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How to reduce the risk of residual shunt during percutaneous treatment of ventricular septal defects. Response

Cómo reducir el riesgo de cortocircuito residual durante el cierre percutáneo de comunicación interventricular. Respuesta

To the Editor,

We were pleased to receive the comments by Fanyan Luo and Haison Bu regarding our study.[1] They draw attention to the relevance of measuring the diameter and velocity of the jet in patients with residual shunt after ventricular septal defect closure using the NitOcclud device because of the risk of hemolysis, and highlight as key aspects a diameter of 2 mm or a velocity of at least 3 m/s.

In our experience, the nature of the shunt should be determined to minimize the risk of hemolysis. If the shunt is inside the device (between its coils), jet velocity is damped and is typically low, making it easier to disappear over time. To encourage this situation, we also delay the start of salicylate administration for 2 weeks after implantation. On the other hand, if there is an external shunt, between the external coil and the wall of the anatomical defect, shunt velocity is high and the risk of mechanical hemolysis is greater. If this phenomenon is detected before release, we try to reposition the NitOcclud device itself and reassess the situation, confident that, once repositioned, its position will change little during the release maneuver due to the virtual absence of tension during this maneuver. As a second option, we oversize the original device and retry the implant procedure. If residual shunt persists after release, we consider placing a second closure device, either a second smaller NitOcclud device, other soft expandable nitinol devices (ADO II or ADO II AS), or controlled release coils (Gianturco coil). Therefore, in cases of external shunt, we believe that the risk is higher and should be minimized to the greatest extent possible, and if this is not achieved, surgical closure should be considered.

We studied this problem with the manufacturer of NitOcclud, and suggested extending the position of the polyester fibers in the portion of the device that is finally positioned toward the left ventricular cavity. However, this proposal would need the sheath profile to be extended, and other possible effects of this design modification would have to be analyzed.

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AUTHORS' CONTRIBUTIONS

Both authors wrote and reviewed this article.

CONFLICTS OF INTEREST

J.L. Zunzunegui is proctor for PFM (NitOcclud Lê VSD Coil).

Ruth Solana-Gracia[a,b,*] and José Luis Zunzunegui[b,c]

[a]*Unidad de Cardiología Infantil, Hospital Universitario Infanta Leonor, Madrid, Spain*
[b]*Instituto de Investigación Sanitaria Gregorio Marañón, Madrid, Spain*
[c]*Unidad de Cardiología Infantil, Hospital Universitario Gregorio Marañón, Madrid, Spain*

[*] Corresponding author:
E-mail address: ruth.solana@salud.madrid.org (R. Solana-Gracia).

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Fever and Brugada electrocardiographic pattern

Fiebre y patrón electrocardiográfico de Brugada

To the Editor,

We have read with interest the scientific letter published by Santiago-Cortés et al.[1] The distinction between Brugada phenocopies and Brugada syndrome can sometimes be a complex diagnostic challenge. This differential diagnosis requires restraint and precision, as these entities differ not only in their etiology, but also in the potential prognostic implications for patients.[1,2]

The scientific letter by Santiago-Cortés et al. describes the case of a 12-year-old adolescent with a type 1 Brugada electrocardiographic pattern in the context of febrile syndrome (pediatric multisystem inflammatory syndrome associated with~SARS-CoV-2) that resolved after stabilization and improvement of the infectious-inflammatory condition.[3] Outpatient provocation testing with flecainide and genetic testing were both negative and, therefore, Brugada phenocopy was diagnosed.[3]

First, we agree with the authors that the overall findings were consistent with Brugada phenocopy, namely, a Brugada electro-