Restoration of atrial function after the maze procedure

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Atrial fibrillation, one of the most common tachyarrhythmias of clinical significance, has a major impact on survival and subjective feeling of wellbeing. The most common mode of treatment, cardioversion with or without subsequent antiarrhythmic drug therapy, is a far from completely effective therapy[1]. In patients with atrial fibrillation and congestive heart failure, antiarrhythmic drugs may actually add to the deterioration of the clinical condition due to their negative inotropic effect[2]. Ablation of the atrioventricular junction and subsequent pacemaker implantation may be a therapeutic alternative for patients with refractory atrial fibrillation. It does, however, leave the atria in the fibrillating state with a remaining threat of thromboembolic complications. The restoration of sinus rhythm by means of catheter ablation is so far available only for a minority of patients and the future role of this therapeutic option remains to be established.

In this perspective the surgical maze procedure, as developed by Cox and colleagues, is an alternative for severely symptomatic patients and those who have experienced systemic thromboembolism[3]. The procedure was designed to cure atrial fibrillation with three specific goals: (1) ablation of the fibrillation, (2) restoration of atrioventricular synchrony and (3) preservation of the atrial transport function. The surgical technique involves excision of both atrial appendages, isolation of the pulmonary veins and the creation of a specific route along which sinus node-induced electrical impulses are conducted to the atrioventricular node. The entire atrial myocardium, with the exception of the atrial appendages and pulmonary veins, is electrically activated by blind alleys that arise from the ‘main route’ between the sinus node and the atrioventricular node. In a 5-year follow-up of this procedure, Cox et al. reported restoration of atrial function in 64 of 65 patients of whom only six were on antiarrhythmic drugs[4]. The atrial function was specifically studied 8 ± 7 months after the maze procedure with echo-Doppler in 46 patients who for several years had suffered paroxysmal (n = 25) or chronic (n = 21) atrial fibrillation. In the majority of this population (n = 33) this procedure was the only surgical intervention[5]. Active atrial contraction was detected in 40 of the subjects (87%), in whom this was apparent on the right side in 38 (83%) and on the left side in 28 (61%). Notably the atrial function (measured as the atrial filling fraction of the ventricles) was essentially normal on the right side when compared with that of control subjects (32% vs 33%). It was, however, markedly reduced on the left side (20% vs 36%). In addition, left atrial filling also appeared reduced. These findings have subsequently been confirmed by other echocardiographic techniques[6]. It has been suggested that the maze procedure itself may decrease atrial function. This may be the result of a less than optimal atrioventricular synchronization on the left side in turn related to an artificially prolonged conduction route. Another mechanism could be tethering of the posterior wall of the left atrium by the suture lines[7]. Although many cardiologists may hesitate to refer patients to open heart surgery on the basis of atrial fibrillation only, the alternative to perform the maze procedure concomitant with other, clinically needed surgical procedures seems attractive.

In this issue Yashima and co-authors[8] report on the follow up of 35 patients with chronic atrial fibrillation with a duration of 9.5 ± 6.5 years (mean ± SD), in whom the maze procedure was performed in addition to other surgical interventions. Initially 74 patients were operated on, in whom sinus rhythm was restored in 53 (72%). Thirty-five subjects were adequately studied in the early (19 ± 8 days) and late (245 ± 134 days) postoperative phase. Mitral valve replacement, valvuloplasty or open com-
misurotomy was performed in 28 patients, aortic valvuloplasty or valve replacement in eight, tricuspid valvuloplasty in 12, and atrial septal defect closure in four. The atrial function was studied with trans-thoracic and transoesophageal echocardiography. Transmitral, transtricuspid, superior vena caval and pulmonary venous flow velocities were measured by pulsed Doppler echocardiography.

All patients had detectable atrial contractility at the final evaluation, although five of the patients, despite sinus rhythm, exhibited no sign of a transmitral atrial filling wave in the early postoperative phase. The left atrial contractile function improved from the early to the late phase, although still depressed, while the left atrial storage function did not improve. The right atrial contractile function was already improved in the early phase. There was also a trend towards further improvement. The right atrial storage function was notably lower than that on the left side, but exhibited a late improvement. Postoperative tricuspid regurgitation may have contributed to the low values in 11 of the patients. The left atrial contractile function improved from the early to the late phase underlines the importance of prolonged treatment with anticoagulants in patients who otherwise would not have been given life-long therapy due to the insertion of a mechanical valve prosthesis. As the authors emphasize, a prospective comparison between patients undergoing valve repair with and without the maze procedure is demanded to further elucidate these aspects. Such a study would also clarify whether the procedure itself adds to the surgical risk of valve repair. This may be a concern because of the need for additional cardiopulmonary bypass and aortic cross-clamping times. Thus, this is a field that deserves further clinical research.

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References