The R'' wave in V1 and the terminal QRS vector in aVF combine to a novel 12-lead ECG algorithm to identify slow conducting anatomical isthmus 3 in patients with tetralogy of Fallot

J. Wallet1, Y. Kimura1, N.A. Blom2, S. Man1, M.R.M. Jongbloed3, K. Zeppenfeld1

1Leiden University Medical Center, Department of Cardiology & Willem Einthoven Center of Arrhythmia Research and Management & CAHAL, Leiden, Netherlands (The)
2Leiden University Medical Center, Department of Pediatric Cardiology & CAHAL, Leiden, Netherlands (The)
3Leiden University Medical Center, Department of Cardiology, Department of Anatomy & Embryology, and CAHAL, Leiden, Netherlands (The)

Funding Acknowledgements: Type of funding sources: Foundation. Main funding source(s): We acknowledge the support from the Netherlands Cardiovascular Research Initiative: An initiative with support of the Dutch Heart Foundation and Hartekind, CVON2019-002 OUTREACH.

Background: Patients with repaired tetralogy of Fallot (rTOF) have an increased risk for ventricular tachycardia (VT), with slow conducting anatomical isthmus 3 (SCAI 3) as the dominant VT substrate. Right bundle branch block (RBBB) is common in these patients. SCAI 3 can lead to additional activation delay affecting the terminal RV activation.1 We hypothesize that these alterations can be detected by sinus rhythm (SR) 12-lead ECG.

Purpose: To develop a novel algorithm to non-invasively identify SCAI 3 with the SR 12-lead ECG in rTOF patients with RBBB.

Methods: Consecutive rTOF patients aged ≥16 years old with RBBB who underwent electroanatomical mapping (EAM) at our institution between 2017-2022, and 2010-2016, comprised the derivation, and validation cohort, respectively. 12-lead ECGs in SR were analyzed for presence of R'' wave in V1 (any positive deflection after R' in V1) and for duration of a negative terminal QRS portion (NTP) in aVF (ms between QRS complex traversing the isoelectric line from positive to negative vector until QRS offset) (Figure, A).

Results: Forty-six rTOF patients (aged 40±15 years, 70% male) comprised the derivation cohort. Patients were repaired at a median age of 3.3 [IQR 1.2-6.8] years via a ventricular incision in 21 (55%). Mean QRS duration was 165±23ms and 12 (26%) had a QRS duration of ≥180ms. SCAI 3 was detected in 31 (67%) patients and 15 (33%) had a normal conducting AI 3. Monomorphic sustained VT was inducible in 16 (35%). Among patients with SCAI 3, 17 (55%), and 16 (52%), had an R'' in V1, and NTP ≥80ms in aVF, respectively, compared to only 1 (7%), and 1 (7%) patients with normal conducting AIs. Of the 24 patients with an R'' in V1 and/or NTP ≥80ms in aVF both remained independently predictive of SCAI 3 (OR 15.4, p=0.017, and OR 10.0, p=0.042, respectively).

Combining R'' in V1 and NTP ≥80ms in aVF into a diagnostic algorithm (Figure, B) yielded a sensitivity of 71% and specificity of 87% in detecting SCAI 3. In the validation cohort (n=34, age 44±14 years, 19 (56%) with SCAI 3 and 15 (44%) with normal conducting AI 3) the diagnostic algorithm had a sensitivity of 84% and specificity of 80% for a SCAI.

Conclusion: A diagnostic algorithm including R'' in V1 and NTP ≥80ms in aVF had excellent sensitivity and specificity in detecting SCAI 3 in rTOF patients with RBBB in SR. These ECGs characteristics likely represent a delayed terminal RV activation directed towards the lateral RVOT caused by slow infundibular conduction. Whether this algorithm can contribute to non-invasive risk stratification for VT in a general rTOF population requires additional studies.