that, in the case of infarction, thrombosis of the coronary artery subserving a region of acute transmural necrosis is almost always detected when cardiac catheterization is performed within four hours of the inception of the clinical episode. However, since complete thrombotic occlusion appears less frequently in patients catheterized 12 to 24 hours after the onset of infarction but can follow recurrent bouts of proven coronary vasospasm in patients who eventually suffer myocardial infarction, spasm may contribute to the pathogenesis, establishment, and evolution of myocardial infarction. In the absence of pathologic or physiologic coronary spasm, transient platelet aggregations, relatively stable platelet thrombi, occlusive thrombi subjected to rapid recanalization and morphologically altered coronary plaques could alternatively be the mechanisms of unstable angina or acute myocardial infarction. It is interesting that patients with unstable angina frequently have asymmetric coronary lesions with a narrow neck and irregular borders on angiogram; such lesions, which suggest ruptured plaque and/or superimposed thrombi, are uncommon in patients with stable angina.10

The interaction of atherosclerosis and coronary vasospasm may also influence the short- and long-term clinical course of patients with ischemic pathology. Variant angina can be recurrent and often follows acute myocardial infarction.11-13 However, manifestations of coronary vasospasm eventually abate spontaneously14 and cannot be provoked by ergonovine testing in long-term follow-up. Spasm must play very little or no role in the long-term course of patients with documented pathologic vasoconstriction on original presentation. The extent of fixed coronary atherosclerosis determines the morbidity and mortality of patients with a history of coronary vasospasm.15,16

In summary, a continuum of varying interactions between coronary atherosclerosis, pathologic and physiologic spasm, heightened platelet aggregability, thrombus formation and morphologic alterations of atherosclerotic plaque seem to underlie clinical expressions of ischemic heart disease and the development of arterial atherosclerosis.

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The Technique of Catheter Ablation of Cardiac Arrhythmias

In recent years, a catheter technique for closed-chest ablation of the atrioventricular (AV) junction has emerged as a therapeutic alternative for the treatment of supraventricular tachyarrhythmias.4,16 The technique involves delivery of one or more direct-current shocks through a cardioverter to a catheter electrode (cathode) that records the His bundle electrogram and a patch electrode (anode) that is placed adjacent to the left scapula. The amount of electrical energy used in each shock ranges from 35 to 500 (usually at least 200) J. Successful ablation is judged by the development of
complete AV block produced by the thermal injury at the AV junction. This can be accomplished in approximately 60 percent of patients during a first attempt which includes one or more shocks.3 Implantation of a permanent pacemaker is mandatory following a successful procedure.

Although no complications are observed in a small number (seven) of patients reported by Ector et al in this issue of Chest (see page 676) the procedure is by no means benign. In an international registry of 127 patients who underwent catheter ablation of the AV junction, one developed ventricular fibrillation, one cardiac tamponade, and one transient hypotension as immediate complications.3 Myocardial damage as evidenced by a slight rise of creatine phosphokinase MB was not infrequently observed. In the same registry of patients, three developed ventricular tachycardia, one hemothorax, one thrombosis of the left subclavian vein, three sepsis related to infection of the pacing catheter and/or the pacemaker pocket within one month after the procedure. During a mean follow-up period of 9.9 ± 8.2 months, 90 (71 percent) of the 127 patients remained in complete AV block, three died suddenly, four died of progressive heart failure, and four died of noncardiac causes.

The technique of catheter ablation of AV junction, thus, provides an effective means for controlling supraventricular tachyarrhythmias but creates pacemaker dependency and may produce serious complications. This therapeutic modality should be performed only by experienced electrophysiologists and should be reserved only for patients with disabling supraventricular tachyarrhythmias refractory to medical therapy, including the use of experimental antiarrhythmic drugs. Future efforts should be directed toward refinement of the technique such that AV conduction will be modified but not eliminated, and damage to the surrounding ventricular myocardium avoided; that is, to control supraventricular tachyarrhythmias without the need for pacemaker implantation and without creation of potentially arrhythmogenic lesions in the ventricular myocardium.

At present, the use of this technique for treating patients with tachyarrhythmias associated with anomalous AV bypass tracts or recurrent ventricular tachycardia is limited.4,5 Precise localization of anomalous AV bypass tracts and the origin of ventricular tachycardia is often difficult. Electrical shock delivered in the coronary sinus can cause perforation leading to cardiac tamponade, and may also damage the adjacent atrial and ventricular tissues. Electrical shock to the ventricle may further impair already compromised ventricular function and may produce potentially arrhythmogenic myocardial lesions. More work is needed to develop a safer and more effective technique for ablating anomalous AV bypass tracts and ventricular arrhythmias.

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