index was < 1.5 in only 2 patients. The use of RT3D TEE permitted percutaneous closure of the leak in 8 patients, and the major diameter was used to choose the device size. Table 1 compares the patients in this study based on the results of 2D and RT3D TEE.

In this study, RT3D TEE allowed for an accurate diagnosis of the EROA dimension, proper choice of closure device, and guided percutaneous leak closure.

Long-term Antihypertensive Treatment Decreases LV Twisting and Untwisting in Patients With Hypertension

Written by Nicola Parry

Ignatios Ikonomidis, MD, PhD, University of Athens Medical School, Attikon Hospital, Athens, Greece, presented results of a 3-year follow-up study, which demonstrated that long-term antihypertensive treatment improved the twist and untwist mechanics of the left ventricle in patients with hypertension, in addition to reducing blood pressure (BP), left ventricular (LV) mass, and arterial stiffness.

According to Prof Ikonomidis, LV function in patients with hypertension is determined by factors such as BP, arterial stiffness, LV mass, and coronary microcirculation. With this in mind, the researchers conducted a study to investigate the long-term effects of antihypertensive treatment on these parameters in this patient population.

The study enrolled 75 untreated patients (mean age, 54 ± 11 years) with essential hypertension and 50 healthy control participants of a similar age and sex distribution. All patients with hypertension were treated with angiotensin receptor blockers, and characterized as having well-controlled BP if their 24-hour systolic and diastolic BP were < 130/80 mm Hg.

At baseline and after 3-year follow-up, 24-hour ambulatory BP monitoring was performed in all study participants. Additional parameters were also assessed using conventional and speckle tracking echocardiography, including: carotid to femoral artery pulse wave velocity (PWV); coronary flow reserve (CFR) after adenosine infusion; LV mass/in^2, twisting (Tw); peak Tw velocity; untwisting at the mitral valve opening (unTwMVO), at the peak of the E wave (unTwE), and at the end of the E wave (unTwendE) of the mitral inflow; and untwisting (unTw) velocity.

Prof Ikonomidis explained that since LV torsional dynamics are sensitive markers of LV function, short-axis 2D images were analyzed for LV torsion, which was defined as the difference in rotation between the basal and apical planes.

Compared with control participants, those with hypertension had lower CFR (2.5 ± 0.6 vs 2.9 ± 0.6) and higher PWV (9.2 ± 1.5 vs 11.7 ± 2 m/s), Tw (13 ± 4 vs 20 ± 4 degrees), Tw velocity (89 ± 21 vs 126 ± 38 deg/s), unTwMVO (8.8 ± 3.2 vs 15.7 ± 5 degrees), unTwE (5.8 ± 3.1 vs 10 ± 5 degrees), unTwendE (2.2 ± 2.1 vs 5.8 ± 4 degrees), unTw velocity (–93 ± 31 vs –104 ± 37 deg/s), LV mass/m^2 (70.7 ± 14 vs 81 ± 16), and BP (P < .01 for all comparisons).

After 3 years of antihypertensive treatment, BP was well controlled in 70% of the patients with hypertension. Compared with baseline, these patients had reduced PWV (11.7 ± 2 vs 10.8 ± 1.5 m/s), Tw (20 ± 4 vs 15 ± 4 degrees), Tw velocity (126 ± 38 vs 110 ± 21 deg/s), unTwMVO (15.7 ± 4 vs 10.5 ± 4 degrees), unTwE (10.5 ± 4 vs 7.2 ± 4 degrees), unTwendE (5.8 ± 4 vs 3.9 ± 4 degrees), unTw velocity (–104 ± 37 vs –94 ± 31 deg/s), LV mass/m^2 (81 ± 16 vs 75 ± 16), and 24-hour BP (systolic 138 ± 10 vs 123 ± 14 mm Hg; and diastolic 87 ± 9 vs 75 ± 8 mm Hg; P < .05 for all comparisons). However, CFR remained similar (2.5 ± 0.6 vs 2.5 ± 0.9).

Prof Ikonomidis therefore concluded that long-term antihypertensive treatment improves LV twisting and untwisting in patients with essential hypertension, concomitant with reductions in BP, LV mass, and arterial stiffness.

Extent of Infarction Early Post-STEMI Does Not Correlate With Long-term Myocardial Recovery

Written by Nicola Parry

José Fernando Rodríguez Palomares, MD, University Hospital Vall d'Hebron, Barcelona, Spain, shared preliminary results from the PROMISE trial [NCT00781404] in patients with STEMI in which transmural infarction was identified by cardiac magnetic resonance imaging (CMR) early after STEMI. At the 6-month follow-up, the results demonstrated a decreased extent of infarction in many affected myocardial segments, with significant improvements in their contractile function.

According to Prof Rodríguez Palomares, in patients with acute myocardial infarction, the extent of transmural necrosis, as determined by CMR in the early post-STEMI phase, has been established as an excellent predictor of improvement in myocardial contractile function. However, data from some studies have suggested that the extent of transmural necrosis can be overestimated during the acute phase because of

the presence of edema in the infarcted tissue. Prof Rodríguez Palomares noted that after percutaneous coronary intervention, the recovery of dysfunctional myocardial segments that have an intermediate extent of transmural necrosis is variable, is difficult to predict, and may be related to the contractility of the remaining viable but stunned myocardium. He indicated that additional studies have also addressed this issue in chronic ischemic heart disease, showing that low-dose dobutamine test CMR is superior to extent of infarction for the prediction of functional recovery of myocardial contraction.

With this in mind, Prof Rodríguez Palomares and colleagues conducted the PROMISE trial to investigate the accuracy of the extent of transmural infarction in predicting the presence of contractile reserve and recovery of myocardial function at 6 months post-STEMI.

The study enrolled 95 patients with an acute STEMI. Following successful primary angioplasty, participants underwent CMR and a dobutamine stress echocardiogram during the first week and at 6-month follow-up.

The transmural extent of infarction in myocardial segments was determined by CMR. The researchers evaluated a total of 1520 myocardial segments and categorized transmural infarction as an extent of necrosis >50%. They determined peak longitudinal systolic strain (SS) and systolic strain rate (SSR) at baseline and after infusion of 10 μg/kg/min of dobutamine in 16 myocardial segments in each participant (except the apex, segment 17).

The results showed a decrease in deformation parameters as the extent of transmural necrosis increased: SS and SSR at baseline, after infusion of 10 μg/kg/min of dobutamine, and at 6-month follow up, were decreased with increased extent of transmural infarction. However, at 6 months, the extent of necrosis was significantly decreased, even in transmural infarcts ($P<.001$). Eighty percent of myocardial segments with 50% to 75% infarction, and 48% of those with >75% infarction in the acute phase demonstrated contractile reserve and improvement in myocardial contractile reserve, with concomitant decrease in the extent of transmural necrosis.

Prof Rodríguez Palomares therefore concluded that assessment of the transmural extent of a myocardial infarct early post-STEMI does not accurately predict functional recovery at 6 months and therefore should not be relied upon as an accurate short-term surrogate marker of improvement in myocardial contractile function.