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 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 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
when the quality of distal vessels permits, saphenous
ein bypass grafts should be inserted and that sur-
ery should not be withheld simply because of the
existence of coronary arterial aneurysms.

The patient described by Macchi et al.² had an
anomalous communication between the left cor-
ary artery and a peripheral branch of the right
pulmonary artery. Occlusive lesions were present in
the coronary arteries. This sort of anomaly is so rare
as to constitute a medical curiosity; however, the
report is worthwhile, since it emphasizes the value of
complete coronary arteriographic study and also
because the lesion is potentially correctable. The au-
thors' patient apparently did not undergo surgery. In
theory, such an anomalous vessel should be readily
identifiable, and simple ligation should suffice.
Saphenous vein bypass grafts could then be inserted
as indicated.

Lardani and Sheldon³ identified ectopic origin of
the left anterior descending coronary artery from the
right coronary sinus in a patient thought on first
study to have an occluded left anterior descending
coronary artery. The anomalous left anterior de-
sceding coronary artery is described as passing
posterior to the pulmonary artery. Our principal
contact with patients having a left anterior descend-
ing coronary artery arising from the right coronary
system is in patients with tetralogy of Fallot in
whom the vessel passes anteriorly and crosses the
outflow tract of the right ventricle to reach the
interventricular groove. This creates a problem in
correcting intracardiac anomalies, since the incision
in the right ventricle must be placed to avoid the
anomalous left anterior descending coronary artery.
Adequate angiographic study before surgery is val-
uable because injury to an anomalous left anterior
descending coronary artery usually produces a fatal
outcome. If the outflow obstruction cannot be
relied on by excising the obstructing infundibular mus-
cle and by pulmonary valvotomy, for example,
in patients with hypoplastic pulmonary annulus, a con-
duct must be placed between the right ventricle and
pulmonary artery to preserve the anomalous left
anterior descending coronary artery.

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Aortico-Left Ventricular Tunnel
The Case for Early Treatment

Communications between the aorta and left ven-
tricle that bypass the aortic valve have come to
be known as aortico-left ventricular tunnels. Such
communications may be acquired or congenital in
nature. The acquired ones are usually secondary to
bacterial endocarditis and, classically, make their
appearance in adult life. The congenital variety is
rare. Okoroma and associates¹ found two cases amon-
g 1,700 examples of congenital heart disease.
The peculiarity of the congenital aortico-left ven-
tricular tunnel is that it makes its presence known
early in infancy.

In the case reported by Nichols and associates in
this issue of Chest (page 74), a to-and-fro murmur
was noted when the patient was only 15 days old,
and surgical closure of this fistula was accomplished
when the patient was five months of age. Okoroma
and associates¹ emphasized that the young age at
which a murmur is detected leads to distinguishing
congenital aortico-left ventricular tunnel from other
conditions in which there is abnormal runoff from
the aorta associated with murmurs both in the sys-
tolic and diastolic periods, conditions such as rup-
tured congenital aortic sinus aneurysm, ventricular
septal defect associated with aortic insufficiency, and
fistulas involving the coronary arteries. The uncom-
monly great degree of dilatation of the ascending
aorta seen in congenital aortico-left ventricular
tunnel is yet another characteristic.

Several authors, including Nichols and associates,
have observed that in patients undergoing surgical
repair of aortico-left ventricular tunnel, there is
a disturbing incidence of postoperative aortic valvu-
lar insufficiency. This functional state has been
charged, in part, to the prominent dilatation of the
aortic root.² Another cause of aortic insufficiency
mentioned are the valvular changes secondary to
turbulence caused by flow through the tunnel. Also,
it has been pointed out that as the tunnel passes
close to the aortic valve, surgical obliteration of it
may result in distortion of aortic valvular orientation

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EDITORIALS

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