

percutaneous intervention vs CABG patients after 1 year and 56% vs 82% after 2 years^[13,15]. Therefore, in diabetic patients with multivessel disease amenable to percutaneous intervention the increased rate of major cardiac events in the follow-up period must be emphasized and carefully weighed against the increased perioperative mortality and morbidity.

Most patients with multivessel coronary artery disease, including patients with high operative risks, can safely be treated with either percutaneous intervention or CABG. Diabetic patients with multivessel disease are the only group that is better served with CABG in the long-term. The future availability of coated stents may dramatically reduce restenosis rates. Since restenosis is responsible for most repeat interventions in percutaneous intervention patients, this will surely change our practice. If restenosis rates are substantially decreased, percutaneous intervention might become the preferred revascularization technique in all patients with lesions amenable to percutaneous interventions.

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Atrial fibrillation — a curable condition?

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Atrial fibrillation is the most common arrhythmia in clinical practice. It is a significant public health

problem, affecting 0.4% to 2% of the general population, and as many as 5% of patients are older than 69 years. It is one of the most common causes of hospital admission^[1]. Nevertheless, for many years atrial fibrillation was considered an arrhythmia which either did not require treatment or could be managed adequately with some digoxin. Cardioversion of atrial

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fibrillation lasting >6 months has been a medical taboo, due to the fear of thromboembolic stroke upon defibrillation and evidence that fibrillating atria undergo a progressive electrical remodelling process that favours maintenance of the arrhythmia and may result in degradation of contractile function even after sinus rhythm restoration^[2-4]. In addition, it was common belief that atrial dimensions were critical for recovery of atrial contractile function, and the likelihood of stable cardioversion in patients with enlarged atria was considered very low^[3,4].

There are, however, several good reasons to attempt to restore sinus rhythm even in patients generally considered unsuitable for a curative treatment, such as those with long standing atrial fibrillation, forms recurrent after multiple cardioversions or associated with organic heart disease. First, epidemiological data shows that the arrhythmia increases the risk of death, is a potent risk factor for ischaemic stroke, and may also reduce quality of life, functional status and cardiac performance^[1]. In addition, patients with resistant forms or underlying heart disease are indeed among those at higher risk of complications from atrial fibrillation^[3,4].

Although there are benefits from restoring sinus rhythm in patients with atrial fibrillation, is it feasible to do so, and how? Successful defibrillation can be achieved with pharmacological or electrical cardioversion, but the chances for long-term maintenance of sinus rhythm are not ideal even when using anti-arrhythmic drugs which prevent recurrence in less than 50% of cases^[4]. As a result, during the last two decades, several surgical techniques have been designed to eliminate the arrhythmia and restore atrial contraction.

The first curative approach was the surgical MAZE procedure, conceived with the goal of modifying the arrhythmogenic substrate by interrupting all possible macroreentrant circuits responsible for atrial fibrillation, with suture lines of conduction block placed sufficiently close to each other in a labyrinth pattern^[5]. The operation makes it impossible for an electrical impulse to emanate from any point in the atrium without crossing a suture line, whereas atrial conduction is maintained along several blind alleys of atrial myocardium, and atrial transport function can be restored postoperatively. The surgical MAZE procedure, initially attempted in lone atrial fibrillation either paroxysmal or chronic, has been refined and is typically performed in association with mitral valve or coronary artery bypass surgery, with success rates of 74% to 90% at 2 to 3 years postoperatively and an operative mortality rate below 1%^[6]. Therefore, the role of the cardiac surgeon is invaluable for atrial fibrillation patients who require open-heart

operations, as they can have their arrhythmia eliminated with little or no increase in morbidity and mortality.

Recently, attempts have been made to duplicate the effects of the surgical MAZE procedure by using intraoperative cryoablation or radiofrequency endocardial ablation with the aim of minimizing blood loss and reducing the cardiopulmonary bypass time. In this issue, *Deneke et al.*^[7] evaluated a modified surgical MAZE procedure using cooled-tip radiofrequency endocardial ablation in a small yet randomized series of patients with chronic atrial fibrillation undergoing mitral valve surgery. Combined biatrial surgical ablation and valve replacement was effective in restoring sinus rhythm in 80% of the patients at 1 year compared to only 27% in patients randomized to mitral valve replacement alone. Biatrial transport function was documented in the majority of patients in sinus rhythm, but no benefit was observed in terms of New York Heart Association functional class, maximum oxygen uptake and functional capacity measures. In addition, complexity of the procedure led to prolonged aortic cross-clamp time and duration of cardiopulmonary bypass. The time-consuming nature of the operation may lead to increased morbidity and delayed postoperative recovery, which may preclude widespread application of this approach.

Recent advances in understanding the mechanisms of atrial fibrillation have led to further simplification of surgical and transcatheter ablation techniques. Mapping studies of patients with paroxysmal or chronic atrial fibrillation with or without heart disease have suggested a general tendency for reentrant circuits or ectopic foci to exist in the left posterior atrial wall but not the right atrium, and postulated that the left atrium acts as an electrical driving chamber and should be the target chamber for MAZE surgery or ablation^[8-10]. At the same time, demonstration that ectopic foci from the pulmonary veins could initiate and/or maintain atrial fibrillation^[11,12] led to an hypothesis that pulmonary vein isolation through transmural ablation lesions could cure the arrhythmia.

These findings, inevitably, produced a paradigm shift from the theory of atrial 'critical mass' as described by *Moe*^[13] to the new notion of 'critical zone' specific for atrial fibrillation, which can be identified with the region of junction between the left atrium and the pulmonary veins in the vast majority of patients. Limited intra-operative ablation, by encircling all four pulmonary veins, has been associated with the disappearance of atrial fibrillation in 78% of mitral valve patients, with recovery of left atrial contractility in 71%. The performance of small lesions

encircling ipsilateral pulmonary veins without isolating the posterior left atrium produced stable sinus rhythm in 60%^[9,14]. Importantly, pulmonary vein isolation can be performed today by using an epicardial radiofrequency approach without cardiopulmonary bypass^[15] and sinus rhythm can be restored in about 80% of chronic atrial fibrillation patients with mitral valve disease^[16]. Compared with intra-operative endocardial ablation, the epicardial approach is less time-consuming and easier to perform, and the contiguous nature of the lesions applied can be validated under direct vision. Compared with the surgical MAZE procedure, epicardial pulmonary vein isolation has several advantages, including reduced thrombogenic potential, preservation of atrial vascularization and lower risk of bleeding. A potential limitation of this technique is the possibility of creating non-transmural lesions^[15], thereby increasing the risk of proarrhythmic reentrant circuits.

From a clinical standpoint, surgical and transcatheter ablation experiences have greatly increased our understanding of atrial fibrillation and its mechanisms. Many of the available antiarrhythmic procedures offer high cure rates, but several questions need to be answered to allow widespread application of these techniques. Which features should characterize an 'ideal' therapeutic intervention for atrial fibrillation? It should be as simple as possible, selective to critical atrial areas rather than blindly destructive, and with good feasibility and safety profiles so as to be suitable for widespread application. Because intra-operative approaches require open-heart surgery and prolonged cardiopulmonary bypass is unlikely to be applied as a stand-alone procedure in large numbers of patients with atrial fibrillation, the future ideal curative approach might be used in conjunction with minimally invasive surgery without the need for sternotomy or for percutaneous transcatheter ablation. As for the latter, segmental pulmonary vein ostial ablation along the vein circumference can eliminate pulmonary vein spike potentials expressing conduction between the vein and the atrium. However, this approach showed a continued high recurrence rate of atrial fibrillation due to unmasked foci from the ostial edge or atrial tissue, characterized by difficulty in precise mapping and absence of a similar ablation end-point^[17]. The prospective choice of creating anatomically guided transcatheter circumferential radiofrequency lesions around all pulmonary veins can overcome some of the technical problems associated with the former approach. This novel technique, based on electroanatomical guidance, has been successfully applied to cure patients with either paroxysmal or permanent atrial fibrillation with a low

risk of major complications^[18]. From a pathophysiological viewpoint, the efficacy of circumferential pulmonary vein ablation in suppressing atrial fibrillation may be related to additional mechanisms beyond isolation of pulmonary vein foci, as suggested by the lack of a significant relationship between clinical outcome and lesion completeness defined by voltage reduction inside the circular ablation lines^[19]. In contrast, the amount of postablation low-voltage encircled area seems to be a predictive criterion for a successful ablation, and importantly, the low-voltage area extends outside the ablation line. It is therefore likely that ablation, when effective, results in profound atrial electroanatomical remodelling of the area encompassing the pulmonary vein ostia and involving to some extent the left posterior atrial wall, to the point that the substrate for atrial fibrillation is no longer present^[19].

In conclusion, the most important lesson learnt from cardiac surgeons is that atrial fibrillation is a curable condition, regardless of its duration or the presence of organic heart disease. Electrophysiologists have, in turn, demonstrated on-line elimination of the arrhythmia after ablation of pulmonary vein potentials and identified the optimal site for ablation in the region of the pulmonary vein-left atrial junction, harbouring critical pathways and circuits^[11,12,17-19]. This notion should be adopted by cardiac surgeons, who should modify their approach to one with less extensive lesions limited to the left posterior atrial wall and involving mainly the areas around the pulmonary veins. Altogether, the accumulating evidence supporting the possibility of successfully eradicating atrial fibrillation should press clinicians to change a long-established conservative management approach, whereby stable sinus rhythm restoration is considered feasible only in a few selected cases. Should we, in fact, devise new therapeutic indications for patients with atrial fibrillation? Based on the available evidence, it appears that the substantial benefits from regained atrial rhythm and contraction outweigh the complexity and potential risks of the antiarrhythmic interventions. With improved anticoagulation protocols, the risk of thromboembolic events after restoration of sinus rhythm is virtually absent after appropriate patient screening and prophylactic treatment. Therefore, both cardiac surgeons and cardiologists should attempt interventions on as many of their patients as possible, except for those with specific contraindications.


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Enterovirus infection of the heart — a causal or contributory factor in chronic rheumatic heart disease?

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Infectious agents are an important cause of acquired heart disease, and may damage the heart directly through the effects of microbial replication within cardiac tissues, or indirectly through the induction of cardiac immunopathology or autoimmunity. Rheumatic heart disease is one of the commonest forms of acquired heart disease, and is a major manifestation of rheumatic fever, a post-infectious sequel to group A streptococcal pharyngitis (reviewed by Cunningham^[1]). Streptococcal infection does not involve the heart, and rheumatic heart disease is believed to result from humoral or cellular immune

responses to streptococcal antigens which cross-react with epitopes present on heart valve or muscle, although the precise molecular pathogenesis is poorly understood. Up to half of patients in whom cardiac manifestations are present during acute rheumatic fever will subsequently develop chronic rheumatic heart disease^[2], a common cause of congestive heart failure.

Human enteroviruses are the most commonly identified cause of viral myocarditis, and mounting evidence suggests that viral persistence following acute infection may result in progression of acute myocarditis to chronic myocarditis or dilated cardiomyopathy^[3], another common cause of congestive heart failure. Although the relative contribution of direct viral cytotoxicity vs indirect immune-mediated mechanisms to cardiac pathology remains a matter of