

Conduction recovery at the mitral isthmus triggers atrial fibrillation in a patient with rate-dependent 'block' and recurrent atrial fibrillation after previous pulmonary vein isolation and left atrial linear ablation

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During a redo procedure for paroxysmal atrial fibrillation (AF) recurrence, mitral isthmus-dependent atrial tachycardia due to a gap in the previous line and rate-dependent slow conduction in sinus rhythm were demonstrated. After initial achievement of mitral isthmus block, adenosine reproducibly triggered conduction recovery and initiated AF. Further ablation led to AF termination and stable bidirectional block.

Case report

After previous pulmonary vein (PV) isolation and linear ablation at the roof and the inferolateral mitral isthmus of the left atrium (LA) for persistent atrial fibrillation (AF), a 70-year-old woman was referred for a redo procedure due to recurrence of now paroxysmal AF not controlled with antiarrhythmic drugs. Catheter movement induced AF that spontaneously went into a regular atrial tachycardia (AT, CL 350 ms). Despite widely split double potentials (116 ms) observed epicardially (see Supplementary material online, *Figure S1A*), activation and entrainment mapping (see Supplementary material online, *Figure S1A*) demonstrated a macro-reentrant 'counterclockwise' mitral isthmus-dependent mechanism (see Supplementary material online, *Figure S2*). After the first entrainment, the AT degenerated again into AF which spontaneously terminated. In sinus rhythm, careful mapping of the previous line between the left inferior PV and the mitral annulus revealed the presence of rate-dependent slow conduction (see Supplementary material online, *Figure S1B*). Endocardial ablation (ThermoCool NaviStar, Biosense Webster, 43°C, 35 W, infusion rate of 33 mL/min) led to prompt achievement of bidirectional block (see Supplementary material online, *Figure S1C and D*). After confirmation of PV isolation, block at the LA roof and the cavotricuspid isthmus, each vein was tested for durability of isolation with adenosine. Following drug injection (30 mg) during distal coronary sinus pacing to evaluate the left superior PV, recovery of conduction over the mitral isthmus line and AF induction was reproducibly observed (*Figure 1A*). Following the third adenosine injection AF sustained. Additional endocardial/epicardial (43°C, 20 W, 17 mL/min) ablation achieved termination (see Supplementary material online, *Figure S3*), stability of block as well as adequate differential pacing was proved also during adenosine injection (*Figure 1B–D*), and AF could no longer be induced. At 6 months follow-up, the patient was free from arrhythmic episode in the absence of any antiarrhythmic drugs.

Discussion

The presence of widely split double potentials (≥ 50 ms) during macro-reentrant AT is thought to identify conduction block of previous linear ablation. However, this may be misleading, especially when part of the circuit uses epicardial or intramyocardial connections, as in this case. The presence of rate-dependent block (pacing from 800 to 600 ms, *Figure 1B*) suggests that the widely split double potential seen in the epicardial portion of the circuit during tachycardia (*Figure 1A*) is due to slow conduction rather than 'true' block. Adenosine testing has been shown to improve arrhythmia-free survival in patients undergoing catheter ablation for paroxysmal AF¹ and to confirm stability of block after cavotricuspid isthmus² and 'inadvertent' mitral isthmus ablation during accessory pathway ablation.³ Although the hypothesis that adenosine-induced recovery of conduction at the mitral isthmus 'directly' triggered AF is not certain, because other 'indirect' (i.e. increase in 'critical' LA mass related to restored conduction) mechanism might be possible, AF termination during inferolateral mitral isthmus ablation and the impossibility to induce AF again despite several drug-test after achievement of stable bidirectional block show that, at least, some causal relationship exist. Thus, in some patients, LA substrate modification for persistent AF may be necessary in order to increase long-term success rate, adenosine may 'unmask' the reason for recurrence and help to understand durability of conduction block after linear ablation.

Supplementary material

Supplementary material is available at *Europace* online.

Authors' contributions

M.B. and T.D.: data analysis/interpretation, drafting article, critical revision of article, data collection. A.C.: data analysis/interpretation, critical revision of article, data collection, J.C.G.: data analysis/interpretation, drafting article, critical revision of article.

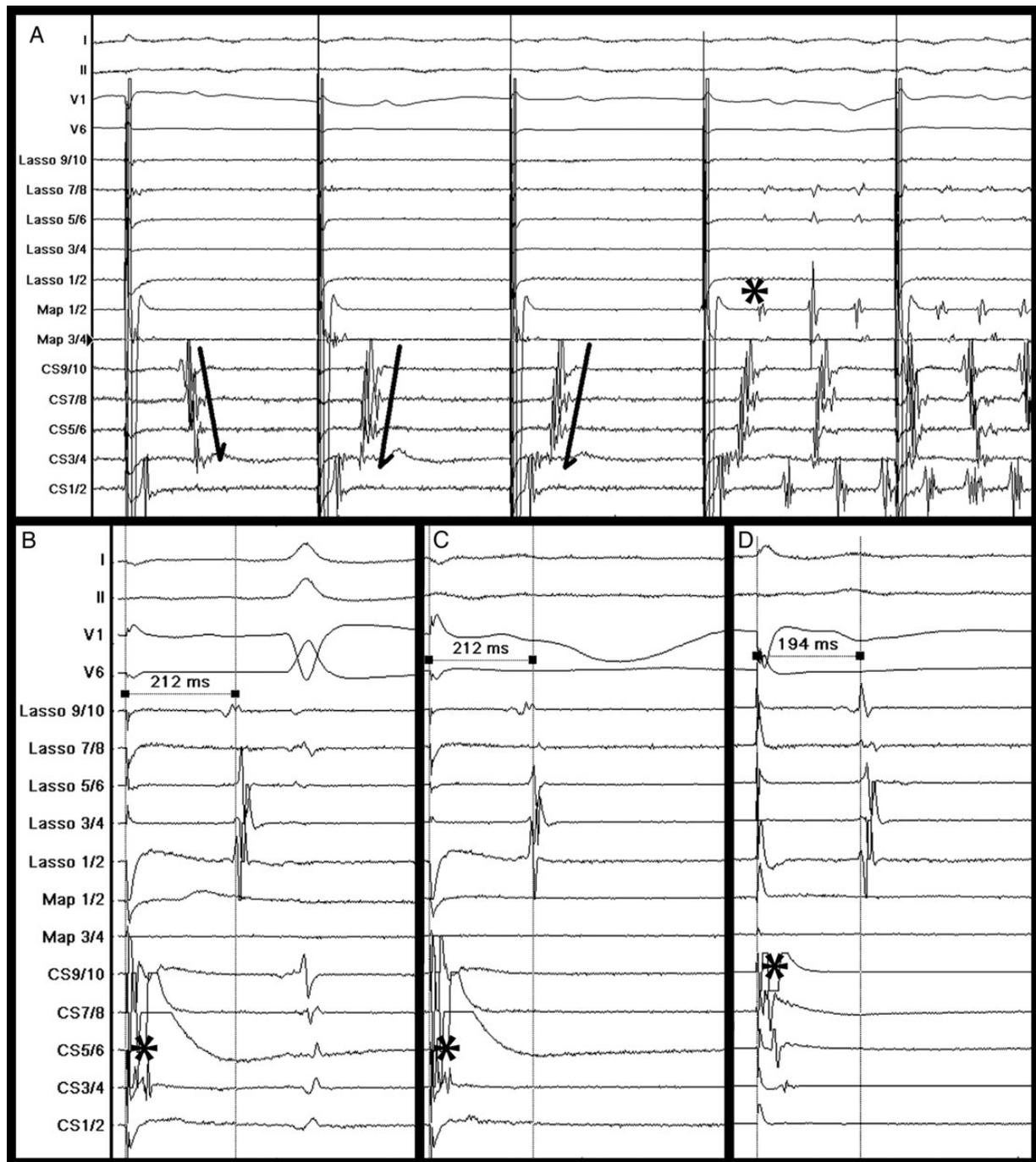


Figure 1 Adenosine injection (30 mg) leads to recovery of conduction over the mitral isthmus (inversion of the activation sequence in the coronary sinus, CS) and, after the third beat atrial fibrillation starts (*, A). The Lasso™, ablation, and CS catheter are placed in the left superior pulmonary vein, anterior wall of the left atrium and distal CS across the inferolateral mitral isthmus line, respectively. After radiofrequency ablation, stability of block is proved with adenosine test (pacing from electrode 5/6 of the CS catheter – B vs. C) and adequate differential pacing (electrode 9/10 of the CS, E) during drug administration is also confirmed. Lead I to V₆, surface ECG; Map, Lasso, and CS, intracardiac recordings from the ablation, the Lasso™, and CS, respectively. Paper speed 67 mm/s (A), 100 mm/s (B–D).

Conflict of interest: M.B. has received speaker fees from St. Jude Medical. J.C.G. is a consultant for St. Jude Medical, Biosense Webster, Boston Scientific and Medtronic, and has received speaker fees from St. Jude Medical, Boston Scientific and Medtronic.

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