so troublesome to Guiraudon and colleagues1 that in patients with myocardial scarring due to coronary artery disease, they have merely excluded the scarred area of the heart by an endocardial incision, basing the diagnosis and localization upon the pre-operative assessment. Mapping only the epicardium also may not be relied upon to localize the origin of the arrhythmia. In the patient reported by Camm et al, the origin of the arrhythmia was found when the right ventricle was opened; for the offending area was on the ventricular septum. When one deals with a left ventricle, scarred from an infarction, this may be even more complicated in that the most severe and extensive involvement will be in the endocardial portion of the myocardium. The next problem with mapping patients with MVA is brought about by the arrhythmia itself. After one or two beats, the characteristics of the MVA may change, and moreover, sustained MVA may be difficult to initiate; but one or two beats may be relatively easy to induce. Thus, in order to obtain an accurate map, it is necessary to have the capability of analyzing one beat in the operating room. Harrison et al.,2 working with Gallagher at Duke, are now using such a device in the laboratory which makes simultaneous recordings from multiple myocardial points; and then with a computer, the activation sequence of the one complex can be quickly determined. A pattern of surface electrodes and plunge intra-myocardial electrodes offer promise for more accurate intraoperative analysis of the MVA.

The authors have used cryothermia for ablating the myocardium that causes the MVA, as advocated by Harrison and colleagues3 and Mikat and co-authors.4 A special instrument of considerable capacity is required for this purpose. Cryothermia kills the cells in the area of the freeze, but does not cause immediate disruption of the tissue. By the time the non-viable area becomes absorbed, scar tissue has grown in to effectively heal the area. This reduces to a minimum injury to surrounding myocardium. The only precaution one has to use is not to freeze the coronary arteries, since changes do occur in the wall that eventually may lead to their occlusion.5

The report by Camm and associates is important. Although it is the report of a single case, it demonstrates that the MVA can originate from a small area of ventricular myocardium. The area can be identified and successfully ablated by the application of a modality that causes relatively little disruption of surrounding myocardium. It is hoped that a year from now Camm et al will inform us concerning the outcome in their patient.

Perhaps of greatest significance is the demonstration that other cardiac arrhythmias with the potential to cause death and disability can be corrected by the direct surgical approach, a field largely neglected in the rapid advances of cardiac surgery. The direct surgical treatment of arrhythmias could well be the last frontier in cardiology open to the surgeon.

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Atrial Myxomas

Triumph of Machine over Man

Not too long ago, the diagnosis of atrial myxomas resided entirely within the domain of the pathologist at autopsy. Lamenting this sad situation, Prichard in 1951 commented:

Tumors of the heart are rarely diagnosed before autopsy. There, diagnosis is either impossible or a matter of chance. Seen from the threshold of an era of even bolder cardiac surgery, these tumors present a dismal diagnostic prospect.5(p39) Prichard was correct in that the surgical remedy for this lesion was far superior to the diagnostic methods. In fact, in 1952 the first atrial myxoma was removed at the Johns Hopkins Hospital, and this myxoma was among the earliest to be surgically excised; however, until recently, almost all myxomas recognized before death were stumbled upon at catheterization. Since angiographic study was virtually the only way to make the clinical diagnosis of myxoma, only those hemodynamically significant lesions associated with symptoms were detected.

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Those patients with an increased first heart sound or with mild vague complaints of shortness of breath would not, and clearly should not, have undergone cardiac catheterization.

What has made the difference recently and has largely solved Prichard’s dilemma is the echocardiogram. The echocardiogram has made accessible an area previously inaccessible except through invasive techniques. We have recently studied a young woman who had one episode of paroxysmal atrial fibrillation. Her first heart sound was somewhat increased in intensity, but otherwise the findings from physical examination and laboratory evaluation were entirely normal. With a consideration of mild mitral stenosis, an echocardiogram was obtained; and, surprisingly, a myxoma was found. Two-dimensional echocardiographic studies have taken us one step further by noninvasively providing an image of a left atrial myxoma every bit as good as by an angiogram, if the patient’s thoracic cavity allows a technically good echocardiogram to be obtained. Two-dimensional echocardiograms also allow us to see excursion of the tumor and perhaps to detect those that do not protrude into the mitral orifice during diastole.

This triumph of machine over man is well illustrated by the experience with left atrial myxomas at the Johns Hopkins Hospital. Twenty-five consecutive myxomas have been identified over the past 50 years at this institution (Fig 1). At a glance, one can see that the number of atrial myxomas has increased. Undoubtedly, this increase does not reflect a true increase in the incidence of the lesion but rather reflects improved diagnostic abilities. Prior to 1950, none of these lesions was recognized before death. In the decades of the 50s and 60s, over one-third of the lesions were diagnosed before death; however, in the past eight years, 90 percent (9/10) of the atrial myxomas have been recognized clinically and have been appropriately treated with surgical excision. Of the four most recent consecutive myxomas, the diagnosis was first made by echocardiograms.

Although we now are better at making this diagnosis, myxomas are nevertheless mainly diagnoses of surprise. Only one of the clinically recognized lesions noted in Figure 1 was seriously suspected before the diagnosis was made, either invasively or by echocardiogram. Myxomas continue to be the greatest masqueraders of other diseases, including rheumatic disease of the mitral valve, collagen vascular disease, and bacterial endocarditis; however, if one looks at how myxomas affect the organism and the way that they mimic other diseases, it appears that they usually simulate atypical variants of those diseases. The misdiagnosed mitral stenosis usually occurs in a patient who has had symptoms for less than a year’s time, who has never had a murmur described before, and whose heart is in sinus rhythm. Furthermore, the murmurs may change from examination to examination, as may the patient’s symptoms. When collagen vascular disease is simulated by an elevated erythrocyte sedimentation rate, elevated concentrations of globulins in the serum, fever, and leukocytosis, this occurs with normal serologic data and with normal findings on arterial biopsies. Similarly, infective endocarditis, when simulated, is always culture-negative infective endocarditis. Unfortunately, these atypical features are too often only best appreciated retrospectively, after the echocardiogram, cardiac catheterization, surgical exploration, or, most unfortunately, an autopsy shows the presence of the tumor.

Although the diagnosis of atrial myxomas is being made more commonly, there is still a tendency to stumble upon them, rather than anticipate their presence. In large part, this reflects the paucity of clinical signs and symptoms that may accompany such a lesion. With the dramatic increase in recognition of these lesions clinically, thanks to our new noninvasive tools, we will hopefully become more astute in anticipating echocardiographic findings and, finally, in making myxomas a man-made (or woman-made), and not a machine-made, clinical diagnosis.

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FIGURE 1. Number of atrial myxomas seen at Johns Hopkins Hospital over 50-year period. Shaded part of each bar represents those that were recognized clinically, and white part of bar represents those that were diagnosed at autopsy.
Noninvasive Detection of Fungal Endocarditis

Fungi rank rather low among microbial agents causing endocarditis. Nonetheless, the occurrence of fungal endocarditis with the development of large mycotic vegetations attached to the aortic or mitral leaflets is increasing in frequency. Three distinct predisposing conditions include (1) disseminated mycotic infection, (2) intravenous narcotic abuse, and (3) impaired host defenses.

Changes in the epidemiology of infective endocarditis have been attributed in part to medical progress. Examples of "advances" in medical technology that predispose to infection by impairing or bypassing host defenses include prolonged therapy with antibiotic drugs, intravascular portals of entry (eg, indwelling plastic catheters), cardiac prosthetic valves, and sophisticated cardiovascular surgery. It is ironic that therapy for bacterial endocarditis using high-dose therapy with antibiotics administered via long-term indwelling catheters should predispose to fungal endocarditis.

The most common fungi to cause endocarditis are Candida species, although aspergilli are an increasing cause of infection following cardiac surgery. The clinical features of fungal endocarditis generally resemble those of bacterial endocarditis; however, embolization to a major artery is particularly suggestive of a fungal etiology. Mycotic vegetations on cardiac valves tend to be large, friable, and easily detached. Histologic examination and culture of the surgically removed embolic material aid greatly in establishing a fungal etiology. Also, the sheer bulk of fungal vegetations often results in changing cardiac murmurs, an otherwise overstated criterion for infective endocarditis. With the possible exception of cryptococcal infection, no reliable serologic tests are yet available to diagnose mycotic endocarditis. Finally, although cultures of blood are often positive when Candida species are causative, other fungi are rarely isolated from the blood, despite specific attention to culturing these agents.

M-mode echocardiography appears to be a promising aid for the detection of valvular vegetations, which in the appropriate clinical setting may direct the physician to a diagnosis of mycotic valvulitis. Candida vegetations have been visualized on aortic, mitral, and prosthetic valves and have been seen to mimic a left atrial myxoma.

Because vegetations must reach a certain size (2 mm or more) to be visualized by this method, echocardiography is an insensitive tool for screening for endocarditis; however, the usual large size of fungal vegetations should permit a greater yield in this form of endocarditis. In addition, the mere detection of vegetations may be helpful prognostically; such patients frequently do not survive with medical therapy alone. It should be appreciated that echocardiography cannot distinguish infectious from noninfectious vegetations or from myxomatous degeneration. No studies of echocardiography are yet available to indicate its true efficacy as a diagnostic tool.

Although the use of the cross-sectional (real-time, two dimensional) echocardiogram in the detection of vegetations appears promising, its contribution over and above the standard M-mode echocardiogram requires further evaluation. In the presence of the often repeated clinical setting of fever, anemia, cardiac murmurs, and "negative" blood cultures, particularly when the patient already has other serious illnesses, echocardiography may be the only noninvasive tool that will keep the clinician on a course to the correct diagnosis.

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