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EDITORIAL

An implantable defibrillator and what else?

Lluís Mont*, Eduard Guasch, and Antonio Berruezo

Thorax Institute, Hospital Clinic, Department of Medicine, University of Barcelona, Institut per la Recerca Biomèdica August Pi i Sunyer (IDIBAPS), Barcelona, Catalonia – Spain

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This editorial refers to 'Recurrence of ventricular arrhythmias in ischaemic secondary prevention implantable cardioverter defibrillator recipients: long-term follow-up of the Leiden out-of-hospital cardiac arrest study (LOHCAT)'†, by C.J.W. Borleffs *et al.*, on page 1621

Once implantable cardioverter defibrillators (ICDs) were proven effective in treating ventricular tachycardias (VTs) and reducing sudden death and total mortality,[1] arrhythmologists felt a kind of relief for a while, a feeling that 'the task has been completed'. That is all an arrhythmologist can do for a VT patient at risk for sudden death. Cardiac resynchronization therapy (CRT) put everybody back in the saddle again, and now large studies have shown that CRT alone or in combination with an ICD also reduces mortality in patients with advanced heart failure and a wide QRS.[2] It seemed that arrhythmologists could take a break again. However, the stabilization of the field was once again an illusion, as new studies such as the report by Borleffs *et al.*[3] send us in new directions. The question is: now that the defibrillator is in place, what else can we do for our patients to improve their quality of life and survival?

Borleff *et al.* analysed which baseline variables in patients receiving a defibrillator for secondary prevention are predictive of recurrent life-threatening ventricular arrhythmias and mortality. The main independent predictors were atrial fibrillation (AF), wide QRS, ejection fraction, and VT as a presenting arrhythmia. Theoretically, these four predictors can be modified at the moment of ICD implantation, and therefore there is plenty of scope for research to demonstrate that taking the necessary steps to do so will further benefit patients in the long run.

The obvious and already proven benefit is to implant a CRT in Class III–IV patients with a wide QRS.[2] However, in the near future, new data will be available from studies of patients in Class II. The INCIDENCE and MADIT III[4] studies are analysing secondary and primary prevention patients, respectively, and may clarify whether better results are obtained from implanting a CRT, rather than conventional ICD devices, in Class II patients with a wide QRS and severe left ventricular dysfunction. The results of the REVERSE trial demonstrated that implanting a CRT at an early stage of the disease (Class I and II) induces reverse remodelling, without definitively establishing any functional benefit.[5] Although several small studies have found potential for CRT in patients with a narrow QRS, until now this has not been proven in a large randomized study. The potential benefit of CRT in patients with a narrow QRS is being explored by the ECHO-CRT trial,[6] which is recruiting patients with severe systolic dysfunction and dyssynchrony, proven by echocardiographic criteria. The aim is to establish whether patients with a narrow QRS could also benefit from CRT therapy.

AF is mentioned in Borleffs *et al.* as the most powerful predictor of life-threatening arrhythmias and death. Despite its association with increased mortality, for many years AF was thought merely to represent a marker of more severe disease, rather than being a cause itself of increased mortality. More recently, epidemiological studies[7] have firmly established the link between increased mortality and AF, after adjusting for relevant covariates such as age, heart disease, etc. Therefore, there is an increasing awareness of the independent negative prognostic value of AF in several clinical situations. For example, AF is independently associated with increased mortality in primary prevention ICD trials[8] and in patients with heart failure, even when treated with CRT.[9]

What are the possible mechanisms that link AF and mortality? On one hand is the worsening of heart failure. Fast, irregular ventricular rhythm and loss of atrial contraction certainly will decrease cardiac output and precipitate heart failure. Furthermore, chronic fast rhythm induces the so-called 'tachycardiomyopathy' with ventricular dilatation and remodelling, mimicking dilated cardiomyopathy. The study by Borleffs *et al.*[3] suggests another interesting hypothesis. In that study, AF was a predictor of appropriate shocks, suggesting that AF plays an important role in inducing ventricular arrhythmias. In fact, several case reports and studies have shown that a fast irregular ventricular rhythm induces ventricular arrhythmias. Therefore, whether through worsening the ventricular remodelling to cause heart failure or by provoking ventricular tachycardia, AF may increase mortality.

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* Corresponding author. Tel: +34 93 227 5551, Fax: +34 93 451 3045, Email: lmont@clinic.ub.es

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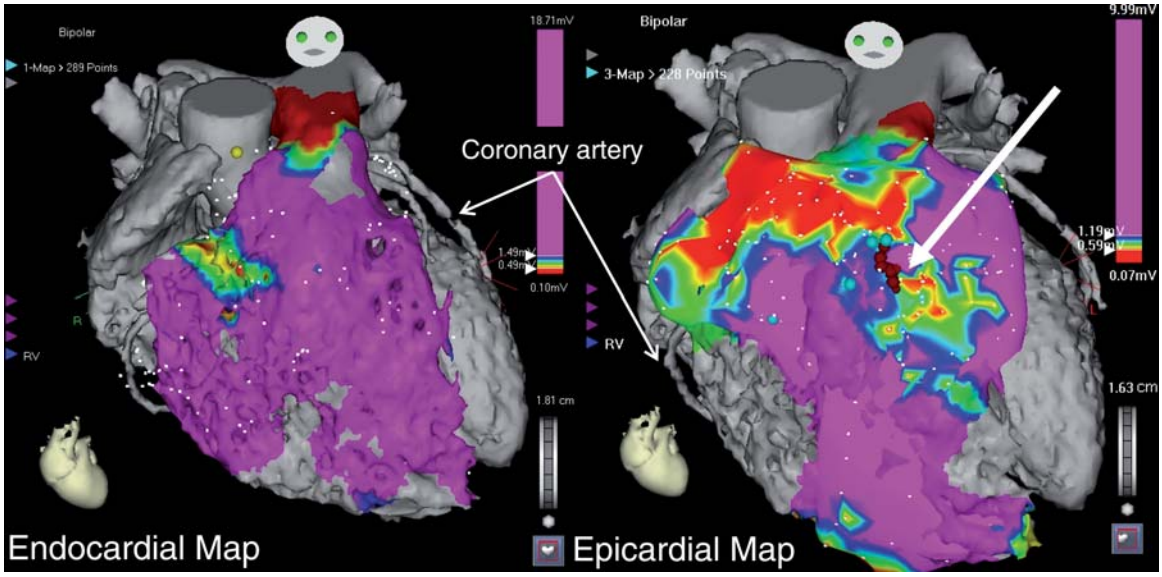

Figure 1 Three-dimensional voltage map in sinus rythm performed with the CARTO sytem and merged with the cardiac CT scan, in a patient with right ventricular dysplasia and multiple shocks due to a repetitive unstable monomorphic sustained ventricular tachycardia (VT) refractory to antiarrhythmic medication. The VT circuit was epicardial (right panel) where the scar tissue was more extensive than in the endocardium (left panel). An ablation line was traced (arrow) closing a conducting channel, rendering the VT non-inducible.

On the other hand, AF ablation has proven effective in suppressing AF and improving ventricular function in selected patients,[10] and, therefore, in theory, may offer an opportunity for improvement in such patients. Both the AMICA trial[11] and that described in Natale *et al.*[12] are attempting to demonstrate a benefit of AF ablation in patients who receive an ICD for primary and secondary prevention or for depressed left ventricular ejection fraction.

Finally, Borleffs *et al.*[3] identify VT at baseline as an independent predictor. It is well known that patients with VT have more severe LV dysfunction than patients presenting with ventricular fibrillation. On the other hand, patients with VT tend to suffer more recurrences than do those presenting with ventricular fibrillation (VF). Therefore, the negative effect of VT on prognosis could be related to a worse baseline profile, but also to a higher number of shocks. In fact, several studies have proven that patients suffering shocks have a worse prognosis, although no casual relationship has been clearly established so far.[13] Repeated VT episodes or shock may have an additive negative effect. On the other hand, ICD shocks (whether appropriate or not) have a negative impact on quality of life. Therefore, any step towards decreasing the number of arrhythmic episodes (ventricular or supraventricular) may in theory have a positive impact on the course of the disease. The recent SMASH VT study[14] has shown that prophylactic VT substrate ablation decreases the number of ICD therapies during follow-up. As a result, perhaps in the near future prophylactic ablation therapy at the time of implantation will be promoted. Other trials are in progress, attempting to demonstrate this hypothesis.[15]

In summary, there are several actions, such as CRT, AF ablation, or VT ablation, that may improve the quality of life and prognosis in patients receiving an ICD for secondary prevention. The results of several ongoing trials may shed more light on the road ahead.

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CARDIOVASCULAR FLASHLIGHT

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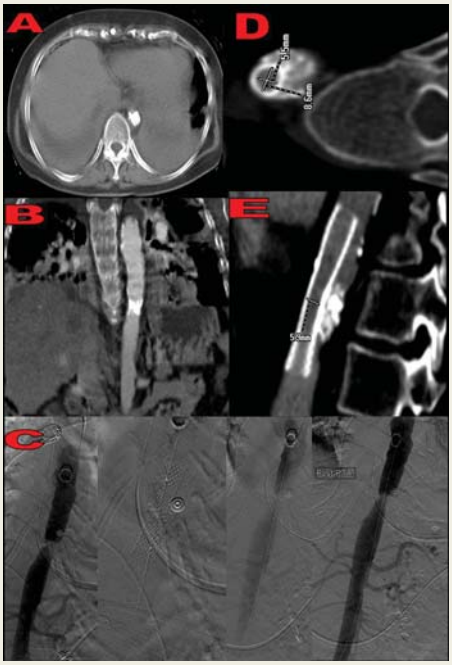
Middle aortic syndrome, severe hypertension, and endovascular repair

Juan José González-Ferrer*, Enrique Balbacid, and Isidre Vilacosta

Hospital Clinico San Carlos, Prof. Martin Lagos s/n, Madrid, Spain

* Corresponding author. Tel: +34 91 330 3149, Fax: +34 91 330 3142, Email: jjgferrer11@yahoo.es



A 55-year-old woman presented to the emergency room with dyspnoea, epigastric pain, and dizziness. She was severely hypertensive. Physical examination revealed a systolic murmur in the left sternal edge (II/VI), bilateral basal rales, and absence of pulses on both lower limbs. The chest radiography supported the diagnosis of acute pulmonary oedema. The 12-lead electrocardiogram showed sinus tachycardia with non-specific changes of repolarization. Echocardiography showed severe concentric left ventricular hypertrophy and low ejection fraction. Thoraco-abdominal CT was performed showing a critical and severely calcified aortic stenosis in the junction between the thoracic and the abdominal segments of the aorta (*Panels A* and *B*). Aortography confirmed the presence of severe stenosis of descending aorta with a gradient of 160 mmHg. The patient underwent urgent percutaneous stent implantation with an acceptable luminal gain and a residual gradient of 30 mmHg (*Panels C*, *D*, and *E*). The patient's haemodynamic condition improved during the next few hours following the endovascular procedure, and she was discharged asymptomatic 1 week later. Three months after hospital discharge, her blood pressure remains normal.

Middle aortic syndrome is a diffuse narrowing of the distal thoracic and abdominal aorta that commonly involves the visceral and renal arteries. This condition typically presents as severe hypertension in young patients who have weak or absent femoral pulses and an abdominal or lower back bruit. The aetiology of the middle aortic syndrome is controversial. The diagnosis of this rare syndrome is made by computed tomography, magnetic resonance imaging, or angiography. The natural history of this rare syndrome is unknown. Most authors agree that these patients should undergo revascularization whenever feasible. Aorto-aortic bypass, patch aortoplasty, and percutaneous techniques are the most frequent approaches used for the treatment of patients with middle aortic syndrome.

Panel A. Non-contrast computer tomography shows a severe calcified stenosis in the transition from the thoracic aorta to the abdominal aorta.

Panel B. The same lesion at a coronal view.

Panel C. The sequence of percutaneous intervention (pre- and post-intervention).

Panels D and E. Multidetector computer tomography showing the implanted stent at axial and sagittal views.

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