In patients with congenitally corrected transposition of the great arteries (CCTGA), the morphological left ventricle (mLV) and the morphological right ventricle (mRV) support the pulmonary and systemic circulations, respectively. Conventional repair, with the mRV retained in the systemic position, is associated with poor outcomes. An alternative approach, known as anatomical repair or double switch (DS), has been proposed to improve outcomes. Because the mLV is reallocated to systemic circulation in DS, the mLV should have the ability to sustain the systemic pressure load. In patients with low mLV systolic pressure, the mLV is deconditioned. Left ventricular training (LVT) by pulmonary artery banding (PAB) is necessary in these patients before DS is performed.

Zartner et al. [1] reported a novel approach for LVT. They performed loose PAB and concomitant atrial septectomy to increase both pressure load and volume load on the mLV. This approach, termed enhanced LVT (eLVT), was applied to six patients with CCTGA and deconditioned mLV. The midterm results were excellent. The target mLV systolic pressure to mRV systolic pressure ratio [left ventricle to right ventricle ratio (LV/RV ratio)] at eLVT surgery was set at 0.5. The mean LV/RL ratio increased to 1.0 after a median training period of 1.2 years. All patients underwent DS successfully. In previous studies on LVT without atrial septectomy, the target LV/RV ratio was set between 0.65 and 0.8, and the ratio of patients achieving DS candidacy was between 64 and 80%.

Myers et al. [2] performed LVT in 25 patients. The target LV/RV ratio was set between 0.66 and 0.75. Two patients required PAB tightening. Eighteen patients (72%) underwent DS after a median training period of 10 months. The mean LV/RV ratio increased to 0.94 before DS in these patients. Quinn et al. [3] performed LVT in 17 patients. The target LV/RV ratio was set between 0.66 and 0.80. Five patients required PAB tightening. Eleven patients (65%) underwent DS after a median training period of 436 days. The mean preoperative LV/RV ratio increased to 0.98 before DS in these patients. Many LVT candidates are reported to have mRV dysfunction and/or systemic tricuspid regurgitation [2]. An adequately tight PAB shifts the ventricular septum from a leftward to a midline position. The resultant reduction in the mRV volume decreases tricuspid regurgitation and improves mRV work efficiency. However, an overly tight PAB induces mLV dysfunction, whereas a too loose PAB results in a low LV/RV ratio and thereby fails to induce sufficient myocardial hypertrophy necessary for DS candidacy. Perhaps, a mild volume load with the eLVT procedure shifts the ventricular septum adequately without abruptly increasing the mLV systolic pressure excessively. A mild volume load with atrial septectomy may induce mild LV expansion, enhancing LV diastolic compliance, which leads to a gradual increase in the volume load and the LV/RV ratio. The study by Zartner et al. showed that eLVT caused a considerable increase in the LV/RV ratio during the training period and resulted in the achievement...
of a high rate of DS candidacy. Thus, eLVT could be an attractive option for LVT.

Animal studies have shown discrepant results. Szabó et al. [4] created a volume overload in dogs by placing arterio-venous shunts for 3 months and subsequently closed these. A pressure load created by PAB was imposed on normal and volume-overloaded RVs. The contractility of normal RVs was increased to maintain cardiac output, whereas the end-diastolic pressure and the volume of volume-overloaded RVs were increased to maintain output. The slope of the end-systolic volume–pressure relationship (Ees) was lower in volume-overloaded RVs than in normal RVs, indicating that volume-overloaded RVs are more prone to pressure overloading. Yerebakan et al. [5] compared 3 months of pressure overloading by PAB with 3 months of volume overloading by transannular right ventricular outflow patch placement. Volume-overloaded ovine RVs showed inferior diastolic and systolic capacities compared with pressure-overloaded RVs under dobutamine stress conditions. Borgdorff et al. [6] examined murine RV adaptation to pressure loading by monocrotaline-induced pulmonary hypertension with or without volume overloading by arterio-venous shunts. The RV Ees was lower after pressure overloading with volume overloading than after pressure loading without volume overloading, although the difference was not statistically significant. Diastolic function, indicated by the Ees, was worse after pressure overloading with volume overloading than after pressure loading without volume overloading. Thus, animal studies unanimously show that chronic volume overloading has a deleterious effect on RV function.

The inconsistency between clinical observations and animal experiments could be explained as follows. Firstly, volume-loading mechanisms by animal models and by eLVT are distinct. Arterio-venous shunts increase both systemic and pulmonary blood flow, whereas eLVT increases pulmonary blood flow alone. Arterio-venous shunts and pulmonary regurgitation create mandatory shunt flow, whereas shunt flow across an atrial septal defect depends on the pressure difference between the right and left atria and the difference between RV and LV diastolic compliances. Secondly, the amount of volume loading may differ. In experimental settings, a large amount of the shunt is burdened to emphasize the effect of volume loading. On the other hand, the mean pulmonary-to-systemic flow ratio was only 1.25 after eLVT. Thirdly, animal RV is not an accurate model of deconditioned mLV. Comparative, preferably randomized, studies are required to elucidate the role of volume loading in LVT.

REFERENCES