To catheterize the CS, a sheath is used in first intention in 81%. CS angiography is performed in 90%, with an inflated balloon in 59%

In case of atrial fibrillation with CHA2DS2-Vasc=2, 38% implant without VKA interruption, 57% stop VKA without substitution, and unfractionned heparin (UH) or low weight heparin (LWH) substitution is chosen in 5%, vs respectively 69% and 11% and 19% if CHA2DS2-Vasc >4.

Conclusion: Most of implantations are performed under local anesthesia. Left sided is preferred, especially in case of CRT-D implantation. Most physicians combine the venous accesses, start with the RV septal lead, and perform a CS angiogram via an inflated balloon. In AF patient, VKA interruption is preferred in low risk patients but not in high risk ones. Few implanters choose VKA substitution.

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Routine face-profile fluoroscopic screening may be useful for earlier detection, monitoring and management of externalized conductors in patients implanted with Riata leads
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Increased rates of structural abnormalities have been reported in the Riata family of implantable cardioverter-defibrillator (ICD) leads. The reliability of defibrillation leads with insulation damage, or abraded cables that are not immediate cause of failure is unknown. The incidence of these defects can be underestimated due to the absence of abnormal electrical parameters detected by regular ICD interrogation. Little is known about the time lag for emergence of functional abnormalities in such leads.

Methods: Forty eight patients who received small-caliber leads of the Riata family (models 1570, 1572, 1580, 1582, 7000, 7002) in our institution between May 2002 and March 2008 were systematically called for an additional visit including face-profile thoracic fluoroscopy to ascertain integrity of the leads followed by routinely repeated fluoroscopies at every six-month visits. We assessed the prevalence of insulation defects with externalized cables and their relation with adverse events requiring lead revision. We tried to evaluate the time lag for emergence of their electrical dysfunction.

Results: Thirty five patients, mean age=64±10 years, with at least 7-month completed fluoroscopy follow-up were included in analysis. After 55-month mean follow-up, 57% of patients completed 2 or more spaced fluoroscopies. Externalized conductors were identified in 7 (20%) patients after a 48-months (12-114) mean delay and in 4 (11%) cases the images were classified as borderline. Three of 7 patients with insulation damage required lead replacement for the reason of clinical adverse events. In two other patients an increase of abrasion line was observed on repeated fluoroscopy without changes in electrical parameters.

Conclusion: Insulation damage with externalized conductors was identified in 20% of patients implanted with Riata leads. Routine fluoroscopic screening allowed earlier detection of these defects and may be useful for their closer monitoring and better management.

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Is myotonic dystrophy part of the Brugada syndrome?
Results of ajmaline challenge in Steinert disease
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Introduction: Both type 1 Myotonic Dystrophy (Steinert disease) and Brugada syndrome may be complicated by conduction disturbances and sudden death. ST elevation in the right precordial leads is the hallmark of Brugada syndrome but may be seen in some myopathies. Mutations in DMPK gene in Steinert pts may lead to cytosolic accumulation of mutated RNA or altered alternate splicing of some RNA potentially causing sodium channel dysfunction. The prevalence of Brugada ECG pattern in Steinert disease is unknown.

Methods: we perform ajmaline challenge test (1 mg/kg over 5 min) during electrophysiologically (EP) testing in a population of 44 Steinert disease pts (27 men, 41±15 years old) without ST elevation at baseline. Left ventricular EF was normal in each case. The presence of type 1 ST elevation (> 2 mm J elevation with coved ST and negative T wave) after ajmaline challenge was correlated to clinical, ECG and electrophysiological variables.

Results: 8 pts (18%) present type 1 ST elevation in the right precordial leads after ajmaline infusion. Brugada pattern was more often seen in men: 7/27 (26%) vs 1/17 (6%) (p=0.09). Patients with negative ajmaline test presented more often with fascicular block: 13/35 (27%) versus none (p=0.03).

Brugada pattern was not correlated to age, symptoms, PR interval, QRS QT or QTc durations, HV interval (at baseline or after ajmaline), presence of bundle branch block, of late potentials at SA-ECG or inducibility of ventricular arrhythmias at EP study.

Nine pts were implanted with a pace maker and four with an ICD. Significant or symptomatic bradycardia did not happen in any non implanted pts, while only one pt presented with malignant ventricular arrhythmias during the 6.3±2.6 years follow-up (ventricular fibrillation with hypokalemia in an ajmaline negative pt).

Conclusion: Brugada ECG pattern can be elicited by class 1 drug in 18% of Steinert disease pts and especially in men. Presence of type 1 ST elevation under class 1 drug in Steinert disease do not seem to have some significant clinical or ECG correlations.

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A simple method to implant epicardial AICD using two separated coils
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Purpose: Surgical epicardial AICD implantation has been quite abandoned since development of endocardial leads and decrease in AICD size. Epicardial patches did not improve since 30 years, are rigid and need to be sewed to the epicard which favors bleeding. In some rare cases, epicardial AICD implantation remains indicated. Therefore we tested a new simple implant technique in patients undergoing open chest surgery through a median sternotomy.

Methods: In 3 cases surgery was performed for percutaneous infected lead extraction failure. The two other patients required surgical operation and AICD implantation (one for mitral valve regurgitation, one for tricuspid repair). A screw-in bipolar pace/sense lead (St Jude Myoxid™TM (1084T) was placed on the right ventricular free wall and two defibrillation leads (Medtronic Transven®6937) were respectively sewed on the pulmonary artery trunk and on the diaphragmatic wall of the right ventricle and then connected to an AICD (Biotronik Lumax 540) placed in an abdominal pocket.

Results: Best defibrillation configuration (31 J tested) was obtained with a shock delivered between the two coils (AICD passive), from the inferior one to the superior one. There were no major cardiovascular complications. Clinical and telemonitoring follow up (mean 10 months) showed stable ventricular stimulation and detection thresholds as well as lead and coil impedances. No arrhythmias occurred.

Conclusions: Epicardial AICD implantation is feasible in a simple way. Long term follow up is needed to confirm post operative results.

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Pacemaker patients’ perception of daily life activities and medical follow-up: a french survey
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