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PHYSIOLOGICAL AND TECHNICAL NOTES

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Left ventricular rotation: a neglected aspect of the cardiac cycle

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Abstract *Purpose:* To describe the mechanics and possible clinical importance of left ventricular (LV) rotation, exemplify techniques to quantify LV rotation and illustrate the temporal relationship of cardiac pressures, electrocardiogram and LV rotation. *Materials and methods:* Review of the literature combined with selected examples of echocardiographic measurements. *Results:* Rotation of the left ventricle around its longitudinal axis is an important but thus far neglected aspect of the cardiac cycle. LV rotation during systole maximizes intracavitary pressures, increases stroke volume, and minimizes myocardial oxygen demand. Shearing and restoring forces accumulated during systolic twisting are released during early diastole and result in diastolic LV untwisting or recoil promoting early LV filling. LV twist and untwist are disturbed in a number of cardiac diseases and can be influenced by several therapeutic interventions by altering preload, afterload, contractility, heart rate, and/or sympathetic tone. *Conclusions:* The concept of LV twisting and untwisting closely linking LV systolic and diastolic function may carry potential diagnostic and therapeutic importance for the management of critically ill patients. Future clinical studies need to address the feasibility of assessing LV twist and untwist as well as the relevance of its therapeutic modulation in critically ill patients.

Keywords Left ventricular twist · Untwist · Torsion · Recoil · Systolic function · Diastolic function · Critically ill patients

Introduction

Our current clinical understanding and diagnostic evaluation of heart function focus on conformational changes of the heart in its radial and longitudinal dimensions, while overlooking an important component of left ventricular (LV) deformation. Even though Leonardo Da Vinci had already noticed that the mammalian heart rotates around its longitudinal axis during the cardiac cycle, it is only recent technical advances that have allowed noninvasive imaging and assessment of this circumferential LV motion and expanded our knowledge about this thus far neglected aspect of the cardiac cycle [1–3].

Definitions

Several nomenclatures have been used to describe the deformation of the heart around its longitudinal axis. According to the definition proposed by Sengupta et al. [2] in 2008, the term "rotation" refers to the circumferential motion of the LV around its longitudinal axis. The

unit of rotation is degrees. Rotation is defined as the angle between radial lines connecting the center of the LV cavity in a cross-sectional plane of the assumedly circular LV to a specific point of the myocardium in the same plane at end diastole and at any other time point during the cardiac cycle. Counterclockwise rotation viewed from the LV apex is arbitrarily defined as positive. Torsion is defined as the difference between rotation at an apical and a basal level of the LV over the period of a cardiac cycle. Furthermore, the term "twist" refers to systolic torsion, whereas "untwist or recoil" describes the diastolic changes in apex-to-base rotation.

Mechanics of LV torsion

Recent physiologic studies indicate that the longitudinal displacement of the atrioventricular plane from the base to apex of the heart accounts for about 60% of the total LV stroke volume. Radial compression and circumferential shortening are believed to contribute substantially to the remaining stroke volume [3]. Considering the heart as a complex biological pump, it must be assumed that the interaction of myofibrillar contraction in all three dimensions determines the final stroke volume.

Oblique orientation of the muscle bands in the LV wall set the anatomical basis for LV torsional deformation during systole and diastole. The myofiber geometry changes from a right-handed helix in the subendocardium to a perpendicular fiber orientation in the mid-wall and a left-handed helical configuration in the subepicardial muscle layer [4]. LV systolic contraction results in longitudinal and radial shortening, as well as opposite rotation of the base and apex around the heart's longitudinal axis. During LV diastole longitudinal and radial enlargement of the LV chamber is accompanied by release of the torsion (recoil or untwisting). The sequence of twisting and untwisting thereby plays an essential role for both systolic and diastolic conformation and function (see Figs. 1, 2, video sequences in the electronic supplementary material) [1–3].

Torsion in systole

During early isovolumic contraction, there is a low amplitude inital clockwise rotation of the LV apex followed by a sustained counterclockwise rotation during the LV ejection period. In contrast, the LV base rotates counterclockwise during the isovolumic and early ejection period followed by pronounced clockwise rotation lasting into the isovolumic relaxation period. Compared to the apex, rotation of the LV base is smaller in amplitude but relevantly contributes to net torsion (Figs. 1a, 2) [1–3]. Maximum counterclockwise LV torsion (apical rotation minus basal rotation) is reached at the end of the LV ejection period with peak values of ~12° at rest and up to 25° during physical exercise (Figs. 1a, 2) [2]. Compared to the base and apex, mid-ventricular wall segments do not rotate significantly during the cardiac cycle [5–7]. Systolic torsion aids in generating maximum intracavitary pressures with minimum fiber shortening thereby reducing myocardial oxygen demand [1–3]. A mathematical model indicated that LV torsion results in sarcomere shortening of 0.2 μm in the subepicardium and 0.48 μm in the subendocardium. Exclusion of LV torsion from the model resulted in significantly less subepicardial (0.1 μm) but more pronounced subendocardial shortening (0.55 μm). This corresponded to a relevant increase in myocardial oxygen consumption confirming the hypothesis that LV torsion minimizes myocardial energy expenditure during systole [1–3].

As a result of the torsional deformation of the LV during systole comparable to the wringing of a wet towel,

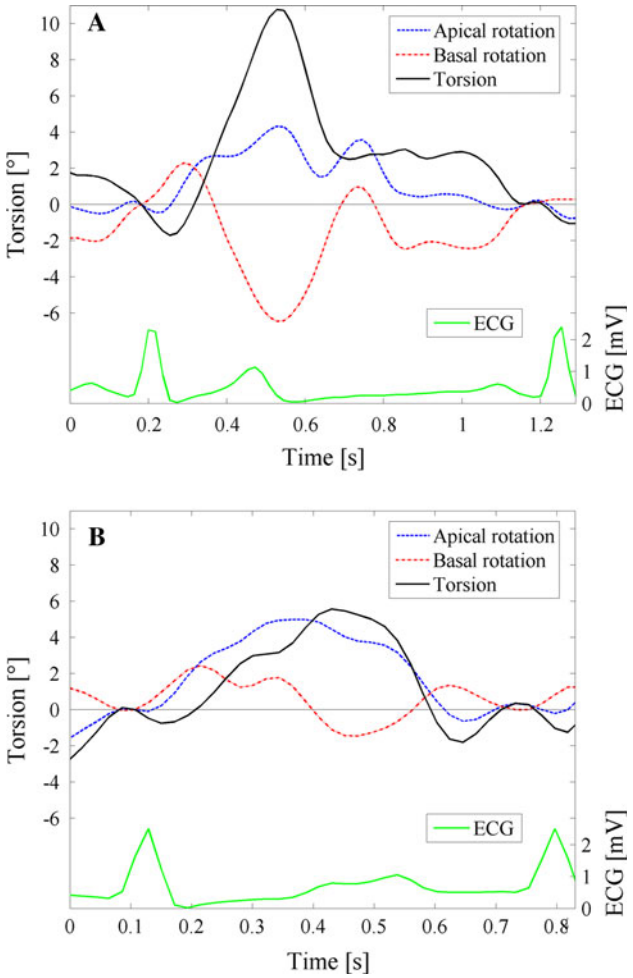

Fig. 1 Diagrams of normal left ventricular torsion in a healthy volunteer (**a**) and impaired left ventricular torsion in a patient with septic shock (**b**). *ECG* Electrocardiogram

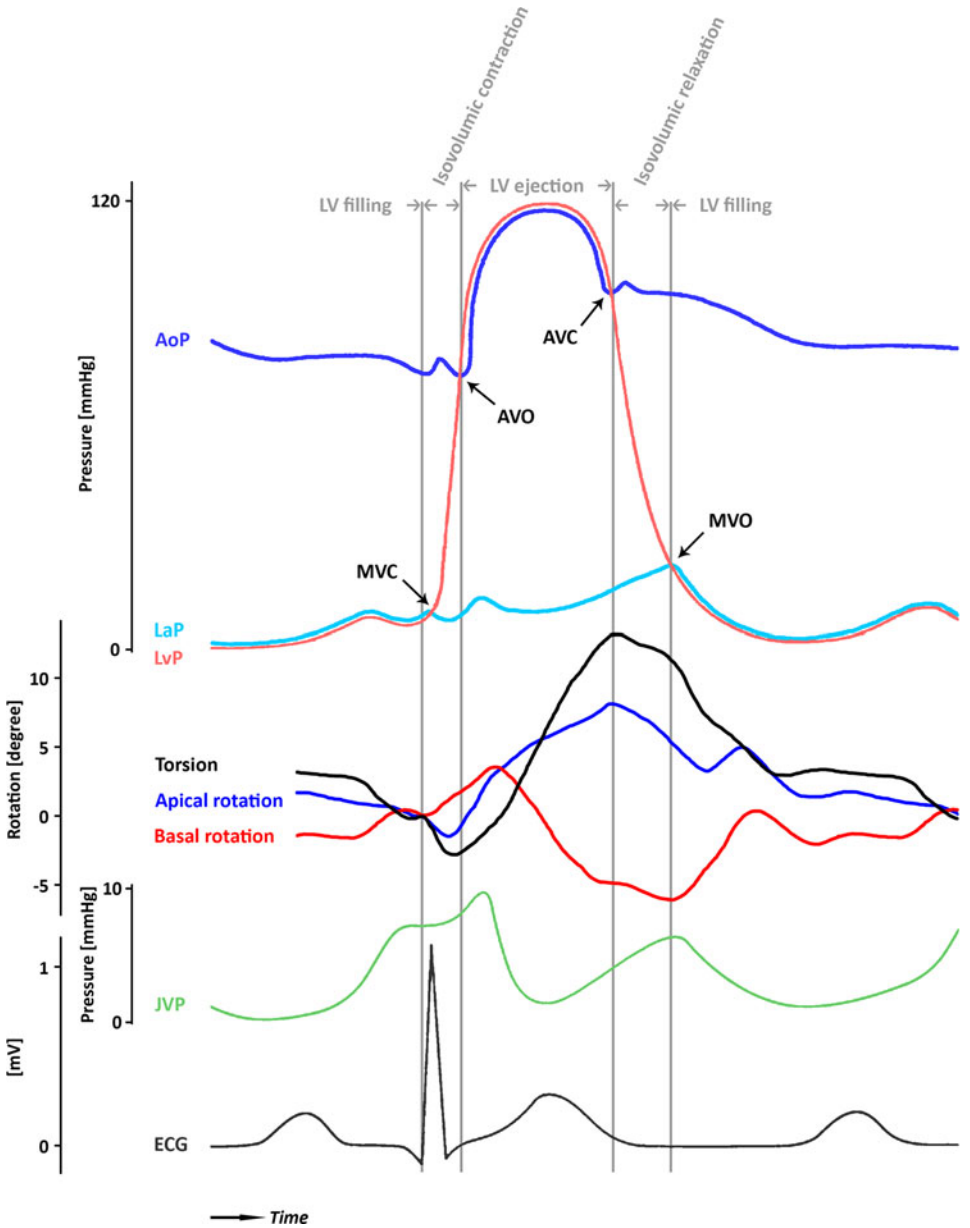

Fig. 2 Diagram depicting the temporal relationship of apical and basal LV rotation, LV torsion, cardiac pressure curves, and electrocardiogram (*ECG*). *AoP* Aortic pressure, *LaP* left atrial pressure, *LvP* left ventricular pressure, *JVP* jugular venous pressure, *MVC* mitral valve closure, *AVO* aortic valve opening, *AVC* aortic valve closure, *MVO* mitral valve opening

spiralling blood flow occurs in the left ventricle [8], the aorta, and the major conduit arteries [9]. Spiralling of blood flow is a more efficient means of energy transfer than propulsion of blood along a straight jet and may play a role in vital organ perfusion. Distortion of spiralling blood flow in the abdominal aorta, for example, was associated with deterioration of renal function in patients with renal artery stenosis [9].

Torsion in diastole

Systole and diastole are closely interlinked sequences of the cardiac cycle. Each systolic contraction has a direct impact on the following diastole and vice versa. One important link is LV twist, generated during systole, which is then progressively released during diastole, a process termed LV untwist or recoil [1–3, 10]. Systolic LV torsion does not only aid systolic ejection of blood, but also stores energy by twisting and shearing myofibers and the interstitial matrix. Near end-systole, when counterclockwise LV torsion is maximum and muscle bands start to relax sequentially from the subepicardium to the subendocardium, restoring forces are released and induce a reverse rotational LV deformation (Figs. 1a, 2). This LV recoil with the apex turning clockwise and the base counterclockwise occurs largely during isovolumic relaxation and the early LV filling period. A correlation

exists between the peak systolic LV torsion and the peak LV untwisting rate. The maximum untwisting rate in turn is correlated with the time constant of LV pressure decay (tau), a catheter-derived measure of LV relaxation [10]. Diminished systolic torsion is therefore inevitably associated with impaired LV recoil and reduced early diastolic filling offering an explanation why patients with systolic heart failure frequently exhibit impaired diastolic function [11].

The key role of LV recoil in diastolic function is further underlined by the correlation of the rate of LV apical untwisting with established parameters of diastolic function such as early diastolic mitral inflow velocity and the ratio of early diastolic mitral inflow velocity to mitral septal annular velocity in adults with preserved LV systolic function [12]. Similarly LV recoil was correlated with both passive (*e*-wave of mitral inflow) and active (*a*-wave of mitral inflow) filling of the left ventricle [5]. Furthermore peak LV untwisting rate is an independent predictor of the intraventricular base-to-apex pressure gradient that is considered a measure of diastolic suctioning during early diastolic filling [10]. In summary, disturbed LV recoil is paralleled by impaired LV filling, thereby decreasing end-diastolic volume and finally stroke volume [1–3, 10].

It remains a matter of debate if LV recoil is a truly active or passive process and where recoiling energy is stored during systolic LV torsion. One assumption is that the large sarcomeric protein titin accumulates energy during systole and acts as a bidirectional spring during diastole. Further potential sources of elastic recoiling forces built-up during LV twisting are shear stress among subendocardial and subepicardial muscle bands as well as the three-dimensional extracellular matrix [1–3].

As outlined above, the magnitude of LV torsion has a significant impact on LV recoil and diastolic filling. This mechanistic link of systole and diastole becomes particularly crucial when tachycardia reduces diastolic filling time (e.g., during exercise). Atrial pacing at 160% of the baseline heart rate increased rotation of both the anterior and posterior walls of the left ventricle in a canine model [13]. Similarly, pacing augmented the total amplitude of LV apical rotation by 42% in open-chest dogs [14]. Tachycardia induced augmentation of LV rotational deformation results in improved diastolic suction and may explain why stroke volume is maintained or even slightly increased during tachycardia and exercise despite shortened LV diastolic filling times [2]. During short-term exercise, LV rotation may become almost doubled [15]. Interestingly, long-term exercise training can decrease the amplitude of systolic LV torsion at rest, and trained athletes thus exhibit lower LV twist and untwist velocities than untrained subjects [16]. It is assumed that reduced systolic LV torsion at rest in athletes represents a functional reserve to be recruited during exercise [2].

Quantification of LV torsion

Until two decades ago, LV torsion and recoil could only be quantified by implanting radiopaque tantalum markers into the myocardium during open heart surgery. Recent technological advances have made noninvasive imaging and assessment of LV rotation possible. Currently, tagged MRI is considered the gold standard to evaluate LV torsional motion despite a restricted temporal resolution. Due to the need for transport, long examination times, and the requirement for repeated breath holds, MRI appears unsuitable for the critically ill patient. Transthoracic echocardiography using speckle tracking imaging, on the other hand, can be performed at the bedside and measurements of torsion correlate well with those obtained by MRI in humans ($r = 0.93$) during rest and dobutamine infusion. Disadvantages of speckle tracking imaging include the reliance on adequate image quality, the attempt to track a three-dimensional motion with a two-dimensional image and a relatively high intra- and inter-observer variability (5–10%) [1–3, 17]. Frame rates of at least 40 frames per second (fps), ideally 50–90 fps, are needed to accurately depict rotational motion by echocardiography. Only recently a transesophageal system has been developed capable of acquiring three-dimensional data sets and computing torsion based on those data sets although so far with the drawback of inadequately low frame rates for image acquisition (for illustration, see video sequences in the electronic supplementary material).

Quantification of LV rotation has been suggested to be a sensitive tool to detect early changes in LV systolic and diastolic function. Current data indicate that rotational deformation in heart failure patients is a more accurate marker of changes in cardiac muscle performance or predictor of outcome than conventional measures such as ejection fraction or stroke volume [1–3, 18].

LV torsion during health and disease

The characteristics of LV torsion undergo substantial changes during the aging process. LV systolic torsion gradually increases from infancy to adulthood [1–3]. LV systolic torsion continues to increase with aging as a result of increased apical rotation and decreased rotational deformation delay between the apex and base. This may offer an explanation for the preservation of LV ejection fraction in the elderly [19]. Nevertheless, the increase in peak systolic torsion is paralleled by a progressive decline in subendocardial function with aging reducing elastic and restoring forces of the left ventricle and therefore impairing LV recoil. Accordingly, diastolic function is frequently impaired in this age group [1–3].

Table 1 Left ventricular torsion in different cardiac diseases [2]

Type of cardiac disease	LV twist	LV untwist
Systolic heart failure	Reduced	Reduced
Dilated cardiomyopathy	Reduced	Reduced
Subendocardial ischemia	Normal	Normal or reduced
Anterior wall MI	Reduced	Reduced
Chronic mitral regurgitation	Reduced	Reduced
Diastolic heart failure	Normal or increased	Normal or reduced
Aortic stenosis	Normal or increased	Reduced
Hypertrophic cardiomyopathy	Normal or increased	Reduced
Restrictive cardiomyopathy	Normal or increased	Normal
Constrictive pericarditis	Reduced	Reduced
Diabetic cardiomyopathy	Normal or increased	Normal or reduced

LV Left ventricular, *MI* myocardial infarction

The amplitude and timing of LV torsion has been assessed in different cardiac pathologies (Table 1) [2]. In patients with impaired systolic function and reduced ejection fraction, LV systolic torsion and hence LV recoil were significantly reduced when compared with healthy controls. Since the anterior free wall and the apex significantly contribute to LV twisting, patients with anterior wall myocardial infarction presented with decreased peak systolic LV torsion. Again, in those patients diastolic recoil of the LV and LV filling were reduced and delayed. Chronic mitral regurgitation resulted in the loss of rapid LV recoil during early diastole and disruption of the close coupling between LV recoil and filling [2]. In patients with chronic pressure overload due to aortic stenosis or with hypertrophic cardiomyopathy with obstruction of the LV outflow tract, counterclockwise rotation of the apex was increased resulting in unchanged or even increased amplitude of systolic LV torsion (Fig. 3a). Such an augmentation of LV torsion was postulated to represent a compensatory mechanism of the LV to overcome increased afterload as LV torsion optimizes energy expenditure by maximizing intracavitary pressures with minimal myofiber shortening. However, such a compensatory increase in systolic LV torsion does not translate into enhanced diastolic LV recoil. Several authors observed that diastolic untwisting was delayed and peak rotation velocity reduced offering another potential explanation for the frequently observed diastolic dysfunction in these patients (Fig. 3a) [20].

Therapeutic modulation of LV torsion

Several therapeutic interventions commonly applied in critically ill patients affect LV torsion and recoil.

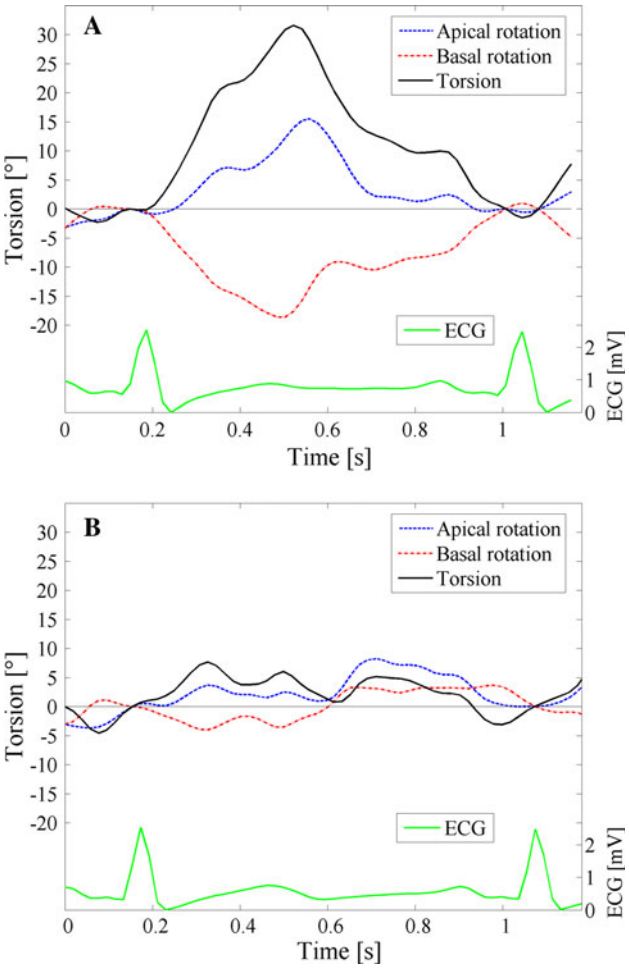

Fig. 3 Diagrams of left ventricular rotational parameters in a patient before (**a**) and 24 h following aortic valve replacement (**b**) with a biologic valve for severe degenerative aortic valve stenosis

Basically, a directly proportional relationship between LV torsion and end-diastolic volume as well as an inversely proportional relationship between LV torsion and end-systolic volume exist. Thus, changes in loading conditions and contractility influence the magnitude of LV rotation during the cardiac cycle [1–3]. Augmentation of torsion parameters in response to volume loading or inotropic support was termed positive plecotropy [21].

Changes in loading condition

First studies evaluating the effects of changes in loading conditions on LV twist and untwist reported conflicting results. When isolated heart models were investigated, it became clear that LV rotation is substantially affected by both preload and afterload [22]. By using MRI tissue tagging in an isolated, blood-perfused, ejecting heart model, Dong et al. [22] reported that at a constant end-systolic

volume, LV systolic torsion increased with higher end-diastolic volumes (end-systolic volume of 14 mL: 9.4 ± 3.4° vs. 12.2 ± 4° at an end-diastolic volume of 21.7 and 26.7 mL, respectively; end-systolic volume of 22 mL: 6.4 ± 5.1° vs. 8.1 ± 3° at an end-diastolic volume of 26.7 and 32.4 mL). However, increasing preload by administration of fluids in patients with normal systolic function only moderately augmented LV torsion but significantly decreased the proportion of untwisting prior to mitral valve opening and untwisting acceleration and additionally delayed the timing of peak untwisting [23]. Similar effects of increasing preload on LV untwisting were observed by other authors [24, 25]. Increasing afterload at a constant end-diastolic volume (26.7 mL) in the above-mentioned isolated, blood-perfused, ejecting heart model, decreased LV torsion significantly from 12.2 ± 4° to 10.7 ± 3.9° to 6.4 ± 5.1° as the end-systolic volume increased from 14.5 to 17.1 to 21.5 mL [22]. These results are in accordance with two earlier studies [24, 26]. However, in heart-transplanted patients, no change in LV torsion was observed in response to a methoxamine-induced increase in afterload [25, 27]. This discrepancy most likely results from the fact that methoxamine may not only increase afterload but may augment preload as well, thereby compensating for the afterload-induced reduction in LV torsion. Decreasing both preload and afterload with glyceryl trinitrate resulted in improved diastolic recoil and earlier onset of LV untwisting [23].

Changes in contractility

Apart from their intrinsic ability to speed LV relaxation under physiologic conditions (positive lusitropy through phosphorylation of phospholamban and activation of the sarcoplasmatic reticular calcium ATPase), beta-adrenergic drugs were found to influence the extent of LV rotation. In more detail, dobutamine infusion increased LV torsion by augmenting the rotational amplitude of both base and apex, accounting for additional accumulation of restoring energy with higher untwisting velocities facilitating early diastolic filling [9, 13, 22, 25, 27, 28]. In the above cited model of isolated, blood-perfused hearts, dobutamine significantly increased LV torsion (from 8.7 ± 1.5° to 13.3 ± 4.3°) and peak systolic pressures at constant end-diastolic and end-systolic volumes suggesting a direct inotropic effect on LV torsion not mediated by changes in volume but by changes in force [22]. Dobutamine administered to cardiac transplant recipients at 5 μg kg^{-1} min^{-1} resulted in a significant augmentation of both LV twisting and untwisting [27]. In contrast application of a beta-blocker resulted in a decrease in both LV twisting and untwisting [10]. When taking into account these observations with the focus on critically ill patients, one has to remember that all mentioned investigations were either performed in healthy subjects or patients with maintained systolic function. Detrimental effects of beta-stimulation on both systolic and diastolic function have been reported in various disease states [29].

Dobutamine is the only inotropic substance that has been investigated so far for its effects on LV rotation. The plecotropic effects of other positive inotropic agents may be delineated according to their pharmacological properties. For example, a beta-adrenergic-mediated augmentation of both LV twist and untwist could partly be counteracted by an alpha-adrenergic-induced increase in afterload during therapy with epinephrine. The use of phosphodiesterase inhibitors or calcium sensitizers may exert particularly beneficial effects on LV rotation due to a concomitant increase in contractility and a decrease in LV afterload. Levosimendan's positive lusitropic effects could specifically improve LV untwist parameters [30].

Changes after cardiac surgery and cardioplegia

Although no data have been published so far on the effects of cardiac surgery and cardioplegia on LV torsion, it is conceivable that LV twist and untwist may be significantly impaired early after cardiac surgery. Figure 3 depicts torsional parameters of a patient with severe aortic stenosis before (Fig. 3a) and 24 h after biologic aortic valve replacement (Fig. 3b). LV torsion appears relevantly impaired emphasizing the need for future studies on the effects of cardiac surgery and cardioplegia on LV torsion as well as therapeutic strategies to improve rotational parameters during the early postoperative phase.

Potential clinical implications for the management of critically ill patients

So far, no data on LV torsion and recoil in intensive care patients have been published. However, evolving knowledge on LV rotation and its impact on systolic and diastolic function may have important implications for the hemodynamic management of critically ill patients. First, given that LV twist and untwist are impaired in several cardiac diseases, the intensivist must assume that a relevant number of critically ill patients present with disturbances of LV torsion due to their underlying heart disease (Fig. 3a). Whether detection of acute changes in LV torsion during critical illness can improve detection of cardiac dysfunction or prediction of patient morbidity and mortality needs to be investigated in future studies. Finally and probably most importantly, the concept of LV torsion and recoil can substantially contribute to the pathophysiologic understanding of acute heart failure in critically ill patients and improve the use of therapeutic interventions in these patients. Moreover, the dependence of both LV recoil and early diastolic filling on the force and magnitude of LV torsion could open new therapeutic

windows for the treatment of patients with acute diastolic heart failure. Particularly, the therapy approach to these patients, which currently involves heart rate control, cautious fluid therapy, and avoidance of positive inotropic drugs [31], could be dramatically challenged. Hypothetically, afterload reduction and the infusion of beta-adrenergic drugs such as dobutamine may improve cardiac function even in the presence of diastolic dysfunction by enhancing LV torsion and consequently LV recoil and diastolic filling. However, future clinical trials need to prove the superiority and safety of such a therapy approach in critically ill patients with acute diastolic heart failure.

Conclusions

Rotation of the left ventricle around the heart's longitudinal axis is an important but thus far neglected aspect of the cardiac cycle. LV torsion during systole maximizes intracavitary pressures, increases stroke volume, and minimizes oxygen demand. Shearing and restoring forces generated during systolic torsion are released during early diastole and result in LV recoil, which promotes LV suction and diastolic filling. LV torsion is disturbed in a number of cardiac diseases and can be influenced by several therapeutic interventions that affect preload, afterload, contractility, heart rate, and/or sympathetic tone. The concept of LV rotation closely linking systolic and diastolic function may carry crucial diagnostic and therapeutic implications for the management of critically ill patients. Future studies need to address the diagnostic relevance and therapeutic modulation of LV torsion in critically ill patients.

Conflict of interest No author has a conflict of interest in regards of methods, techniques, or drugs discussed in this manuscript.

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