclinical heart failure or latent pulmonary disease.

I conclude that, in our experience, the development of atrial fibrillation during acute myocardial infarction predicts a poorer prognosis and demands careful therapeutic intervention.

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## Unusual Coronary Artery Abnormalities

Three reports<sup>1-3</sup> on unusual abnormalities of the coronary arteries have been published recently. Even though these conditions are rare, the reports were timely because of the increasing interest in and performance of coronary arterial surgery and the growing frequency with which coronary arteriograms are utilized.

Falsetti and Carroll<sup>1</sup> described 11 patients with coronary arterial aneurysm and added these 11 to 23 cases diagnosed ante mortem which were previously reported in the world literature. Unfortunately, in our experience, such aneurysms tend to be multiple and associated with diffuse coronary arterial occlusive disease, making the patients poor candidates for bypass grafting. The authors were able to insert saphenous vein bypass grafts in seven of the 11 patients, and six of these survived. We believe that