A 30-year-old man with a history of surgical repair of tetralogy of Fallot (TOF) and percutaneous replacement of a pulmonary valve (Melody, Medtronic, Minneapolis, MN, USA) was admitted to our Emergency Department due to symptomatic palpitations. The 12-lead electrocardiogram showed a monomorphic ventricular tachycardia (VT) with a heart rate of 160 b.p.m., inferior axis and right bundle branch block morphology. Tachycardia resolved after intravenous administration of metoprolol 5 mg and the patient was scheduled for a radiofrequency catheter ablation (RFCA) procedure. Four years before, prior RFCA for recurrent VTs was performed. Of note, the patient repeatedly refused an implanted cardioverter-defibrillator. At the index procedure, a macro-reentry mechanism for the VTs was found and a linear ablation at the anatomical isthmus between the right ventricular outflow tract (RVOT) and the tricuspid valve successfully treated the arrhythmia. Pre-procedural cardiac workup, including cardiac multislice computed tomography (MSCT), was performed. At the electrophysiological study, programmed ventricular stimulation failed to induce any arrhythmia and the clinical VT was reproducible only during infusion of high dose isoproterenol. An activation map, performed with the ablation catheter in a point-by-point fashion, showed an ‘early-meets-late’ pattern at the anatomical isthmus $1.1$ However, no mid-diastolic potentials and only half of the VT cycle length was recorded, thus suggesting a focal mechanism and a line of block, due to the previous RFCA (Supplementary material online, Video). The site of origin (SOO) was localized at the septal aspect of the prosthetic pulmonary valve (local activation $-17$ ms to QRS begin, QS unipolar), VT overdrive pacing from the SOO resulted in a very similar QRS morphology with short stimulus to QRS and post-pacing interval. Radiofrequency energy delivery at this site was not possible because of abrupt impedance drop due to intermittent contact of the ablation catheter with the valve struts (Panel A). Arrhythmia elimination was achieved from the right coronary cusp (Panel B).
site and no angiography was performed. Radiofrequency energy application promptly terminated the VT and rendered the arrhythmia non-inducible, also after drug challenging. A gradual impedance drop of 10 Ω was observed during the successful lesion. The similar bipolar local activation time and the QS morphology of the unipolar signal on both right and left outflow tract suggested an intramural origin. No procedure-related complications have been encountered. In a 6 months of follow-up, no VT recurred. Macro-reentry VT circuits associated with a surgical TOF repair have been well described. Nevertheless different arrhythmia mechanism, such focal or micro-reentry mechanism, even if more rare, can be encountered. The presence of scar and/or low-voltage area due to previous ablation procedure or to the repair itself could render the understanding of the arrhythmia mechanism challenging. In our case, the presence of the prosthetic pulmonary valve made an intramural substrate inaccessible from the RVOT. Pre-procedural imaging and the use of a 3D-electroanatomical mapping system provided a good understanding of cardiac anatomy and allowed successful ablation from the RCC.

**Supplementary material**

Supplementary material is available at Europace online.

**Conflict of interest:** none declared.

**Reference**