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Evidence for vagal denervation as a possible mechanism of successful RF-ablation of atrial fibrillation

Case description

A 70-year-old former athlete complained about a twenty-year history of paroxysmal atrial fibrillation with onset at rest and during the night, which became persistent in the last three years. Previous medication including sotalol, flecainide, disopyramide and amiodarone had been unsatisfactory. A circumferential left atrial ablation was performed after transseptal puncture (fig. 1). During delivery of radiofrequency energy at the posterior left atrium several episodes of vagal activation occurred with slowing of AV nodal conduction and concomitant acceleration of atrial fibrillation cycle length (fig. 2). We repeatedly observed the coincidence of slowed AV-nodal conduction with acceleration of atrial fibrillation cycle length (fig. 3). During ablation atrial fibrillation organised and the driving trigger was found at the right superior pulmonary vein (PV) ostium (ABL: ablation catheter positioned directly at the ostium of the right superior PV; distal electrode with PV electrogram preceding the atrial electrogram on the proximal electrode by 90 msec, fig. 4). During ablation the pulmonary vein tachycardia blocked 2:1 and then persisted 4 seconds after PV disconnection from the left atrium (fig. 5). Afterwards the patient was in stable sinus rhythm and atrial fibrillation could not be induced anymore. At follow-up three months after ablation the patient reported no further episode of atrial fibrillation off anti-arrhythmic drugs. His sinus rhythm rate was generally elevated with a mean rate of 85/min (fig. 6). This case provides unique evidence for the direct effects of vagal activation during delivery of radiofrequency energy: slowing of AV nodal conduction, shortening of atrial fibrillation cycle length and disorganisation of atrial electrograms. After ablation there is evidence of vagal denervation with elevation of the sinus rate lasting up for at least three months.

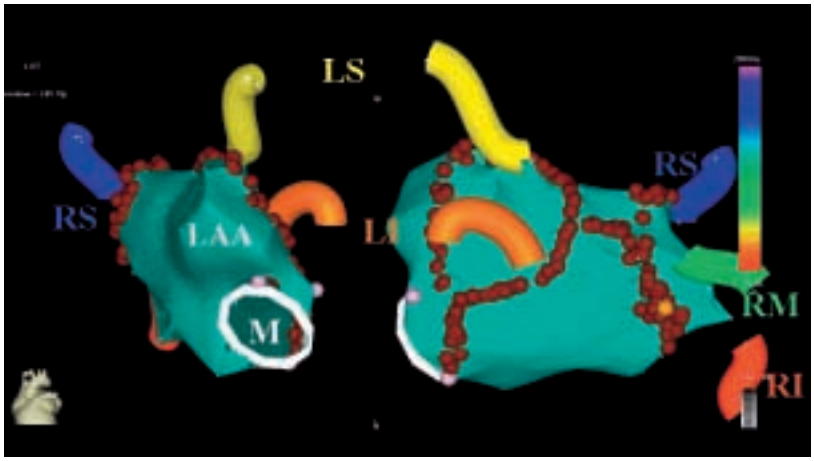

Figure 1
Threedimensional nonfluoroscopic Carto Map of the left atrium. Ablation locations are depicted as red points and are placed around the pulmonary veins on each side and connecting lines between the circles and to the mitral valve in order to prevent subsequent macroreentry.
LS = left superior pulmonary vein; LI = left inferior pulmonary vein; M = mitral valve annulus; RS = right superior pulmonary vein; RI = right inferior pulmonary vein; RM = right middle pulmonary vein draining the right middle lobe.

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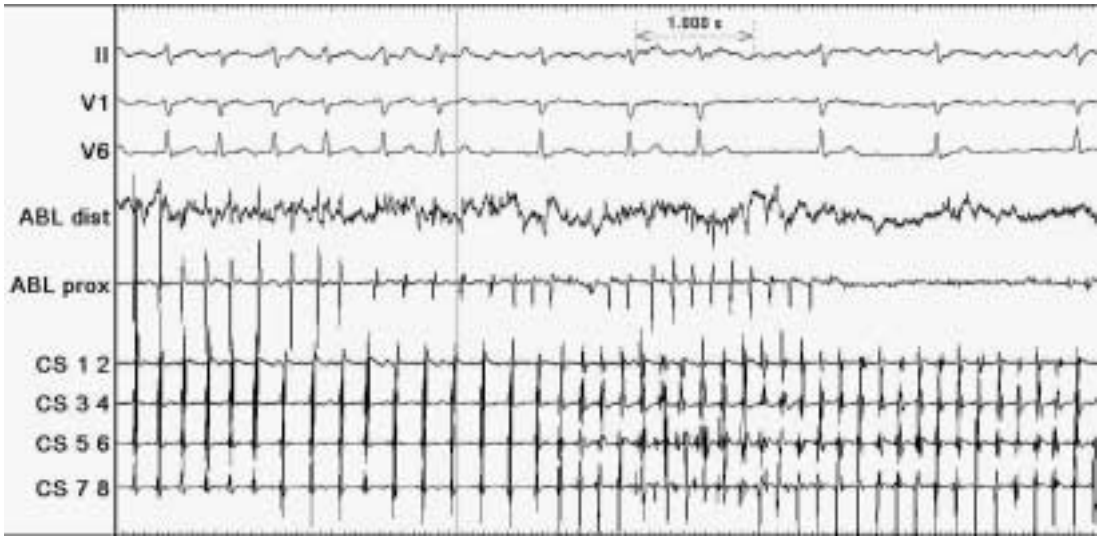

Figure 2
Surface and endocardial ECG recordings during circumferential left atrial ablation: II, V_1, V_6 selected leads from surface ECG. Note the slowing of AV nodal conduction due to vagal activation with concomitant acceleration of atrial fibrillation cycle lenght during delivery of radiofrequency energy (noise on distal ABL). ABL dist = distal electrode of ablation catheter; ABL prox = proximal electrode, distance 4 mm; CS 1–8 decapolar electrode in the coronary sinus.

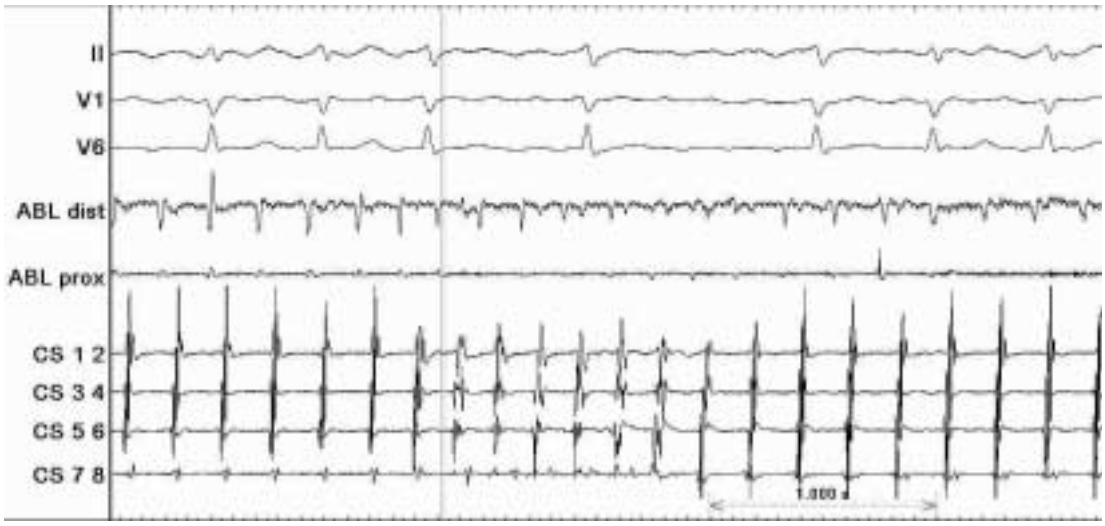

Figure 3
Repeatedly observed vagal activation with slowing of AV nodal conduction and acceleration of Afib cycle lenght.

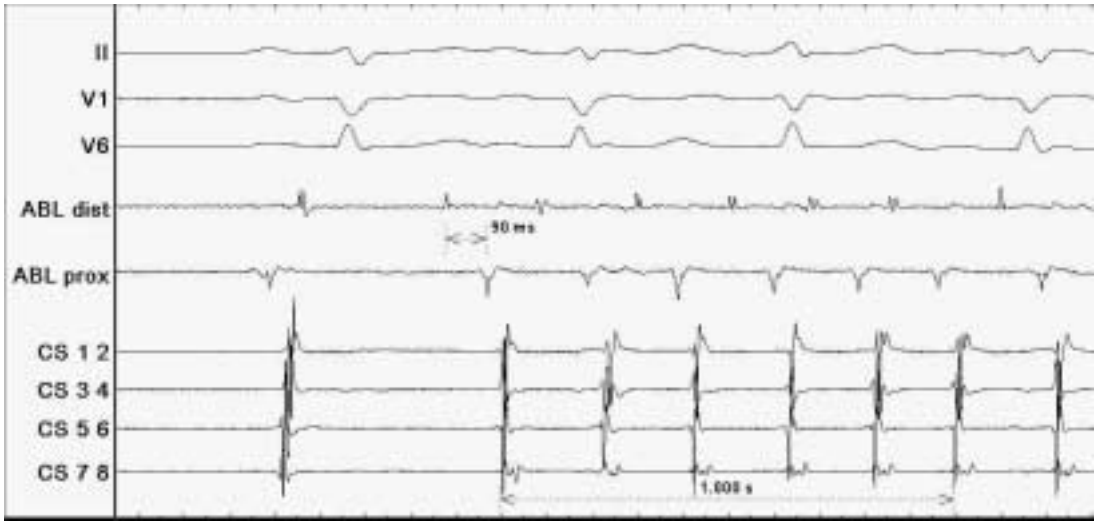

Figure 4
ABL: ablation catheter positioned directly at the ostium of the right superior PV; distal electrode with PV electrogram preceding the atrial electrogram on the proximal electrode by 90 msec.

Kardiovaskuläre Medizin 2005;8: Nr 4

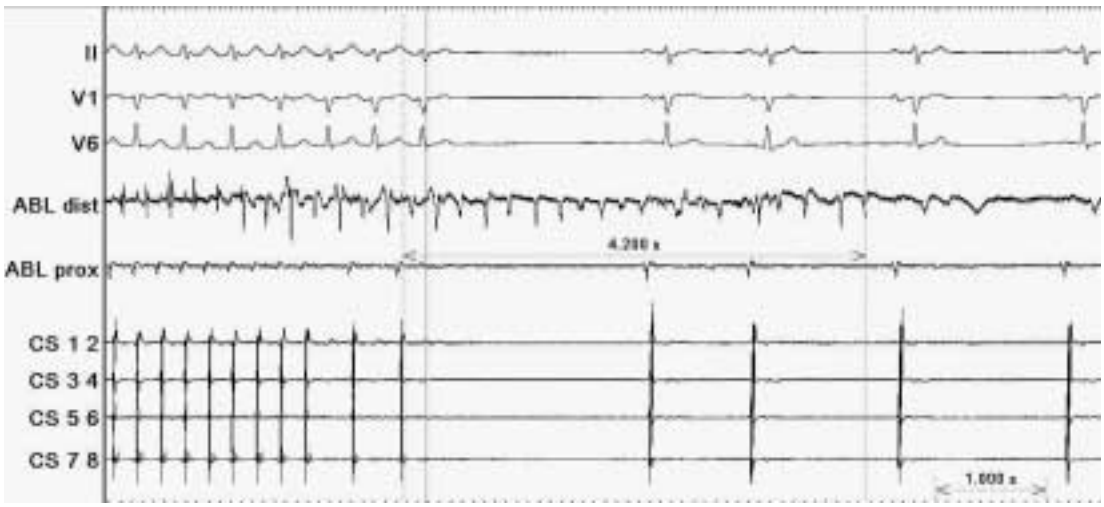

Figure 5
Pulmonary vein tachycardia blocked 2:1 and then persisted 4 seconds after PV disconnection from the left atrium.

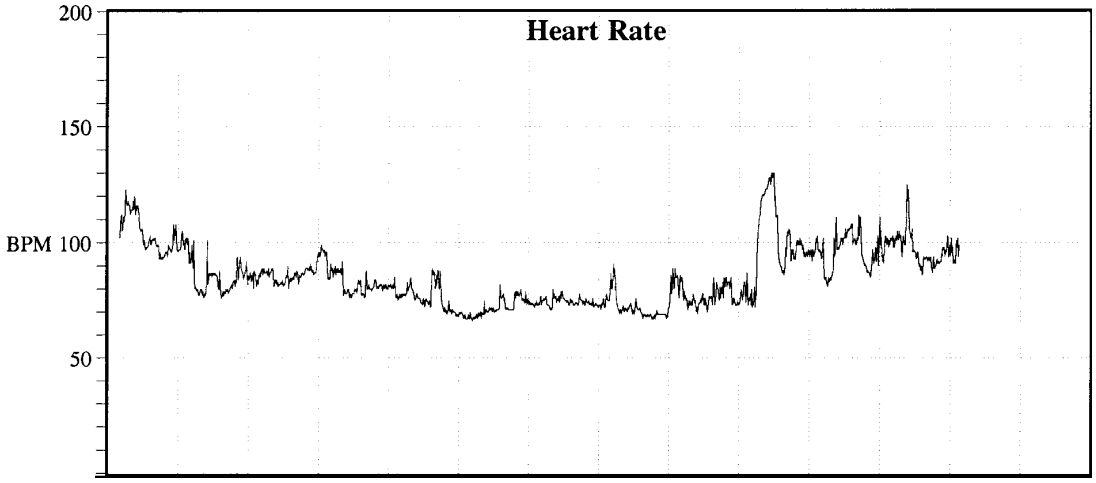

Figure 6
Note the elevated heart rate on Holter ECG 3 months after ablation. The loss of nocturnal rate drop suggests vagal denervation. The patient was in stable sinus rhythm.