Response to Letters Regarding Article, “Clinical Management of Catecholaminergic Polymorphic Ventricular Tachycardia: The Role of Left Cardiac Sympathetic Denervation”

We are glad to have the opportunity to respond to the points kindly raised by Drs Patanè and Gow in reference to our recent article on catecholaminergic polymorphic ventricular tachycardia (CPVT).1

We are obviously aware of the article by Faggioni et al,2 which included 1 of the coauthors of our article, Dr Wilde. We also have direct experience with a few CPVT patients who had a reduction or disappearance of the ventricular arrhythmias induced by exercise when heart rate increased further (usually to >140 bpm). We have not discussed atrial overdrive pacing simply because we do not believe that these data and observations have direct relevance to the ongoing clinical management of CPVT patients in everyday life. The concept of atrial high-rate pacing (>120 bpm) may be of potential value in the in-hospital management of emergencies, but we find it difficult to recommend that heart rate should be increased permanently, or during exercise, to >140 bpm. In addition, how would it be possible to exclude that such an intervention, activated in the out-of-hospital setting, would not precipitate ventricular tachycardia or ventricular fibrillation? Our article was focused on how to reduce the incidence of life-threatening arrhythmias in the safest way. Rate support of CPVT patients who have symptomatic bradycardia in response to therapeutic β-blockade is always a consideration in CPVT.

We acknowledge the comments concerning the possibility of anatomic variations in the cervicothoracic ganglion. Dr Gow has downplayed the statements by a recognized expert in the field (Andrew Armour),3 who wrote that “all major cardiopulmonary nerves were found to arise from the stellate ganglia” and focused on the conclusions by Marcel et al4 and Pather et al,5 largely based on embalmed cadavers and on fetuses, that in ≈20% of cases the inferior cervical ganglion and the first thoracic ganglion were separate. In our personal experience of >40 years in hundreds of patients in whom we performed left (and sometimes right) cardiac sympathetic denervation, we have always found the stellate ganglion to be the fusion of the last cervical and first thoracic ganglia. The current video-assisted thoracoscopic approach allows a clear and comprehensive visualization of the anatomy and excludes the possibility of missing a significant anatomic variant.

Another part of Dr Gow’s letter is clinically relevant. He suggests a correlation between the possibility of anatomic variations of the stellate ganglion and the recurrence of syncope, and very seldom of cardiac arrest, in CPVT patients (and in patients with long QT syndrome) treated with left cardiac sympathetic denervation. Although we consider the issue of anatomic variability of minor importance in favoring the occurrence of arrhythmia recurrences, the deliberate lack of removal of either T4 or of the lower part of the stellate ganglion (T1), to exclude any risk of Horner syndrome, is a true concern. Patients with intentionally incomplete denervation did show a much higher risk of recurrences than patients with complete denervation in our study.1 Should a functionally incomplete denervation be frequent because of anatomic variability, one would expect a higher frequency of recurrences. Be that as it may, an arrhythmia recurrence does not necessarily imply that the left-sided denervation was incomplete. The right cardiac sympathetic nerves also release norepinephrine in the ventricles, and their contribution may be important in some patients; in addition, circulating catecholamines released by the adrenal glands may play a role. Thus, to explain incomplete arrhythmia protection, there is no need to invoke anatomic variability. The suggested possibility of a “further surgical exploration of the same side” does not consider the growth of connective tissue after surgery.
Correspondence

Michael Eldar, MD, FAHA
Heart Institute, Leviev Heart Center
Sheba Medical Center
Sackler School of Medicine
Tel Aviv University
Tel Hashomer, Israel

Maria Kharlap, MD
Department of Clinical Cardiology and Molecular Genetics
National Center for Preventive Medicine
Ministry of Healthcare
Russian Federation
Moscow, Russia

Asaad Khoury, MD
The Bruce Rappaport Faculty of Medicine
Technion
Haifa, Israel

Andrew D. Krahn, MD
Division of Cardiology
University of British Columbia
Vancouver, Canada

Antoine Leenhardt, MD
AP-HP, Hôpital Bichat
Service de Cardiologie et Centre de Référence des Maladies Cardiaques Héritées, Paris
Université Paris Diderot
Sorbonne Paris Cité
Paris, France

Christopher R. Moir, MD
Departments of Medicine, Pediatrics, and Molecular Pharmacology & Experimental Therapeutics
Divisions of Cardiovascular Diseases and Pediatric Cardiology
Windland Smith Rice Sudden Death Genomics Laboratory
Mayo Clinic, Rochester, MN

Attilio Odero, MD
Division of Vascular Surgery
Fondazione IRCCS Policlinico San Matteo
Pavia, Italy

Louise Olde Nordkamp, MD
Heart Centre AMC
Department of Cardiology
Academic Medical Centre
Amsterdam, The Netherlands

Thomas Paul, MD
Department of Pediatric Cardiology and Intensive Care Medicine
University Hospital
Georg-August-University
Göttingen, Germany

Ferran Rosés i Noguer, MD
Royal Brompton Hospital
London, UK

Maria Shkolnikova, MD
Research Clinical Institute for Pediatrics of the Pirogov National Research Medical University
Moscow, Russia

Jan Till, MD
Royal Brompton Hospital
London, UK

Arthur A.M. Wilde, MD, FAHA
Heart Centre AMC
Department of Cardiology
Academic Medical Centre
Amsterdam, The Netherlands
Princess Al Jawhara Albrahim Centre of Excellence in Research of Hereditary Disorders
King Abdulaziz University
Jeddah, Saudi Arabia

Michael J. Ackerman, MD, PhD
Departments of Medicine, Pediatrics, and Molecular Pharmacology & Experimental Therapeutics
Divisions of Cardiovascular Diseases and Pediatric Cardiology
Windland Smith Rice Sudden Death Genomics Laboratory
Mayo Clinic, Rochester, MN

Peter J. Schwartz, MD, FAHA
Center for Cardiac Arrhythmias of Genetic Origin
IRCCS Istituto Auxologico Italiano
Milano, Italy

References


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