Clinical Research in Cardiology (2019) 108:110–113
https://doi.org/10.1007/s00392-018-1330-3

LETTER TO THE EDITORS

Butterfly and reverse butterfly: usefulness of a resistance band to provoke exercise-induced arrhythmias during catheter ablation in a patient refractory to pharmacological stimulation

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Received: 16 March 2018 / Accepted: 16 July 2018 / Published online: 26 July 2018

Keywords Resistance band · Ventricular tachycardia · Physical exercise · Adrenergic stimulation · Right ventricular outflow tract · RVOT · Exercise induced arrhythmias · Case report

Sirs:

Ventricular arrhythmias arising from the right ventricular outflow tract (RVOT-VA) are a frequent finding in young physically active patients [1]. Catheter ablation represents a widely accepted therapeutic option for patients suffering from symptomatic RVOT-VA. However, the exact localization and ablation can be hindered by a low arrhythmia burden during the ablation procedure. In these particular cases, pharmacological stimulation may help increasing the number of RVOT-VA during the procedure. However, even extensive pharmacological stimulation may fail inducing the clinical arrhythmia. Elastic exercise bands (resistance bands) are widely used for strength training in healthy individuals as well as in patients recovering from surgery or phases of prolonged inactivity [2]. Resistance bands are inexpensive and readily available, and exercise can be performed in almost any body position including the supine position. In this case, we tested whether intraprocedural exercise using a resistance band might help provoking RVOT-VA in a patient refractory to pharmacological stimulation.

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We report a case of a 32-year-old male patient that was referred to our center for ablation of highly symptomatic non-sustained monomorphic ventricular tachycardia. Arrhythmia episodes occurred during or shortly after strenuous physical exercise. Only recently, he was forced to stop his triathlon training due to palpitations accompanied by dizziness. As expected, treadmill exercise ECG prior to ablation provoked repetitive monomorphic ventricular runs of up to 12 beats. Arrhythmia burden during rest was rather low with 100–200 premature ventricular beats per day. Prior to admission, the patient had undergone two unsuccessful ablation attempts within the right ventricular outflow tract at an external center. At the time of the procedure the patient was in excellent physical conditions. Invasive electrophysiological study was performed without continuous sedation in the awake state. At the beginning of the procedure no spontaneous ventricular activity could be observed. Pharmacological stimulation was started with a 0.5 mg atropine bolus that was followed by repeated doses of orciprenaline up to a cumulative dose of 0.5 mg. However, even additional intravenous infusion of 200 mg theophylline was not effective in provoking the clinical arrhythmia. Considering the reported association between RVOT-VA and exercise, we next decided to induce physical stress. For this purpose, the patient was asked to repetitively stretch a resistance band (Pinofit^{R} green, PINO Pharmazeutische Präparate GmbH, Hamburg, Germany) (Fig. 1). For the first exercise, the patient freed his arms from under the sterile drape and lifted them upwards. The resistance band was grabbed with both hands and the patient was asked to pull laterally ('reverse butterfly') (Fig. 1a_I and a_{II}). For the second exercise ('butterfly'), the resistance band was wrapped around the case of the X-ray tube underneath the table and the patient was asked

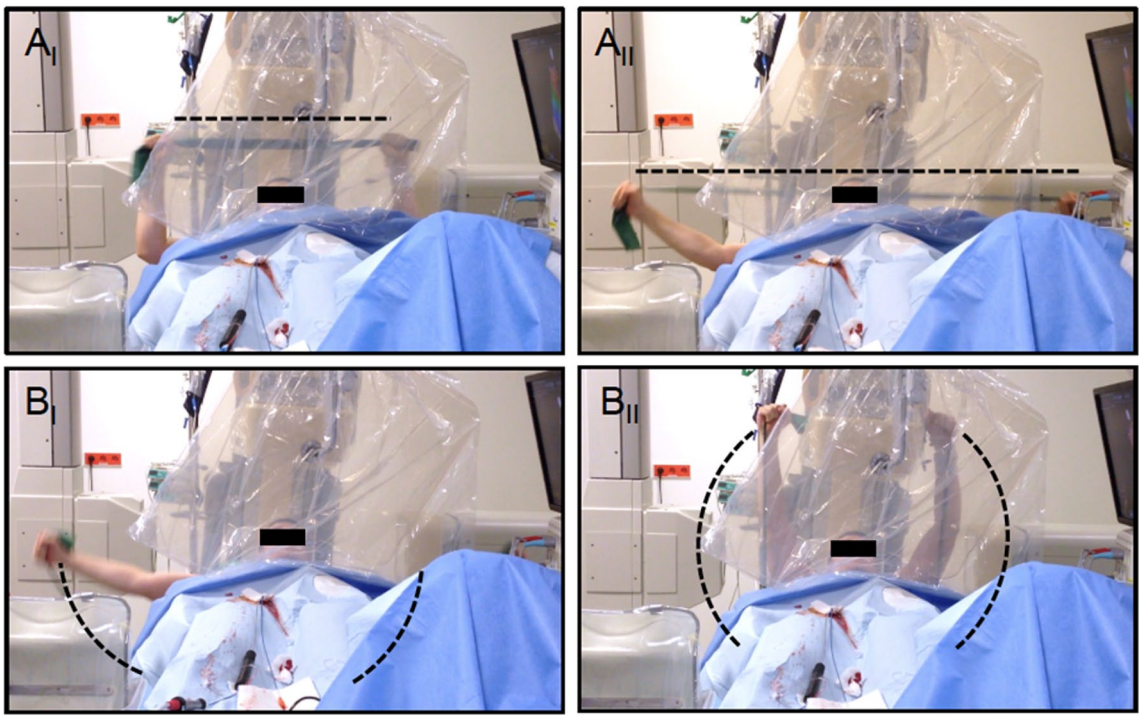

Fig. 1 **a** Intraprocedural exercise training using a resistance band in 'reverse butterfly' technique. The patient is positioned in the supine position. Both arms pull laterally to stretch the band from the relaxed ($\mathbf{a_I}$) to the tensed ($\mathbf{a_{II}}$) position. **b** For the 'butterfly' technique, the resistance band is wrapped around the case of the X-ray tube underneath the table. Both arms pull upward from the relaxed ($\mathbf{b_I}$) to the tensed ($\mathbf{b_{II}}$) position

to push toward the ceiling (Fig. 1b_I and b_{II}). Interestingly, after 4 min of physical exercise using the resistance band in 'reverse butterfly' and 'butterfly' technique, the patient developed a brief episode of ventricular runs strongly resembling the clinical arrhythmia (Fig. 2a, b). As the arrhythmia burden was too low for activation mapping, we decided to perform a systematic pace-mapping approach using the first QRS complex (Fig. 2b, #) as the matching template. The anatomy of the right ventricle was carefully reconstructed using a three-dimensional electroanatomic mapping system (CARTO3™, Biosense Webster, Diamond Bar, CA, USA). Pace-mapping was then performed at various sites within the right outflow tract and the similarity between the template, and the induced QRS complex was calculated using the built-in automated template matching algorithm (Labsystem™ Pro, Boston Scientific, Marlborough, MA, USA). The best and poorest match was 98 and 61%, respectively. By simply converting the degree of similarity into negative local activation times (e.g., 98% → −98 ms), we manually created a three-dimensional pace-map with red indicating best and purple reflecting lowest similarity (Fig. 2c, d). Radiofrequency energy was delivered at the site of best match (red sphere in Fig. 2c, d) and the physical exercise was repeated. Interestingly, despite repeated strenuous physical exercise, no more ventricular arrhythmias could be observed. After successful ablation, only a single premature ventricular beat could be detected by Holter ECG within a recording time of 19 h. Furthermore, no single ventricular premature beat could be provoked by treadmill exercise test up to 200 W. During the follow-up of 9 months, the patients were free from symptoms and therefore decided not to repeat Holter ECG or treadmill test.

RVOT-VA are the most common ventricular arrhythmia among young adults. As indicated by a recent meta-analysis, catheter ablation successfully reduces the number of premature ventricular contractions and improves cardiac function in patients with reduced ejection fraction [3]. However, it is well recognized that catheter ablation may be challenging in cases where programmed electrical stimulation or pharmacological stimulation fails to induce the clinical arrhythmia [4]. In their study, Wang et al. found that non-inducibility was present in 21 of 148 patients (14%) [4]. Triggering factors of RVOT-VA have been shown to be sex specific with hormonal flux playing the major role in female patients and exercise or stress being the dominating factor in male patients [5]. Interestingly, progesterone administration has already been successfully used to provoke ventricular arrhythmias in a female patient suffering from left fascicular tachycardia refractory to isoproterenol infusion [6]. Hasdemir and co-workers found that high-frequency stimulation

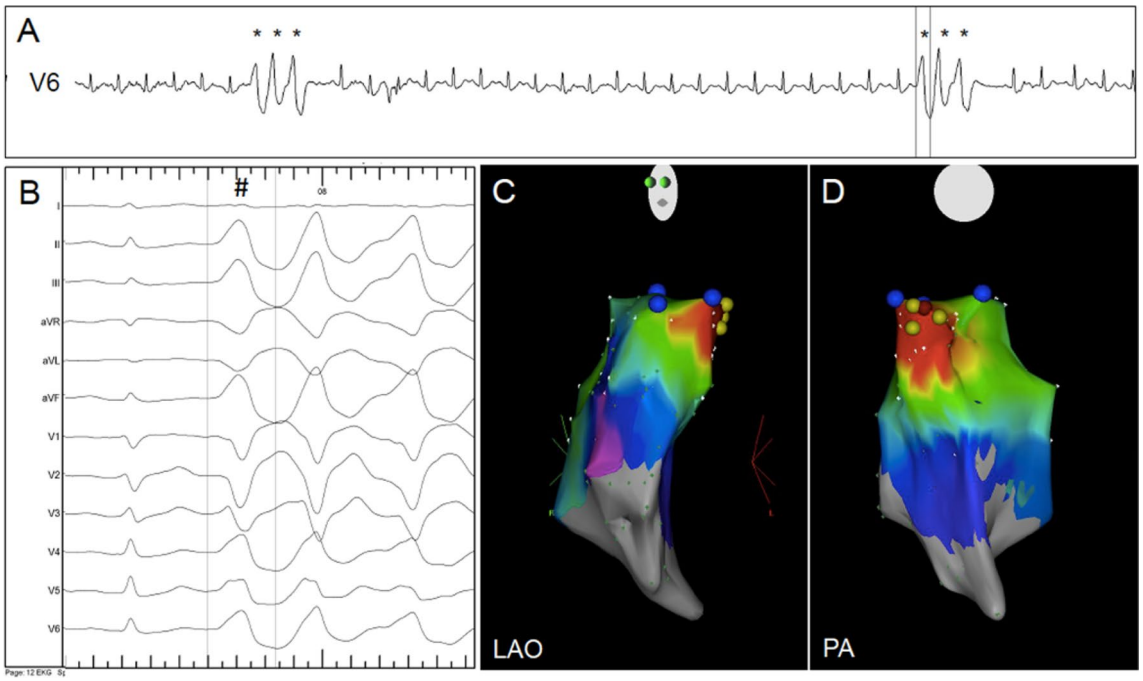

Fig. 2 **a** Single-lead electrocardiogram (V_6) showing two short ventricular runs (*) induced by 4 min of physical exercise using the resistance band. **b** 12-lead ECG recording suggests an origin within the right ventricular outflow tract. The first QRS complex (#) of the second run was used for further systematic pace-mapping. **c, d** Electroanatomic maps of the right ventricular outflow tract showing the quality of pace-map in a color-coded manner. For this purpose, the degree of similarity between the template and the pacing-induced QRS complex was converted into negative local activation time (e.g., 98% → − 98 ms). The blue spheres mark the pulmonary valve, the yellow spheres mark the best pacing sites, and the red sphere marks the site of catheter ablation

within the left pulmonary artery increases the sympathetic input and provokes RVOT-VA [7]. These findings are in line with previously published data showing that sympathetic predominance precedes RVOT-VA [8]. Considering that physical exercise is a simple trigger of sympathetic activity, it is tempting to speculate that intraprocedural exercise might help increasing the percentage of male patients with inducible RVOT-VA. The exact mechanism of how physical activity increases RVOT-VA and why sometimes even better than catecholamine administration is largely unclear. A possible explanation might be given by Zhou et al. speculating that increased sympathetic innervation of the RVOT creates a localized arrhythmogenic focus that is further subject to stimulation by circulating catecholamine [9]. This hypothesis combines classical theories of c-AMP mediated and calcium-dependent mechanisms of triggered activity with an additive influence of sympathetic innervation and might be a good explanation for our observed effects. The reason why the RVOT-VA burden occasionally decreases in some patients when entering the electrophysiology laboratory remains unclear. A possible explanation could be the suppression of automatic activity by sinus tachycardia.

To the best of our knowledge, intraprocedural exercise using a resistance band to provoke RVOT-VA has not been published yet. The 'butterfly' and 'reverse butterfly' exercise using a resistance band is simple and, in our experience, can even be safely performed by older patients. Of note, other exercise techniques, such as handgrip exercise, have not been analyzed in our laboratory. However, it is tempting to speculate that the induction of RVOT-VA is rather dependent on the total amount of physical stress than on the particular way it is applied. Another possible approach to target the RVOT-VA could have been to perform a 12-lead Holter ECG and keep the exact position of the electrodes for the mapping procedure.

Taken together, this case demonstrates the usability and additive value of physical exercise using a resistance band in provoking RVOT-VA in a patient refractory to pharmacological stimulation. The reported technique may also be a helpful technique in facilitating other arrhythmias that are dependent on adrenergic stimulation, such as AV nodal or atrio-ventricular reentrant tachycardia.

Compliance with ethical standards

Conflict of interest On behalf of all the authors, the corresponding author states that there is no conflict of interest.

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