**Background.**— AF following cardiac surgery is a common complication increasing risks of embolic events, hemodynamic instability, postoperative heart failure, hemorrhagic complications and increasing in-hospital length of stay.

**Methods.**— Thirty-one consecutive patients (17 males, 55%), aged (72 ± 9) with severe symptomatic AS (area < 1 cm²), who underwent aortic valve replacement (AVR) were prospectively included in two centers between 2009 and 2010. A complete preoperative echocardiography was performed in all patients, including global and segmental longitudinal strain using 2D speckle tracking. AF was divided into paroxysmal AF when AF lasted less than 48 h and persistent when AF remained at discharge.

**Results.**— The incidence of postoperative AF was (19/31, 61%). Nine patients had paroxysmal AF and 10 had persistent AF. On univariate analysis, heavy weight (P = 0.038) and GLS (P = 0.01) were the only predictors of paroxysmal postoperative AF. Using ROC curves, a cut-off value of −15% for GLS could predict paroxysmal AF with a sensitivity of 74% and a specificity of 67%; area under curve (AUC) 0.75. On univariate analysis, increased LV end systolic diameter (P = 0.006), E/E' ratio (P = 0.001), systolic pulmonary artery pressure (P = 0.03), LV ejection fraction (P = 0.006) and GLS (P = 0.01) were the only predictors of persistent AF. Using ROC curves, a cut-off value of −15% for GLS could predict persistent AF with a sensitivity of 90% and a specificity of 67%; AUC 0.87.

**Conclusions.**— Decreased preoperative GLS could predict postoperative paroxysmal and persistent AF. The best sensitivity and specificity were obtained with a cut-off value for GLS of −15%. Indeed, prophylactic antiarrhythmic therapy should be particularly proposed in patients with impaired longitudinal systolic function before AVR.

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**Evidence for myocardial dysfunction in patients with Friedreich's ataxia, normal left ventricular ejection fraction and mass**

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**Purpose.**— Myocardial involvement in Friedreich’s ataxia (FA) is characterized by iron deposits, diffuse fibrosis and focal necrosis. We hypothesized that subclinical left ventricular (LV) dysfunction might occur in patients with Friedreich’s ataxia who present with normal LV ejection fraction (LVEF) and mass.

**Methods.**— Nineteen patients with FA who were homozygous for the GAA expansion in the FA gene (mean age: 35 ± 16 years) and 19 age- and gender-matched healthy controls (mean age: 35 ± 15 years) were submitted to standard echocardiography. Short axis basal and apical views were analyzed using speckle tracking software. LV twist was defined as the net difference between the apical rotation and basal rotation. Peak systolic LV longitudinal strain was calculated from the apical 2-, 3- and 4-chamber views.

**Results.**— The two groups did not differ in terms of LVEF (68 ± 6% and 66 ± 6% in patients and in controls, respectively). A slightly higher LV mass index was observed in the patient group (93 ± 19 g/m² versus 81 ± 17 g/m², P = NS). The LV filling parameters did not differ between the 2 groups. However, early diastolic mitral annular velocity (Ea) was lower and the ratio of early transmitral flow velocity to Ea was higher in FA patients (table). Peak LV twist and global systolic longitudinal strain (GLS) were significantly reduced in patients as compared to controls. Early diastolic LV untwisting (at 5%, 10% and 15% of diastole) was not significantly different.

**Conclusions.**— In patients with FA, assessment of LV twist and GLS could potentially detect impaired myocardial function when LVEF and mass are still normal. These parameters may prove useful as an outcome measure for the assessment and follow-up of new therapies in the early stages of the disease.

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**Echocardiographic changes of cardiac function during ultra distance and trail running**

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**Background.**— Previous studies have suggested that marathon may induce reversible cardiac dysfunction. However, no previous study has assessed cardiac function during ultra distance and trail running. The aim of this study was to assess systolic and diastolic function during ultra distance and trail running.

**Methods.**— We studied 28 subjects (amateur runners) participating in the 2010 Ecotrail (80 km). All subjects underwent several echocardiographic examinations: before, during the race (Km 21 and Km 50) and at the end of the race. We systematically recorded 2D parasternal long axis and apical 4, 3 and 2 chamber views, allowing to measure conventional LV parameters and longitudinal strain. We also recorded PW mitral inflow and Doppler tissue imaging of the mitral annulus (lateral and septal). All measurements were anonymously performed.

**Results.**— Mean age was 43 ± 9 years. Twenty-one subjects (75%) finished the race. Left ventricular ejection fraction was significantly depressed at the end of the trail (64 ± 4% versus 70 ± 3% before, P < 0.0001), but not at Km 21 and 50. At Km 50, 2D longitudinal strain was significantly reduced as compared to longitudinal strain observed at baseline (−19.4 ± 3% versus −22.1 ± 2.1% before, P < 0.0008). Significant changes intransmitral velocities were observed after 21 km, earlier than the abnormalities observed for the systolic function (E/A ratio: 1.6 ± 0.7 before versus 1 ± 0.4 at Km 21, P = 0.0004).

**Conclusion.**— Our study suggests that ultra distance and trail running leads to abnormalities of systolic and diastolic function in amateur runners. Diastolic dysfunction arises earlier than systolic dysfunction. The assessment of longitudinal strain allows to detect early systolic dysfunction.

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**Is cardiac remodeling physiological in veteran tennis players?**

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**Background.**— Cardiac remodeling may occur in case of intensive training. However, few are known concerning the long-term cardiac effect of sport in veterans. The aim of this study was to analyze the cardiac changes due to tennis practice in veterans.

**Methods.**— The study population consisted in 353 subjects: 303 veteran tennis players (≥35 years) participating in the 2007, 2008 and 2009 veteran Roland-Garros tournaments and 50 healthy patients included in an age- and gender-matched control group. All subjects underwent a physical examination, a 12-lead electrocardiogram and a complete transthoracic echocardiography. Analysis was performed according to the decade of life, to the level of tennis training (intensive ≥10 hours/week) versus moderate training (T + versus T −) and to the Henry’s abacus.

<table>
<thead>
<tr>
<th></th>
<th>Friedreich patients</th>
<th>Controls</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>E wave velocity (cm/s)</td>
<td>72 ± 13</td>
<td>81 ± 17</td>
<td>NS</td>
</tr>
<tr>
<td>E/A</td>
<td>1.6 ± 0.6</td>
<td>2.1 ± 0.7</td>
<td>NS</td>
</tr>
<tr>
<td>Ea (cm/s)</td>
<td>8.9 ± 2.0</td>
<td>12.9 ± 3.2</td>
<td>0.000</td>
</tr>
<tr>
<td>E/Ea</td>
<td>8.7 ± 2.3</td>
<td>6.6 ± 1.6</td>
<td>0.002</td>
</tr>
<tr>
<td>Peak LV twist (°)</td>
<td>9.3 ± 3.4</td>
<td>11.7 ± 2.3</td>
<td>0.015</td>
</tr>
<tr>
<td>GLS (%)</td>
<td>−15.3 ± 2.2</td>
<td>−17.5 ± 1.6</td>
<td>0.001</td>
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