Changes in Left Ventricular Torsion Early Postoperatively After Aortic Valve Replacement and at Long-Term Follow-up

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Objective: In patients with aortic stenosis, left ventricular systolic torsion (pT) is increased to overcome excessive afterload. This study assessed left ventricular torsion before and immediately after surgical valve replacement and tested the instant effect of fluid loading.

Design: Prospective, clinical single-center study.

Setting: Intensive care unit of a university hospital.

Participants: 12 patients undergoing elective aortic valve replacement for aortic stenosis.

Interventions: Echocardiography was performed on the day before surgery, within 18 hours after surgery including a fluid challenge, and after 2.5 years.

Measurements and Main Results: pT decreased early postoperatively by 21.2% (23.4° ± 5.6° to 18.4° ± 6.9°; p = 0.012) and reached preoperative values at 2.5 years follow-up (24 ± 7). Peak diastolic untwisting velocity occurred later early postoperatively (13% ± 8% to 21% ± 9.4%; p = 0.019) and returned toward preoperative values at follow-up (10.2 ± 4.7°). The fluid challenge increased central venous pressure (8 ± 4 mmHg to 11 ± 4 mmHg; p = 0.003) and reduced peak systolic torsion velocity (138.7 ± 37.6/s to 121.3 ± 32/s; p = 0.032). pT decreased in 3 and increased in 8 patients after fluid loading. Patients whose pT increased had higher early mitral inflow velocity postoperatively (p = 0.04) than those with decreasing pT. Patients with reduced pT after fluid loading received more fluids (p = 0.04) and had a higher positive fluid balance during the intensive care unit stay (p = 0.03). Torsion after fluid loading correlated with total fluid input (p = 0.001) and cumulative fluid balance (p = 0.002).

Conclusions: pT decreased early after aortic valve replacement but remained elevated despite elimination of aortic stenosis. After 2.5 years, torsion had returned to preoperative levels.

KEY WORDS: left ventricular torsion, aortic stenosis, aortic valve replacement, transthoracic echocardiography, intensive care unit, fluid challenge

AORTIC STENOSIS POSES a chronically increased afterload on the left ventricle, leading to left ventricular wall thickening and increased energy expenditure.[1] In this state, energy efficiency of the left ventricle is optimized by augmenting left ventricular torsion, the deformation of the heart around its longitudinal axis. Left ventricular torsion describes the systolic difference of the rotational motion of the apex and the base of the left ventricle in which, viewed from the apex, the left ventricular base rotates clockwise and the apex counterclockwise due to the complex interaction of the obliquely arranged endocardial and epicardial muscle fibers. Left ventricular torsion allows for high intraventricular systolic pressures, with minimal muscle shortening resulting in efficient left ventricular contraction.[2] In severe aortic stenosis, left ventricular torsion mainly is driven by an augmented rotation of the apex.[3] Elevation of left ventricular torsion is reversible after aortic valve replacement (AVR), along with normalization of the left ventricular mass.[4] These long-term adaptive changes to optimize cardiac energetics have to be seen alongside acute changes of parameters determining cardiac function, especially acute alterations of loading conditions and contractility that can immediately affect left ventricular rotational motion. An acute elevation of afterload impairs, whereas an increased preload or the administration of dobutamine enhances left ventricular torsion.[5] Either way, left ventricular torsion can be seen as a marker of left ventricular systolic function whereupon therapeutic interventions that lead to an increased torsion (eg, fluids or inotropic drugs) improve cardiac efficiency.[6] Failing to further increase left ventricular torsion during fluid loading may, therefore, indicate the endpoint of fluid responsiveness. In turn, reduced left ventricular torsion may furthermore serve as an early marker for reduced left ventricular systolic function despite a maintained left ventricular ejection fraction as suggested for patients with septic cardiomyopathy.[7,8] Left ventricular torsion inevitably is linked to early diastolic filling since the rapid recoil or untwisting during isovolumetric relaxation contributes to early diastolic suction by generating low left ventricular pressures and consecutively higher transmitral pressure gradients.[6]

It currently is unknown how left ventricular rotational motion is affected early after aortic valve replacement. Transcatheter aortic valve implantation leads to an immediate reduction of left ventricular afterload, entailing left ventricular end-systolic volume reduction without reduction of end-diastolic volume.[9] A reduction of end-systolic volume is known to be associated with augmented torsion and improved diastolic suction, changes that could be expected as well after surgical AVR. Nevertheless, the influence of cardiopulmonary bypass on myocardial function and, especially, left ventricular rotational motion may affect hemodynamic changes after acute afterload reduction by AVR. In addition, right ventricular

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impairment is common after cardiopulmonary bypass, likely by septal dysfunction.[10]

The aim of this explorative study was to assess left ventricular torsion before and immediately after surgical AVR and to test the instant effect of fluid loading on left ventricular torsion. The authors hypothesized that during the immediate postoperative period left ventricular torsion is reduced relevantly by myocardial stunning but may be improved by preload adaptation.

METHODS

This study was conducted as a prospective clinical study at the Departments of Intensive Care Medicine and Cardiac Surgery of the University Hospital Bern, Switzerland. The study was approved by the Ethics Committee of the Canton of Bern (KEK 034/10), and a written informed consent was obtained from all patients.

Patient Selection

Twenty patients aged >18 years with severe aortic stenosis, based on a mean pressure gradient across the aortic valve >40 mmHg or an aortic valve opening area <1 cm^2 and planned for AVR with a biologic valve, were included in the study. Exclusion criteria were left ventricular ejection fraction $<45\%$ that was known or showed in the preoperative echocardiographic examination, prior myocardial infarction with documented wall motion abnormalities, a concomitant more-than-moderate aortic or mitral valve insufficiency or mitral valve stenosis, as well as severe pulmonary artery hypertension with a mean pulmonary artery pressure >40 mmHg. Patients had to be in sinus rhythm without bundle-branch block in the electrocardiogram. Patients were not included if additional cardiac surgical interventions other than aortocoronary bypass surgery were performed simultaneously. Furthermore, insufficient parasternal echocardiographic imaging quality was an exclusion criterion.

Surgical Procedure

Cardioplegia was induced using a single shot of a crystalloid solution (Cardioplexol, Laboratorium Dr. G. Bichsel AG, Unterseen, Switzerland) containing procaine, magnesium and potassium, followed by blood cardioplegia (Buckberg solution) if needed. All procedures were performed under moderate systemic hypothermia (32°C). For additional details see the online-only Supplemental Table.

Echocardiographic Studies

Transthoracic echocardiography (Vivid 7, GE Medical Systems CH, Glattbrugg, CH) was performed by one board-certified cardiologist trained in transthoracic echocardiography (SB) at 3 different time points: On the day before scheduled AVR, in the intensive care unit 14 to 18 hours after surgery in the extubated, spontaneously breathing patient without non-invasive ventilator support or catecholamine infusion, and at 2.5 ± 0.2 years follow-up. Attention was paid so that patients were positioned in a similar left lateral decubitus position for all examinations. The following data were acquired at all time points: Parasternal long- and short-axis views at the level of the left ventricular apex and mitral valve, and apical 4- and 2-chamber views at high frame rate (40-70 Hz). Pulsed-wave Doppler analysis was performed from the left ventricular outflow tract and aortic valve closure was defined. Continuous wave Doppler images were recorded from the aortic and tricuspid valves, and tissue Doppler imaging from the septal and lateral mitral annuli as well as the lateral tricuspid annulus. Images and loops were stored in raw data format for offline analysis. A second postoperative set of parasternal short-axis loops and pulsed-wave Doppler analysis of the left ventricular outflow tract were recorded after a fluid challenge of 250-mL bolus injection of Ringer's lactate solution applied with a 50-mL syringe. At the preoperative echocardiographic examination, arterial blood pressure and heart rate were recorded. In the intensive care unit, central venous pressure, invasive arterial blood pressure and heart rate were documented before and after the fluid challenge.

Offline Analysis

The EchoPac Software (Version 08, GE Medical Systems CH, Glattbrugg, ZH) was used for offline analysis of raw data. Left ventricular mass was calculated as recommended by the American Society of Echocardiography.[11] Left ventricular ejection fraction was calculated using the biplane Simpson method.[11] In case the biplane Simpson method was not feasible, a Teichholz measurement combined with a visual estimate were used to assess left ventricular ejection fraction. Right ventricular function was assessed using tissue velocity of the lateral tricuspid annulus (S') and by measuring TAPSE.[12,13] Valve function and pulmonary artery pressure were assessed according to the American Society of Echocardiography guidelines.[14,15] Left ventricular end-systolic stress was calculated as described previously.[16] Systolic left ventricular pressure was calculated by adding systolic arterial blood pressure and peak systolic pressure gradient across the aortic valve, derived from continuous wave Doppler analysis.

Torsion Parameters and Analysis

Left ventricular torsion was calculated by subtracting the rotation of the basis of the left ventricle close to the mitral valve from the rotation of the apex (Fig 1A). By definition, counterclockwise rotation is positive when viewed from the apex. Peak torsion was deducted from torsion versus time plots (Fig 1A). Torsion velocity corresponds to the first derivative in time of torsion (Fig 1B). Peak systolic torsion velocity and peak diastolic untwisting velocity were derived from torsion velocity versus time plots (Fig 1B). Measurements and normalization of temporal data to percentage duration of systole and diastole were performed as described previously.[17] Systole and diastole each were attributed 100%. Timing of peak torsion and peak systolic torsion velocity were expressed as the percentage of systole at which the highest value was reached and for peak diastolic untwisting velocity, likewise, the percentage of diastole when untwisting velocity reached the maximum negative value (Fig 1B).

In detail, speckle tracking analysis from high frame rate (40-70 Hz) parasternal short-axis images was performed at the level