0.038-inch guidewire was inserted into the guiding catheter and advanced deeply through the celiac axis into the splenic artery. The sidearm was introduced into the celiac axis over the guidewire by manipulating the guiding catheter. After the branched graft was in position, only the main graft was deployed by removal of the nickel titanium wire and pressed against the aortic wall by balloon inflation. The folded sidearm was then deployed in the same way. The guiding catheter was replaced with a balloon catheter and the sidearm was dilated completely. After the procedure, aortography showed the complete exclusion of the pseudoaneurysm and good flow of contrast medium through the branched graft (Fig. 2). Computed tomography, which was performed 1 week after the procedure, showed the thrombosed pseudoaneurysm and a perisplenic hematoma without bleeding (Fig. 3). The hematoma spontaneously resolved. No evidence of paraplegia, aneurysm enlargement, or significant graft stenosis had arisen during a follow-up period of 8 months. Follow-up computed tomography has demonstrated persistent exclusion of the pseudoaneurysm.

In recent years, the feasibility and effectiveness of endovascular grafting with straight and bifurcated grafts for the repair of aortic aneurysms have been reported.³⁻⁵ We attempted reconstruction of the descending thoracic aorta including the celiac axis with a branched graft. The only complication was an injury of the splenic artery resulting from guidewire insertion. We believe this complication can be prevented by gently advancing a J-tipped guidewire into the arterial branches. Endovascular grafting with the use of branched grafts permits repair of true

aneurysms and aortic dissections, including the origin of the major aortic arterial branches such as the renal arteries and celiac axis.

In the near future, endovascular grafting will become a life-saving treatment for patients, like our patient, who cannot undergo surgery. Although our short-term result has been favorable, a long-term evaluation on large numbers of patients will be necessary to prove the long-term effectiveness and safety of transluminal endovascular graft placement.

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SURGICAL EXCISION OF THE SINUS NODE IN A PATIENT WITH INAPPROPRIATE SINUS TACHYCARDIA

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Inappropriate sinus tachycardia is a rare arrhythmia occurring in otherwise healthy persons, possibly owing to increased automaticity of the sinus node or an automatic

atrial focus located near the sinus node. The abnormality also may be related to a defect in autonomic control of sinoatrial automaticity.¹ The current report supports the findings of previous studies concerning the role of surgical treatment of patients with this condition.²

Clinical summary. The patient, a 36-year-old woman, had had persistent tachycardia for 4 years. She had previously undergone two unsuccessful catheter ablation attempts for her tachycardia in a highly experienced ablation center. First radiofrequency energy and then direct current were used. The ablations were followed only by a transient and moderate deceleration of the heart rate. She continued to have an accelerated pulse rate at rest, rarely below 100 beats/min. Even with mild exertion (25 watts) the heart rate exceeded 150 beats/min and the associated symptoms of chest tightness and dyspnea re-

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Fig. 1. Intraoperative view of the heart before excision. The site of earliest atrial activation was found on the ventrolateral aspect of the junction between superior vena cava and right atrium (arrow). Note the fibrotic scars on the lateral aspect of right atrium, presumably representing the sites of ablation attempts.

sulted in a significant disability for the patient. Antiarrhythmic medication with verapamil, diltiazem, propranolol, atenolol, metoprolol, and bisoprolol failed to achieve permanent improvement of the symptoms, resulting only in a transient decrease of heart rate. Drug-induced hypotonia (<90/60 mm Hg) and dizziness precluded long-term administration. A preoperative twelve-lead electrocardiogram (ECG) at rest demonstrated sinus tachycardia at a rate of 120 beats/min, with normal P wave morphology and a PR interval of 0.14 seconds. Exercise stress testing showed the peak rate of 184 beats/min at 75 watts, with no ST segment abnormalities. A 24-hour Holter monitor recorded an average rate of 96 beats/min, the minimal rate of 60 beats/min occurring only during sleep.

Before the operation, the patient signed an informed consent form. The heart was exposed through a median sternotomy. After the aortic and venous cannulas had been placed, intraoperative electrophysiologic mapping was performed. The operator used a handheld probe with a quadripolar epicardial electrode, and the recorded potentials were compared with the surface ECG. The initial heart rate of 76 beats/min increased after administration of 1.5 mg atropine (to 120 beats/min) and 2.0 μ g isoproterenol intravenously (to 150 beats/min). The earliest atrial activation site during tachycardia occurred 80 msec before the onset of the P wave on the surface ECG and was recorded on the ventrolateral aspect of the junction between the superior vena cava and right atrium (Fig. 1, Fig. 2, A). Manual pressure at this location resulted in abrupt termination of the tachycardia. Fibrotic scars with some pericardial adhesions were noted approx-

imately 1.5 cm from this point on the lateral wall of right atrium, presumably representing the sites of radiofrequency and direct-current catheter ablation attempts. The right atrium was then opened and an approximately 2×2 cm portion of right atrial wall surrounding the site of earliest atrial activation was excised. The tissue defect was closed with parts of the right atrial appendage by means of a continuous suture. No pericardial patch was needed.

The ECG tracing immediately after the excision of the sinus node area showed a stable atrial escape rhythm at a rate of 70 beats/min. The P wave contour was markedly different, with the tracing being positive in lead I and negative in inferior leads, suggesting a low right atrial escape pacemaker (Fig. 2, B). The patient recovered quickly and was free of tachycardia after the operation. This was proved by repeated ECG and Holter recordings. Holter data indicated a significant decrease of the heart rate, with an average rate of 78 beats/min, a maximum rate of 103 beats/min, and a minimum rate of 52 beats/min. The comparison of 24-hour heart rate trends and heart rate variability curve underscored this finding. The latter showed a uniform one-peak distribution around the mean rate of 70 beats/min, as opposed to the preoperative two-peak pattern. Because of the presence of longer pauses in repeated postoperative Holter recordings (longest pause being 5.8 seconds), an AAIR pacemaker (Medtronic Thera SR, Medtronic, Inc., Minneapolis, Minn.) was implanted. However, subsequent 24-hour ECGs recorded less than 1% of paced actions. The patient reported a significant clinical improvement and had no complaints while receiving no medications during the postoperative follow-up period of 6 months.

Histologic section of the excised material showed a marked fibrosis of the atrial tissue with hyalinization of fibrous fibers in the subendocardial and especially in the subepicardial zone. Muscle fibers of the sinoatrial node were adjacent to a small nodal artery. The fibers were smaller than those of the atrial muscle and loosely arranged with collagenous fibers. A marked fibrosis was noted within the node.

Discussion. The clinical and electrophysiologic findings in our patient were consistent with the diagnosis of inappropriate nonparoxysmal sinus tachycardia.¹ Thorough clinical investigation has excluded various identifiable causes of the sinus tachycardia. The tachyarrhythmia was neither provoked nor terminated by pacing, implicating an automatic origin and excluding a reentrant mechanism. The cause for the arrhythmia may be sought in autonomic imbalance of the sinus node, either because of increased sympathetic or decreased parasympathetic nerve influence. The importance of autonomic pathways in the genesis of this tachycardia is further underscored by reports of occurrence of inappropriate sinus tachycardia after catheter ablation of supraventricular arrhythmias with subsequent damage to the autonomic fibers. However, an excessive intrinsic heart rate (as achieved after pharmacologic denervation of the sinus node) may also be responsible for the arrhythmia. The significance of this mechanism in our patient was emphasized by the intraoperative pharmacologic induction of tachycardia.

The key role of precise intraoperative mapping is highlighted by the fact that the region of origin of the

Fig. 2, A, Intraoperative epicardial mapping sequence showing the earliest atrial activation potential occurring 80 msec before the onset of the P wave on the surface ECG. **B,** Twelve-lead ECG tracing of a stable atrial escape rhythm after the operation. Note the significant change in the P wave morphology with negative tracing in inferior leads.

tachycardia in this patient was on the anterior aspect of the superior vena caval-right atrial junction, whereas previous experimental studies suggested a slightly more lateral location of the sinus node. Although various types of atrial rhythm may occur after the exclusion of the sinus node, most frequently the pacemaker site that achieves predominance is located in the distal coronary sinus area. This appeared to be the case in our patient.

This case raises the question of failure of two previous catheter ablation attempts to inactivate the sinus node. Boineau and coworkers³ showed that the atrial pacemaker cells may be distributed over a large area of right atrial tissue and that in addition to unifocal nodal or extranodal impulse origin, there may be multifocal impulse origin from two or more extranodal pacemaking sites. Further-

more, experimental studies have shown that extrinsic nerve activity might alter the level of the resting potential in the pacemaker cells, thus causing latent pacemaker cells to develop leading automaticity.⁴ Such observations in patients with inappropriate sinus tachycardia would make the catheter ablation approach extremely difficult. Indeed, it became evident during the operation in our patient that the ablation scars were 1.5 to 2 cm away from the site of earliest atrial activation demonstrated by intraoperative epicardial mapping. We believe that a more epicardial location of the impulse origin also could have been the reason for the failure of catheter ablation in our patient.

The role of surgery in the treatment of this disorder so far is unclear. Hendry and associates² found the surgical treatment of inappropriate sinus tachycardia to be less effective

than for other forms of atrial arrhythmias. Only one of three patients remained free of symptoms during the follow-up period. Recent reports have advocated the feasibility of radiofrequency catheter ablation in curing this arrhythmia.⁵ In their recent review on inappropriate sinus tachycardia, Krahn and coworkers⁶ stated that the therapy of this arrhythmia is empiric. Therefore we believe that considering the severity of symptoms and the failure of catheter ablation attempts, a decisive therapeutic approach in these patients, including cardiac surgery, is justified.

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RECURRENT INFECTIVE ENDOCARDITIS ONE YEAR AFTER MITRAL REPAIR IN A WOMAN ADDICTED TO DRUGS

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The presence of a prosthetic heart valve is one of the major factors associated with a shortened overall survival in patients with infective mitral valve endocarditis.¹⁻² Accordingly, mitral valve repair, which is technically possible in this setting, has increasingly been used, especially in those patients at risk of relapsing infective endocarditis.^{1,2} This approach, which was initially intended to limit the subsequent risk of reinfection associated with prosthetic valves, also provides an opportunity to treat medically the recurrent episodes of infective endocarditis. This option may be of value in the management of infective endocarditis in persons addicted to drugs.

A 29-year-old woman was admitted to the hospital for infective endocarditis. Three months before admission she returned to her intravenous drug abuse, which she had discontinued 1 year previously after a first episode of *Staphylococcus aureus* mitral valve endocarditis. At this

time, the other cardiac valves were not involved and there were no embolic events. The patient was given a 6-week antibiotic regimen consisting of methicillin and gentamicin with adequate determination of serum bactericidal titers. Both fever and inflammatory syndrome disappeared. In view of the severe mitral regurgitation resulting from a 2 cm perforation, the patient subsequently underwent mitral valve reconstruction consisting of repair of the posterior leaflet associated with implantation of a prosthetic annuloplasty ring (Duran No. 29). An echocardiographic examination confirmed that the anatomic result of the surgical procedure was good, with only mild residual mitral regurgitation.

The patient totally recovered and remained symptom-free until she was admitted again 1 year later with fever (39° C), chills, sweats, multiple skin petechiae, tachycardia, and a mitral regurgitation murmur. No signs of heart failure were observed, and she did not have hepatosplenomegaly. An electrocardiogram and a chest roentgenogram showed no abnormalities. Laboratory examinations showed polynucleosis (15,000/mm³), anemia (10.2 gm/dl), and a sedimentation rate of 90 mm at 1 hour. A methicillin-susceptible *Staphylococcus aureus* was isolated from blood cultures. Transthoracic followed by transesophageal echocardiography showed large valvular vegetations attached to the posterior leaflet of the mitral valve, which prolapsed within the left atrial chamber during systole, and a central mitral regurgitant jet. No annular abscess was seen (Fig. 1). Computed tomographic (CT) scans showed a large asymptomatic brain abscess (Fig. 2) and a renal-cortical abscess. A multiple-drug antibiotic regimen

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