reduced LV and LA dimensions, in patients with and without concurrent heart disease (Table 1). Although the improvement in LV function was significantly better in patients with no concurrent heart disease, it was also substantial among those with concurrent heart disease.
Conclusion: Sinus rhythm restoration by catheter ablation in patients with AF and symptomatic CHF is feasible and can improve cardiac function, even in those with concurrent structural heart disease.

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Right atrial compartmentalization improves pacing efficacy in bradycardia tachycardia syndrome

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Background: a substantial proportion of patients with sinus node disease treated with DDDR pacemaker have the bradycardia-tachycardia syndrome with recurrences of atrial fibrillation (AF). We evaluated the role of hybrid therapy (DDDR pacing + right atrial compartmentalization) to improve efficacy in the treatment of AF relapses.
Methods: we evaluated AF recurrences at 6-month follow-up in patients implanted with DDDR pacemaker for bradycardia-tachycardia syndrome (AT 500 Medtronic and Identity DR St Jude Medical). Prevention algorithms were activated after 1 month from implant. Atrial lead was implanted at coronary sinus ostium in all patients. Patients with more than 20 episodes of AF, assessed with EGM recording provided by pacemaker, underwent right atrial compartmentalization (cavo-tricuspidalis isthmus and intercaval posterior lesion with Localisa navigation system). Patients were followed during 6 months after ablation to evaluate AF recurrences.
Results: 102 pts experienced more than 20 AF recurrences in 6 months after pacemaker implant with mean number of automatic mode switching (AMS) of 32.3/pt (range 22-61); 97 patients underwent right atrial compartmentalization. Ablation was successful in all pts (bi-directional block of cavotricuspid isthmus and wide double potentials along intercaval lesion). AF recurrences were observed in 36 pts and 61pts were completely free of AF relapses. In pts who experienced recurrences the mean number of AMS at 6-month follow-up after ablation was 2/pt ($p<0.005$).
Conclusions: right atrial compartmentalization may alter the substrate for AF, thus improving the efficacy of pacing. Because it is relatively safe, it may be reasonable adjunctive intervention to maintain sinus rhythm in patients with bradycardia-tachycardia syndrome with pacing indication.

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Study of antitachycardia pacing of atrial arrhythmias in a biophysical model

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Purpose: antitachycardia pacing (ATP) is clinically used to terminate atrial tachycardias or flutter. However, the impact of ATP on episodes of atrial fibrillation (AF) is not fully understood. In this paper, we used a biophysical model of human atria to study the effect of ATP on AF.
Methods: a three-dimensional biophysical model of human atria was developed, its geometry based on magnetic resonance imaging images, and its model of ion kinetics tuned to experimental atrial cell electrophysiology. Atrial flutter and sustained AF could be introduced using either an S1-S2-S3 protocol or a burst-pacing protocol. The computer model also outputs atrial surface electrograms, which were used to assess AF cycle length. Based on sustained atrial arrhythmias several clinically used ATP protocols were implemented such as ramp or burst pacing with different interval decrement and 50Hz burst pacing. More complex algorithms with pacing rate auto-adaptation based on measured atrial cycle length were also implemented. Several pacing sites were studied, as well as simultaneous multi-site pacing.
Results: atrial flutter could be terminated by single site pacing in the isthmus region followed by a decreasing ramp protocol. Due to the more complex nature of AF, pacing did not lead to termination except for some particular cases, and the success of pacing was difficult to predict. It was possible to capture regions of the atria from any pacing location, but regions with more uninterrupted electrical propagation (such as the right atrium free wall) could be captured without extensive optimization of pacing cycle length. The introduction of heterogeneities such as fibrosis or regional changes in refractoriness greatly affected the ability to capture by pacing. With simultaneous pacing on the right and left appendage, it was possible to increase the captured area during sustained AF; however multi-wavelets interactions were observed at the border between the different pacing controlled zones. For all simulations of ATP during AF, once capture was achieved, it was difficult to decrease the pacing rate progressively to restore sinus rhythm due to wavelets entering the captured area. This phenomenon frequently led to AF reinitiation, once pacing was stopped.
Conclusions: our computer model of human atria confirms the ability to pace terminate atrial flutter. The study highlights the difficulty of applying ATP to complex AF patterns. This biophysical model facilitates the study of the ATP mechanisms and the development of dedicated pacing algorithms.

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Superior vena cava flutter: electrophysiology and ablation

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Reentry within a major thoracic vein has been suggested as a cause of atrial arrhythmias. However, little is known about these potential reentrant circuits.
Methods and results: Atypical atrial flutter was induced and mapped in 67 out of 225 atrial flutter ablation procedures. Reentry around the superior vena cava (SVC) was suspected in 3 patients (2 males, age 46, 68, and 78 years). The first patient had previously undergone palliative and corrective surgeries without atriotomy for Fallot tetralogy and the other two patients had no structural heart disease. The suspected SVC flutter was induced and terminated by pacing in all patients. Simultaneous clockwise septal and counterclockwise free-wall activation fronts in the right atrium and double electrograms at the upper crista terminalis area were recorded in all of them. Fusion was demonstrated during flutter entrainment by subeustachian isthmus pacing in all. The postpacing interval following entrainment by pacing from different sites of the right atrium (RA) or coronary sinus was longer than the flutter cycle length. Macroreentry within the SVC was demonstrated both by sequential activation and a postpacing interval matching the flutter cycle length when pacing from different sites around the SVC in all patients.
Atrial-venous-atrial electrogram sequence was demonstrated following flutter entrainment by atrial pacing, which ruled out SVC focal mechanism (automatism, trigger activity, or microreentry). Flutter was terminated by an electrical stimulus delivered to the SVC, which was not propagated to the trabeculated RA, in one patient, and linear radiofrequency application from the distal SVC to the posterior wall of the RA, or to the superoseptal portion of the crista terminalis, in the other two. Flutter cycle length progressive prolongation as the line was enlarged towards the atrium or the crista terminalis was documented prior flutter termination and suggested enlargement of the macroreentry circuit.
Conclusion: Macroreentry within the SVC is a distinctive mechanism responsible for rapid atrial activation which is different to other reported flutter mechanisms, such as upper loop reentry. SVC longitudinal radiofrequency application can eliminate the arrhythmia without the need for complete electrical disconnection of the vein.