acute heart failure. Percutaneous treatment with dilation of
the coarctation and stenting was decided upon, with good
results.2

In our area, more than 90%–95% of heart failure cases are due to
ischemic heart disease, hypertension, arrhythmias and valvular
disease, but on occasion, as with this patient, the unexpected
may happen. In this case, and contrary to all statistics and initial
data, the sound of hoofbeats were those of a zebra and not of a
horse.

Manuel Anguita Sánchez*, Elena Villanueva Fernández,
Daniel García Fuertes, and José Suárez de Lezo
Servicio de Cardiología, Hospital Universitario Reina Sofía, Córdoba,
Spain
* Corresponding author.
E-mail address: manuelp.anguita.sspa@juntadeandalucia.es
(M. Anguita Sánchez).

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Transapical Technique as an Alternative Approach to
Paravalvular Leak Closure
Vía transapical como alternativa para el cierre del leak
paravalvular

To the Editor,

Paravalvular leaks occur in between 3% and 7% of cardiac valve
prostheses. As many patients in whom closure is indicated are at
high surgical risk, it is often necessary to resort to percutaneous
techniques. Such approaches are complicated by the lack of
specifically designed materials. Nevertheless, results continue to
improve as a result of advances in imaging techniques and the
development of new devices.

Anterograde or retrograde approaches can be used according to
the location of the prosthesis and the leak. Despite the use of stiff
guidewires or looped guidewires to establish arteriovenous loops,
advancing the introducer for the dispensing catheter through the
leak is usually the most complicated step.

We present a case in which a transapical technique1,2 was used
when both anterograde and retrograde approaches failed. This
new technique allowed rapid and straightforward access to the
leak, as well as providing good support for advancement of the
introducer.

The patient had aortic and mitral mechanical valve
prostheses and had been admitted to hospital three times for
acute pulmonary edema. A paravalvular leak estimated by
transesophageal echocardiography (TEE) to be 8 mm high, 6 mm
long, and 4 mm wide was observed in the posteroseptal region
and was associated with severe mitral insufficiency and signs of
hemolysis. As the patient had a logistic EuroSCORE of 32%, a
decision was taken to attempt percutaneous closure. The first
attempt employed an anterograde approach via transeptal
puncture. Despite the use of stiff guide wires, no catheters
could be introduced that would allow positioning of the device. In
the same procedure, a retrograde approach was also attempted.
When the catheter was advanced to the left ventricle through the
aortic valve prosthesis, one of the discs of the prosthesis
was repeatedly blocked, leading to severe hemodynamic deteriora-
tion. A decision was therefore taken to attempt a transapical
procedure.

Forty-eight hours later, the procedure was done under general
anesthesia with TEE guidance. After localization of the apex by
transthoracic echocardiography, a left anterior minithoracotomy
was performed and a tobacco pouch suture prepared. The apex was punctured with a trocar and a standard “J” 0.035 mm guidewire (Bolton Medical Inc. Sunrise, FL, USA) was advanced to place an 8 French introducer (Terumo Medical Corp, Tokyo, Japan). A straight, hydrophilic, 0.035 mm guidewire (Terumo Medical Corp, Tokyo, Japan) was advanced through the leak until it reached the left atrium and then a Vista Brite tip JR 3.5 catheter (Cordis, Miami, FL, USA) was advanced over it. The guidewire was then replaced with a stiff guidewire (Amplatz Super Stiff. AGA Medical. Golden Valley, MN, USA) that allowed the 8 French catheter for the device to be advanced. A Vascular Plug III (AGA Medical. Golden Valley, MN, USA) occlusion device measuring 12 mm × 5 mm was then advanced with the dispensing catheter in the left atrium and when only the distal segment had been opened it was withdrawn towards the left ventricle until it reached the leak, without releasing it. The mitral insufficiency disappeared but the device interfered with one of the discs and a decision was taken to remove the entire system. Another Vascular Plug III occluder measuring 12 mm × 5 mm was advanced and, this time, it was opened completely in the left atrium before inserting it into the leak (Fig. 1). The device did not interfere with any of the discs but left slight residual paravalvular insufficiency. After performing pushing maneuvers and confirming that it was well supported, the device was released (Fig. 2). Finally, the tobacco pouch suture was closed.

During follow-up, echocardiography and direct visualization did not reveal morphologic or functional changes compared with the results obtained immediately following implantation.

The patient was discharged and continued to show clinical improvement.

The transapical technique may speed up, facilitate, and simplify procedures for closure of paravalvular leaks and should be considered in complex cases or in those situations in which percutaneous approaches have failed.

Mariano Larman, Garikoitz Lasa*, Juan Carlos Sanmartín, and Koldo Gaviria

Servicio de Hemodinámica y Cardiología Intervencionista, Policlinica Gipuzkoa, San Sebastián, Guipúzcoa, Spain

* Corresponding author.

E-mail address: garikoitzll@hotmail.com (G. Lasa).

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An Unusual Complication of Exercise Echocardiography

To the Editor,

A 53-year-old woman was referred for stress echocardiography. Her clinical record included a history of high blood pressure and VVIR pacemaker implantation secondary to atrioventricular nodal ablation for symptomatic refractory paroxysmal atrial fibrillation. During the previous 3 months, she reported episodes of precordial oppression and dyspnea triggered by emotionally stressful situations. Exercise echocardiography was indicated because of recurrent pain and the pacemaker rhythm in the electrocardiogram (ECG).

Baseline echocardiography showed concentric left ventricular hypertrophy (12 mm) and a left ventricular ejection fraction (EF) of 67%, with interventricular asynchrony due to pacemaker stimulation without alterations in segmental contractility. At the end of the fifth minute of the Bruce protocol (heart rate 105 bpm [63% of maximum], blood pressure 180/85 mm Hg), the patient experienced intense chest pain. Echocardiography showed septoapical and inferoapical akinesis.

Given the persistent pain, the patient was referred to our center for urgent coronary angiography. The coronary arteries appeared normal. Ventricular angiography showed akinesis of the middle segments of the anterior and inferior wall, apical hypokinesia, basal segment hypercontractility and an EF of 45%–50% without an intraventricular gradient (Fig. 1).

The patient was hospitalized in the coronary unit, where treatment with atenolol was begun. She experienced no new episodes of chest pain. The troponin-T level reached a maximum of 0.19 ng/mL (normal <0.1 ng/mL) in 24 h and normalized at 48 h (0.07 ng/mL); the creatine kinase level was within the normal range. At 24 h, the ECG revealed a negative precordial T wave (Fig. 2). At 1 week, the echocardiogram showed no alterations in left ventricular segmental contractility and the EF had recovered to 65%. Consequently, the patient was discharged with a diagnosis of Tako–Tsubo syndrome.

Tako–Tsubo syndrome—or stress-induced cardiomyopathy—is a clinical entity characterized by the presence of chest pain in the context of a stressful situation, an ECG that mimics ST-segment elevation myocardial infarction, normal coronary arteries, and transitory left ventricular dysfunction due to midapical akinesis or dyskinesis.1 In acute coronary syndromes,2 the estimated prevalence is 0.7%. Knowledge of the pathophysiology is quite limited although it has been suggested that dysfunction of the coronary microcirculation occurs due to the release of catecholamines into plasma triggered by stress.3 However, in some cases, no trigger mechanism has been found.4 In our patient, the only trigger was the physical and emotional stress of undergoing a test for ischemia. The baseline echocardiogram was normal and akinesis appeared during the test. Ventricular angiography performed shortly after this suggested Tako–Tsubo syndrome. In our patient, the dysfunction mainly affected the middle segments, a location that

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Figure 1. Ventricular angiography. Akinesis of the middle segments, apical hypokinesia, and baseline hypercontractility. Diastole (A) and systole (B).