initiated by VE originating from the RVOT with (26,27) or without (28) eliminating targeting VE. In the latter case report, RFCA was considered to alter or remodel arrhythmic substrates to maintain the PVT. Our RFCA targeting for the initiating VE with additional applications around the origin of initiating VE might eliminate both arrhythmogenic triggers and substrate for VF and/or PVT in this study.

Study limitations. First, the 16 patients showed spontaneous VF/PVT in our series, whereas the 85 patients had only monomorphic VT. This may give the impression that polymorphic RVOT-VT is present in 16% of patients with arrhythmias originating from the RVOT. However, this large percentage probably represents a referral bias, because patients with polymorphic RVOT-VT are more likely to be hospitalized and more likely to be referred for RFCA, whereas patients with monomorphic RVOT-VT are more likely to be treated conservatively as outpatients.

Second, VF was induced in only one patient after RFCA, whereas patients with idiopathic VF usually have high VF inducibility rates. The low rate of VF induction is probably associated with the result of our stimulation protocol.

Third, among the five patients with spontaneous VF, only one patient received ICD after RFCA. The ICD as therapeutic backup is particularly important for patients with spontaneous episodes of VF regardless of the success of RFCA. The ICD was not available in Japan when three of the five patients were admitted to our center and underwent RFCA. The remaining patient, a young woman, refused to receive an ICD after successful RFCA.

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