megaly, and peripheral oedema. The resting electrocardiogram showed atrial fibrillation with a high ventricular rate (98 beats min−1) and signs of biventricular overload.

Echocardiography confirmed the presence of multivalvular disease with moderate mitral stenosis, mild aortic stenosis, mild mitral, aortic, and tricuspid regurgitation, and pulmonary hypertension. After optimization of medical treatment the clinical conditions of the patient improved rapidly and cardiac catheterization was performed. The calculated mitral valve area was 1.2 cm², maximum transvalvular aortic gradient 28 mmHg, left ventricular ejection fraction 61%, and right ventricular systolic pressure 34 mmHg; coronary arteries were normal. Mitral and aortic valve substitution, and tricuspid annuloplasty were performed; the postoperative course was uneventful. Anteroposterior left atrial dimension measured by echocardiography 4 days before and 7 days after surgery was 49 mm and 51 mm, respectively. The patient was maintained with digoxin, diuretics, enalaprilat, and warfarin and was seen monthly at our outpatient clinic. Because of the size of the left atrium and the long duration of atrial fibrillation no attempt was made to restore sinus rhythm. Six months after surgery, on a routine visit, the baseline electrocardiogram showed sinus rhythm with a heart rate of 88 beats min−1. Echocardiography showed the dimension of the left atrium to be 51 mm and stable sinus rhythm with atrial ectopic beats (106 day) were demonstrated on the Holter monitor. A year before, she had undergone successful repair of a dissecting aneurysm of the ascending aorta. Appropriate placement of the lead in the right ventricle was confirmed by a chest radiograph. The implantation site was well healed without fluid accumulation.

On admission, the electrocardiogram demonstrated loss of ventricular capture. The chest X-ray revealed twisting and knotting with transection of the lead within the subcutaneous pocket and apical dislodgement. The lead impedance was found to be increased from 509 to 860 ohms. At reoperation, a tightly fitting fibrous capsule was found without fluid build-up. The terminal pin was retracted and removed from the header. Successful reimplantation of a bipolar silicone lead was performed after puncture of the left subclavian...

Spontaneous restoration of sinus rhythm after cardiac surgery

Chronic atrial fibrillation is the most frequent arrhythmia in rheumatic valvular heart disease. This arrhythmia increases the risk of thromboembolism and impairs cardiac performance. Unless sophisticated surgical techniques are used to decrease atrial size, mitral valve substitution rarely eliminates chronic atrial fibrillation in patients with enlarged atria. In addition, postoperative treatment with both quinidine and direct-current shock have limited benefits when patients with mitral valve disease have long lasting atrial fibrillation and large atrial size.

A 53-year-old woman was admitted to our department with chronic heart failure in NYHA functional class III. Chronic rheumatic heart disease was diagnosed at the age of 35 and treated with digoxin and diuretics was begun. The patient did fairly well until the age of 51 when atrial fibrillation was demonstrated on routine electrocardiogram and aspirin was added. On admission, physical examination showed pulmonary rules, congested jugular veins, S₃ gallop, accentuated S₃, murmurs of mitral, tricuspid, and aortic regurgitation and aortic stenosis, congestive hepato-
The old polyurethane transected lead was sealed off and isolated with a rubber cap and imbedded in the pocket. The pacing threshold was 0.8 V, the current 0.5 mA, the intracardiac R-wave 15 mV and lead impedance of 515 Ω. The new lead and the pulse generator were anchored with a non-absorbable suture to the prepectoral fascia using butterfly and header fixation. Six months after re-implantation, she remained free of recurrent problems.

The pacemaker twiddler's syndrome (PTS) is an infrequent, yet important and easily identifiable clinical problem. It was first reported by Bayliss in 1968; since then several reports have been published describing various presentations of this syndrome.

PTS is characterized by permanent, partial or complete, pacemaker malfunction secondary to the twisting of the pacemaker in the subcutaneous pocket with subsequent pacemaker lead displacement. It results from spontaneous, inadvertent or deliberate rotation of the pulse generator by the patient. This condition may be life-threatening especially in patients who are pacemaker-dependent. The recognition and documentation of radiological signs of the permanent pacemaker lead is important as a baseline for follow-up, especially when symptoms of pacemaker and/or pacing lead dysfunction arise. Those at greatest risk appear to be elderly, obese women with abundant, loose subcutaneous tissue that allows for rotation and torsion of the pulse generator in its pocket.

The mechanism of twiddling has been thought to be the twisting of the pacemaker unit in a large loose subcutaneous pocket, which may be patient-induced. Several preventive methods have been used, such as fixation of the electrode at the site of implantation, suturing the electrode to a butterfly, creation of a small pocket, implantation of the pulse generator subpectorally and drainage of fluid from the pocket. Giving breast support in the postoperative period to prevent movement of the pulse generator and enlargement of the pocket has also been suggested. These preventive measures, however, cannot totally prevent PTS.

Eur Heart J, Vol. 17, December 1996