Current Cardiology Reviews, 2022, *18*, e060122200068

REVIEW ARTICLE

Paradoxical Septal Motion after Uncomplicated Cardiac Surgery: A Consequence of Altered Regional Right Ventricular Contractile Patterns

Alfred Stanley[1], Constantine Athanasuleas[2,*] and Navin Nanda[3]

[1]*Cardiovascular Associates of the Southeast, Birmingham AL and Kemp-Carraway Heart Institute, Birmingham AL, USA;* [2]*Department of Surgery, Section of Cardiothoracic Surgery, University of Alabama at Birmingham, Birmingham, AL and Kemp-Carraway Heart Institute, Birmingham AL, USA;* [3]*Division of Cardiovascular Disease, Department of Medicine, University of Alabama at Birmingham, Birmingham AL, USA*

ARTICLE HISTORY

Received: July 31, 2021
Revised: August 20, 2021
Accepted: November 24, 2021

DOI:
10.2174/1573403X18666220106115117

Abstract: Paroxysmal interventricular septal motion (PSM) is the movement of the septum toward the right ventricle (RV) during cardiac systole. It occurs frequently after uncomplicated cardiac surgery (CS), including coronary bypass (on-pump and off-pump), valve repair or replacement, and with all types of incisions (sternotomy or mini-thoracotomy). It sometimes resolves quickly but may persist for months or become permanent. Global RV systolic function, stroke volume and ejection fraction remain normal after uncomplicated CS, but regional contractile patterns are altered. There is a decrease in longitudinal shortening but an increase in transverse shortening in the endocardial and epicardial right ventricular muscle fibers, respectively. PSM is a secondary event as there is no loss of septal perfusion or thickening. The increased RV transverse shortening (free wall to septal fibers) may modify septal movement resulting in PSM that compensates for the reduced RV longitudinal shortening, thus preserving normal global right ventricular function.

Keywords: Paroxysmal septal motion, right ventricular systolic dysfunction, tricuspid annular plane systolic excursion, tricuspid annular systolic velocity, ventricular interdependence, cardiac systole.

1. INTRODUCTION

Paradoxical septal motion (PSM) is an abnormal anterior movement of the septum toward the right ventricle despite normal septal thickening during cardiac systole. PSM frequently develops after uncomplicated cardiac surgery (CS) and has been recognized since the early days of echocardiography (echo) [1-5]. Abnormal systolic and/or diastolic septal motion has been previously observed with several cardiac conditions: right ventricular volume/pressure overload, LBBB, constrictive pericarditis, congenital absence of the pericardium, right ventricular pacing and septal infarction. The pathophysiology affecting each condition has been clearly elucidated [6-10].

The following historical review describes how investigators have used a variety of diagnostic tools, including echocardiography (echo), tissue Doppler imaging (TDI), magnetic resonance imaging (MRI) and computerized tomography (CT) to study the cause of PSM after CS. These studies have enhanced our knowledge, but after three decades, there remain unanswered questions. However, the discovery of a significant temporal relation between altered regional right ventricular function and altered septal function (PSM) after CS suggests a possible mechanistic relationship between the right ventricle and PSM. After CS, there is a decrease in regional right ventricular (RV) longitudinal shortening and an increase in RV transverse shortening in the endocardial and epicardial RV muscle fibers, respectively. RV stroke volume and ejection fraction (EF) remain normal. It appears that PSM is a consequence of the altered regional RV contractile patterns that ultimately preserves global RV function. The cause of altered regional RV contractile patterns after CS to date remains elusive and may be related to surgical technique.

2. PARADOXICAL SEPTAL MOTION AND CARDIAC SURGERY

The reported incidence of PSM after uncomplicated CS ranges from 40% to 100%. The gradual resolution of PSM over time is variable, although it may persist indefinitely in some patients [4, 5, 11-13]. Past theories to explain the cause of PSM after CS have included: 1) impaired septal contractility from ischemia or infarction, 2) altered pericardial restraint after pericardiotomy, 3) artifact due to exaggerated post-operative anterior systolic cardiac mobility that resolves over time with the progressive restraining effect of adhesions [2, 14-19]. Numerous past publications have speculated that PSM after CS reflects post-operative septal injury. Surgical techniques, cardioplegia composition and delivery systems have been modified to allegedly prevent or limit PSM. Because of the septum's major contribution to global RV function, septal myocardial protection strategies have been promoted to prevent post-operative right ventricular

* Address correspondence to this author at the Department of Surgery, Section of Cardiothoracic Surgery, University of Alabama at Birmingham, Birmingham, AL and Kemp-Carraway Heart Institute, Birmingham AL, USA; Tel: 205 492 1827; E-mail: cathanasuleas@uab.edu

1875-6557/22

dysfunction [20]. However, the perception that septal injury causes PSM after CS has largely been refuted by the evidence of normal septal perfusion, viability and thickening following uncomplicated CS [21-24]. Despite being almost universally recognized, the mechanism, prevalence and time course of PSM following uncomplicated cardiac surgery remain controversial.

2.1. Early PSM Observations

Akins, in 1984, assessed the role of cardiopulmonary bypass (CPB) in the genesis of PSM following CS [25]. The study included 22 patients who underwent single-vessel coronary artery bypass grafting (CABG): 11 patients with CPB and 11 patients without CPB. Postoperatively, all 11 patients with CPB had decreased septal function, while 10 of 11 patients without CPB had no change or improvement in septal motion. There was no evidence of perioperative infarction in either group. Other reports also concluded that PSM was directly related to events that occurred during CPB [3, 26].

CPB and cardioplegic arrest represent a clinical model of ischemia-reperfusion (IR) injury and prolonged IR is known to cause cellular dysfunction, injury and death [27]. Surgical strategies are used during CPB to preserve tissue viability and reduce myocardial damage: faster procedures, shorter aortic cross-clamp time, hypothermia and cardioplegia (variable types and compositions). These maneuvers are designed to slow myocardial metabolic activity, reduce oxygen demand and protect against re-oxygenation injury [28]. However, CPB has been-associated with biochemical changes (enzyme release), physiological changes (altered right ventricular function), mechanical changes (reduced left ventricular basal anteroseptal segment rotation) and anatomical changes (right ventricular shape becomes more spherical) [11, 13, 29-35]. These abnormalities have been thought to be the result of IR injury.

A 2007 multivariate analysis of 3,292 patients, performed to determine which CS characteristics correlated with PSM, revealed: 1) aortic and/or mitral valve surgery is more likely to cause PSM than CABG, 2) off-pump CABG causes less PSM than on-pump CABG, and 3) longer aortic cross-clamp times were associated with the development of PSM [4]. The authors concluded that the cause of PSM is multifactorial.

3. RIGHT VENTRICULAR FUNCTION AND CARDIAC SURGERY

The interventricular septum is shared by the RV and many studies have discussed this interdependence in proposed mechanisms of PSM. It is important to review aspects of the anatomy and physiology of the septum. Septal muscle fibers are obliquely oriented as are left ventricular fibers, and generate a systolic twisting contraction pattern. The anatomy of the RV is distinctly different from that of the left ventricle (LV). The right ventricular myocardium is composed of longitudinal subendocardial fibers and transverse circumferentially oriented sub epicardial fibers. There is no significant RV systolic twisting to contribute to overall RV function [36]. In the normal RV, longitudinal shortening accounts for approximately 80% of overall RV function, with transverse shortening contributing 20% [37, 38].

The quantitative assessment of right ventricular function has been historically difficult because of RV geometry and has changed over the years. Imaging techniques have included echo, TDI, and MRI. Indices of right ventricular function have included tricuspid annular plane systolic excursion (TAPSE), peak tricuspid annular systolic velocity (TAV), fractional area change (FAC), speckle tracking-derived peak longitudinal strain, 2-D and 3-D echocardiography and thermodilution pulmonary artery catheterization.

3.1. Early Measurements of RV Function and PSM

Kaul in 1984 introduced TAPSE as a metric of RV systolic function [39]. While comparing a control multi-gated radionuclide global RV ejection fraction (RVEF) to a 2D echo apical 4-chamber cross-sectional area, a chance observation of the RV tricuspid annular systolic motion suggested its possible role in evaluating RV systolic function. There was a close correlation between RVEF and TAPSE. The authors concluded TAPSE to be a simple, reproducible method to estimate RVEF without geometric assumptions or traceable endocardial outlines. Tissue Doppler imaging followed later and measured peak tricuspid annular systolic velocity [40]. Both measurements are surrogates of RV longitudinal shortening. There is excellent correlation between TAPSE and TAV [41]. However, the authors were unaware that they were comparing global and regional RV systolic function.

Abnormal RV systolic regional longitudinal shortening following CS was first described by Wranne in 1993 after observing reduced TAPSE [26]. He was studying the time course and mechanism of PSM with intra-operative 2D transesophageal echocardiography (TEE) in patients undergoing CABG/valvular surgery. Interventricular septal motion and tricuspid annular systolic motion were recorded. Measurements were made during surgery with chest closed (baseline), chest open, pericardium open, after CPB and after chest closure. The investigators observed reduced TAPSE after CPB and recognized the temporal relation between decreased TAPSE and PSM. After CPB, 29% of patients had PSM (open chest) that increased to 86% after chest closure. After CPB, 76% of patients had reduced TAPSE (open chest) that increased to 95% after chest closure. This classic study established that following CS, patients with PSM also have reduced TAPSE and both features simultaneously deteriorate with chest closure. The authors hypothesized that suboptimal RV preservation impairs RV function and TAPSE after CPB and that recruitment of the septum (PSM) would help maintain an adequate RV stroke volume. Previous investigators had also reported that chest closure augmented PSM [3, 18].

Right ventricular regional systolic dysfunction, defined as reduced longitudinal shortening by TAPSE or TAV, is common after CS [11, 13, 30, 31, 42]. Hedman (2004) and Roshanali (2008) independently reported that following CPB, reduced TAPSE and velocity remained for one year

and closely correlated with PSM, *i.e.,* 96% and 97%, respectively [11, 13]. The temporal relation between reduced TAPSE and PSM had been previously described [26, 43]. However, despite persistent reduction of TAPSE following CS, most patients had a better NYHA functional class, experienced an uneventful post-op clinical course and an improved exercise capacity three months after CS [11, 13, 43]. These observations led to uncertainty regarding TAPSE as an accurate reflection of global RV systolic function.

A later study suggested that the RV is selectively impaired during cardiac surgery. Yadav in 2010 measured RV and LV myocardial tissue Doppler systolic velocities in patients undergoing CABG and compared the RV/LV ratio before and after surgery [31]. This ratio compares tricuspid annular systolic velocity and mitral annular systolic velocity that contrast RV and LV systolic longitudinal shortening. He observed a significant fall in the RV/LV ratio immediately after surgery that persisted long-term and concluded there was selective right ventricular impairment following uncomplicated CABG.

3.2. Timing of Regional RV Dysfunction

The precise timing of reduced RV longitudinal shortening during CS has been measured with intra-operative 2D TEE imaging by numerous investigators [3, 26, 44, 45]. Regional long-axis RV systolic function has been assessed by TAPSE, TAV and speckle tracking-derived peak longitudinal strain. Measured time points have included: 1) after anesthetic induction, 2) sternotomy, 3) pericardiotomy, 4) CPB, and 5) chest closure. These studies have all concluded that TAPSE, TAV and longitudinal strain are all significantly reduced following CPB during uncomplicated CS.

Unsworth, in 2010, using angle-dependent tissue Doppler in a small study, described reduced RV long-axis velocities within three minutes after opening the pericardium and concluded that pericardiotomy caused reduced RV longitudinal shortening [15]. However, Bitcon in 2017 measured TAPSE and TAV using angle-independent speckle tracking and reported that pericardial opening and suspension had no effect on these indices [44]. They noted that reduction of TAPSE only occurred after CPB and chest closure, an observation reported by many others [3, 26, 45, 46].

3.3. Global RV Function after Cardiac Surgery

The disconnection between the abnormal reduced RV longitudinal shortening and improved clinical status after CS continued to produce uncertainty about TAPSE as an accurate reflection of post-op global RV systolic function. Tamborini in 2009 evaluated global RV function in patients undergoing surgical mitral valve repair [47]. All patients were studied by 2D and 3D transthoracic echocardiography (TTE) pre-op and post-surgery. TAPSE and TAV were significantly reduced after surgery. Preoperative global RVEF, measured by 3D-echo, was unchanged at 3, 6 and 12 months after surgery. The authors cautioned against the use of TAPSE and TAV to assess post-operative global RV function and suggested that alterations in these long-axis parameters might represent geometrical changes (associated with interventricular PSM) rather than functional changes in the RV chamber. Other investigators studying global RV function after heart valve surgery with 2D echo, speckle-tracking, and 3D echo have confirmed that global RVEF remains unchanged following uncomplicated CS [48, 49].

Gronlykke in 2019 used 3D TEE and pulmonary artery thermodilution catheterization for detailed hemodynamic assessment of global RV function in patients undergoing uncomplicated CABG [50]. Post-operative TAPSE and TAV were significantly reduced, however, RV output, stroke volume and global RVEF remained unchanged by both 3D echo and hemodynamic measurements.

3.4. Regional RV Contractile Patterns after Cardiac Surgery

Raina in 2013 measured regional contractile patterns simultaneously by studying RV fractional area change (RV-FAC), a metric of global RV function, before and after CS with 2D echo and Doppler from the apical 4-chamber view to determine the contribution of longitudinal and transverse shortening to overall RV function [33]. Although global RV-FAC remained stable after CS, there was a marked change in regional RV contractile patterns with a decrease in longitudinal fractional area change (RV base to apex) and a two-fold increase in transverse fractional area change (RV free wall to septum). The authors concluded that CS caused a disruption in native RV contractile patterns that produce a more spherical RV shape.

Recently, others have investigated the observation of normal global RV output and altered regional RV contractile patterns after CS with advanced speckle tracking by directly measuring RV longitudinal and transverse displacement and peak systolic strain, a measure of myocardial fiber contractile movement [32, 45, 46, 51-53]. Strain is a measure of myocardial deformation, a change in the length of a segment relative to its baseline length. The advantage of myocardial deformation parameters (strain, strain-rate) is their independence from tethering and global cardiac motion that impact all wall motion-based parameters (myocardial velocity, displacement). Originally, strain imaging was derived from myocardial tissue Doppler velocities, but in recent years, speckle tracking echo has become the technique of choice because of its angle-independence [54].

Keyl in 2016 studied patients undergoing aortic valve replacement (AVR) for aortic valve stenosis [48]. Twenty patients underwent surgical replacement (SAVR) and 20 patients had transcatheter replacement (TAVR). All patients had 3D TTE one week before and 5-7 days after the intervention. TAPSE, TAV, global RVEF and regional RV mid-cavity transverse shortening were measured in both groups. After surgery, SAVR patients revealed a decreased RV longitudinal shortening and an increased RV transverse shortening. Both parameters remained unchanged in TAVR patients. Global RVEF and RV stroke volume remained unchanged in both groups.

Donauer in 2020 studied regional RV deformation in patients undergoing CABG [52]. All had antegrade cold blood cardioplegia and pericardial closure. Patients had 3D TTE one week before and 3-4 days after surgery as well as intra-operative 3D TEE speckle tracking measurements 1) after anesthesia, 2) before incision, 3) after pericardiotomy and 4) after sternal closure. TEE images were acquired to assess RV longitudinal, lateral free wall and RV inflow-outflow track deformation. Images allowed assessment of circumferential strain from the RV inferior-lateral and anterior-lateral free wall segments. After CS, there was a significant reduction in peak longitudinal strain of the RV lateral-inferior walls and RV outflow track with a simultaneous increase in the circumferential strain of the RV lateral wall. The global RVEF did not change. There was a decrease in peak systolic longitudinal septal strain after sternal closure (31%) that persisted for five days.

Korshin in 2020 studied patients undergoing CABG with intra-operative 2D and 3D TEE and RV speckle tracking software [53]. TAPSE, TAV, FAC, longitudinal and transverse strain were measured before surgery and immediately after weaning from CPB. This study showed a loss of RV longitudinal displacement and a concomitant gain in transverse displacement that occurs immediately after CPB and upon chest closure. Longitudinal and transverse strain did not change significantly at any time during surgery. The authors suggested that the increased transverse displacement might reflect a compensatory mechanism to mitigate the reduced longitudinal displacement and maintain global RV output.

Moya Mur in 2018 measured LV and RV systolic longitudinal displacement and strain and transverse shortening in patients undergoing CABG/valvular surgery [32]. All patients had transthoracic speckle tracking 2D echo from the apical 4-chamber view the day before and three-four days after surgery. The direction and magnitude of longitudinal displacement for basal, mid and apical segments were tracked and displacement curves for the LV septal, LV free wall and RV free wall were analyzed. To measure displacement, the authors identified a stationary point toward which all segments moved in systole, the static longitudinal reference point (sLRP). This point remained immobile during systole. Before surgery, the sLRP for both the LV and RV was the ventricular apex. After surgery, the sLRP shifted. RV systolic displacement moved toward the mid segment of the lateral RV wall that remained fixed and LV systolic displacement moved toward the mid-apical segment of the interventricular septum. The RV basal segment moved toward the new RV sLRP but with less magnitude than before surgery. The RV apical segment displacement was inversed and moved toward the new sLRP. The conclusions of this study are: 1) after surgery, RV longitudinal strain decreased, 2) transverse shortening increased, 3) both the magnitude and direction of segment displacement changed. The RV lateral wall in contact with the sternum remains static, septal motion changes, and the apical septum moves toward the mid RV free wall causing PSM. Therefore, PSM depends on two factors: traction from the fixed RV lateral wall and the increase transverse fiber shortening (Fig. **1a**, **b**).

RV geometry is also altered after uncomplicated CS [32-34]. Rosner studied patients before and 9 months after CABG and assessed RV size, shape and function with 2D echo, tissue Doppler and cardiac MRI [34]. RV volumes and global RVEF did not change after surgery. However, cardiac MRI revealed a shortened RV lateral wall longitudinal length and wider apical and mid-ventricular diastolic and systolic diameters. These changes resulted in a more spherical RV shape that persisted for nine months.

4. RIGHT VENTRICULAR FUNCTION AND SURGICAL APPROACH

The aforementioned studies strongly suggest that PSM after CS is a consequence of altered regional RV contractile patterns. However, many of these studies lack a detailed description of the many surgical variables that may affect these changes. Surgery incorporates the extent of thoracotomy and pericardiotomy as well as myocardial protection strategies to limit ischemic injury. Cardioplegia components and their delivery methods (antegrade *vs.* retrograde) vary widely as does the use or non-use of cardiopulmonary bypass. Some physiological issues and details of surgical approaches that may influence regional RV contractile properties and PSM are reviewed below.

4.1. Ischemia, Cardioplegia, RCA Revascularization, RV Function and PSM

Several investigators have ruled out septal ischemia or infarction as a cause of PSM based on Thallium-201 scintigraphy, cardiac MR imaging and perfusion-gated SPECT imaging [23, 24]. These studies reported normal thickening, perfusion and viability of the paradoxical septum.

Reports of septal strain after CS are limited with varied results. Some studies have shown no reduction in septal strain in CABG patients [46, 53, 55]. Others have reported a decrease in septal strain [51, 52]. Donauer studied RV and septal strain in 30 patients undergoing CABG [52]. All patients had antegrade cold cardioplegia. After CS, there was a significant decrease in longitudinal strain with a simultaneous increase in circumferential strain of the RV lateral wall. There was also a decrease in peak systolic longitudinal septal strain after sternal closure. The authors suggested that further studies should investigate the effect of cardioplegia delivery, flow, duration and perfusion pressure on septal and RV contraction patterns after CS.

Cardioplegic components have been studied in reference to depressed RV function after CS. Zaboni analyzed 44 patients undergoing mitral valve repair using TAPSE to assess RV function. Patients had antero-lateral mini-thoracotomy or full sternotomy. Cardioplegia in the sternotomy group consisted of either cold blood (Buckberg protocol) or cold crystalloid (Custodial solution). In all patients, TAPSE was reduced. Cardioplegia components failed to impact the decreased TAPSE but mini thoracotomy minimalized it [56].

Cardioplegic delivery routes, however, seemed to affect septal function in one study using 3D speckled tracking. The investigators measured segmental strain of the septum and

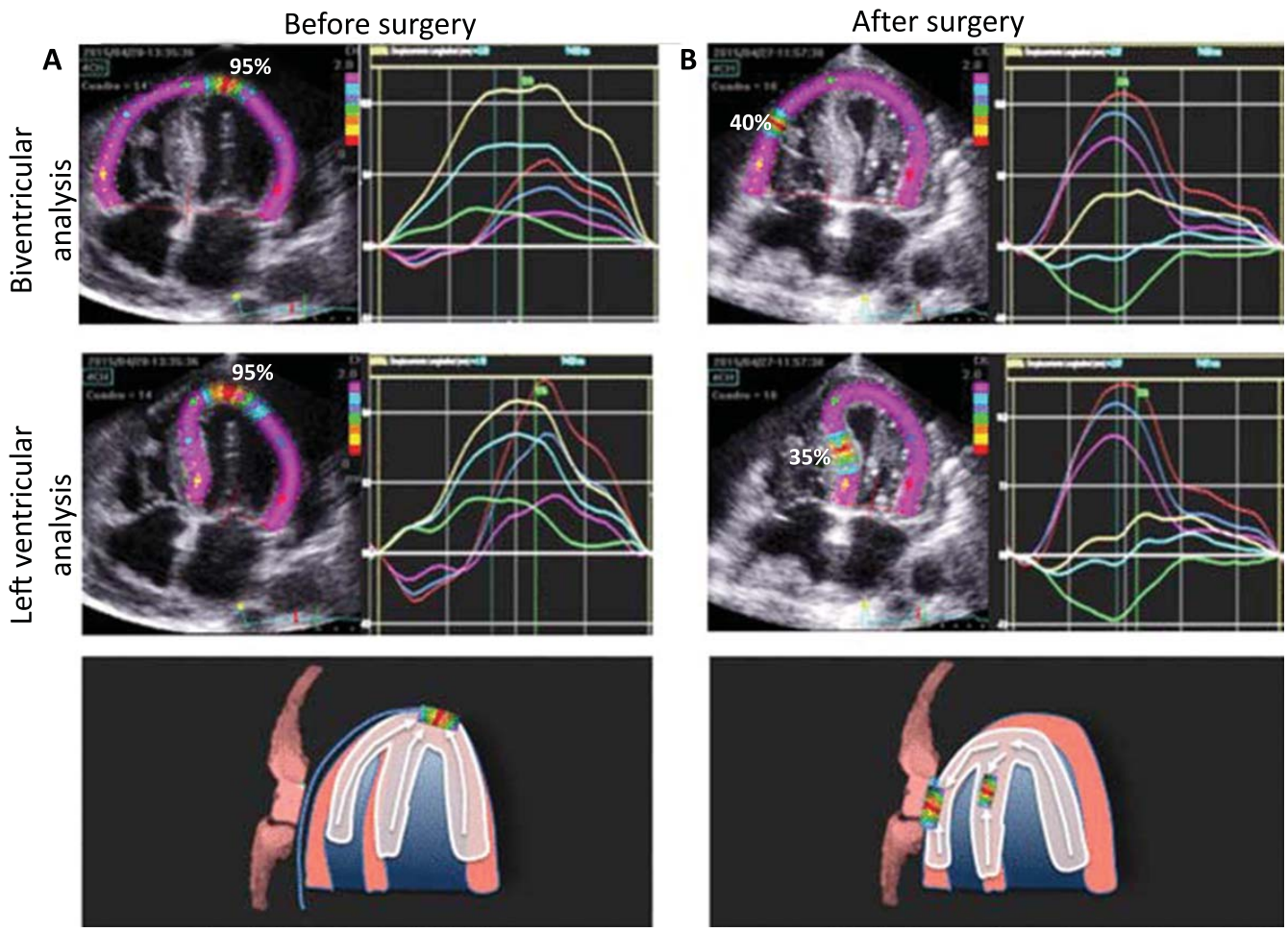

Fig. (1). Analysis of longitudinal displacement from the A4C view in a patient before (**a**) and after (**b**) cardiac surgery. a) Before surgery, the sLRP is located at the apex of the LV (in both biventricular and isolated LV analysis). All segments are moving towards the apex in systole. b) After the surgery, the sLRP is in the middle of RV free wall and septum (represented by the blue line) so that the apical segments of the RV free wall and septum invert their displacement (represented by the green line); the basal segments RV free wall and septum reduce displacement and apical segment VI (red) increases displacement pulled by RV. At the bottom, a scheme of this behavior (brown in diastole and white in systole) shows the relationship of cardiac structures and the anterior chest [32]. (*A higher resolution / colour version of this figure is available in the electronic copy of the article*).

lateral LV wall after CS comparing two modes of cold blood cardioplegia delivery: antegrade *vs.* antegrade plus retrograde *via* the coronary sinus (combined group) [57]. There was decreased mean radial strain of the septum in the antegrade group but not in the combined group, indicating preserved septal motion in the combined group.

Revascularization of the RCA plays no role in global RV dysfunction after CABG [11, 58]. Roshanali studied 240 patients before and after CABG. All patients underwent total revascularization. Patients with RCA revascularization (Group 1) were compared to patients without RCA revascularization (Group 2). TAPSE, TAV and RV strain were measured in all patients. Post-operative reductions of TAPSE, TAV and RV strain were independent of the number of grafts and RCA revascularization. There was no significant difference between the two groups in RV strain preoperatively [11].

4.2. Pericardiotomy, Thoracotomy, RV Function and PSM

Pericardial opening and its association with the onset of reduced RV longitudinal systolic function has been vigorously debated. Unsworth, using angle-dependent pulse wave Doppler, studied RV tissue systolic velocity in 9 patients undergoing CABG [15]. Intra-operative measurements were made with 2D TEE from the mid-esophageal 4-chamber view. RV longitudinal systolic velocity was reduced by 43% within three minutes after opening the pericardium. A later study from the same group included many CABG and valvular patients in whom intra-operative RV tissue Doppler sys-

tolic velocity by 2D TEE was also measured. Among patients who underwent minimally invasive surgery (robotic or small lateral thoracotomy), there was no significant change in post-operative RV longitudinal velocity or TAPSE, indicating that pericardial preservation is important. The authors concluded: "It is the complete opening of the pericardium, not cardiac surgery in general, that causes RV longitudinal axis velocity decline following CS" [59]. This finding was supported in a study by Dalen who reported a randomized trial that compared RV function after mini-sternotomy *vs.* full sternotomy in 40 patients undergoing AVR. Primary outcomes measured were TAPSE, RV tissue Doppler velocity, RV-FAC and basal and mid-RV transversal diameters on postoperative days 4 and 40. TAPSE fell in both groups but more with sternotomy. RV tissue Doppler velocity decreased only in the sternotomy group. FAC was equally decreased in both groups. RV dimensions did not change in either group and persisted from day 4 to 40 [60].

Ultrasound alignment with the lateral tricuspid annulus is difficult from the TEE mid-esophageal 4-chamber view and may lead to inaccurate velocity measurements [42, 44]. Most investigators studying the timing of surgical events with intra-operative 2D or 3D TEE and angle independent speckle tracking have placed the onset of reduced RV longitudinal shortening after CPB [44, 45, 52].

Lindqvist in 2012 investigated the consequent effect of pericardial repair on PSM and RV long axis shortening after CS [61]. Thirty patients scheduled for AVR were randomly allocated to pericardial repair (suture closure) *vs.* open pericardium. Half of the patients had the pericardium closed after operation. All patients had pre-op and one-week post-op echo and tissue Doppler measurements to assess RV long-axis systolic shortening and interventricular septal motion. One week after surgery, all patients had PSM and reduced RV longitudinal shortening. PSM directly correlated with decreased RV longitudinal shortening. PSM and RV longitudinal dysfunction were not normalized by pericardial repair. The authors suggested that PSM was likely a compensatory mechanism, for the decreased RV long-axis shortening-caused by surgery, to maintain a normal stroke volume.

Singh in 2020 studied intra-operative timing of the acute decline in RV long-axis function and global function in 109 patients undergoing CABG, valvular or CABG/valve combined surgery with myocardial cardioplegic arrest [45]. The study also assessed the influence of (1) the surgical approach (midline sternotomy, minimal upper hemi-sternotomy, right thoracotomy) and (2) the post-operative need for ionotropic support (51% in this study) on RV function after chest closure. All patients were monitored with a PA catheter and 2D TEE speckled tracking from the mid-esophageal 4-chamber view. TAPSE, RV strain and FAC were measured (1) after anesthetic induction before surgical incision, (2) after pericardiotomy, (3) after separation from CPB, and (4) following chest closure. All three metrics of RV function (TAPSE, RV strain and FAC) were reduced following CPB and were independent of the cardiac operation, surgical approach and extent of pericardiotomy. There was no difference in post-operative RV function between patients who received ionotropic support *versus* those who did not. The authors suggested that the change in RV function was perhaps related to imperfect myocardial protection or intracardiac air since less RV dysfunction was noted after CABG compared to open heart procedures.

4.3. Cardiac Translation and PSM

Joshi in 2009 used cardiac MRI to study PSM in 23 patients before and three months after CABG [19]. The motion of myocardial structures was assessed with a mid-left ventricular short-axis cine image relative to a stationary anterior reference point. After CABG, the septum moved anteriorly (PSM) in all 23 patients. Motion of the RV free wall was reduced (restricted), whereas systolic anterior movement of the lateral LV wall increased. It was concluded that the entire LV was translocated anteriorly in systole which explained postoperative PSM. There was a positive correlation between the degree of anterior movement of the ventricular septum and the RV ejection fraction. The pericardium was not closed. The restricted RV free wall was thought to be consistent with adhesions (three months post-op), a recognized phenomenon. The anterior motion occurred even while patients were lying supine in the MRI scanner; "The heart lifted against gravity during systole." Interestingly, Moya Mur has also described a post-operative fixed, immobile RV lateral free wall, but only three days after CS [32].

4.4. Cardiac Surgery with or without Cardiopulmonary Bypass

Michaux hypothesized that RV global systolic function, assessed by RV fractional area change and RV myocardial performance index, would be better preserved in off-pump CABG compared to on-pump CABG with blood cardioplegia in a randomized study of 50 patients utilizing 2D-Doppler before operation and three months later [62]. Lateral tricuspid annular velocities suggested persistently impaired systolic function at the level of the RV free wall in both groups that persisted for three months. However, global RV function was equally preserved at three months in both groups. Dyskinetic motion of the septum was low and not significantly different between on-pump *vs.* off-pump (2/25 *vs.* 1/25).

Khani in 2016 studied RV systolic function in patients undergoing off-pump CABG with standard 2D echocardiography. Right ventricular TAPSE, TDI, strain and strain rate were measured in all patients at the lateral tricuspid valve anulus before, 6 days and 3 months after surgery. Following surgery, right ventricular TAPSE, TDI, strain and strain rate all significantly decreased at 6 days and partially recovered at 3 months but did not improve to baseline levels. The authors concluded that RV function declines after off-pump CABG [63].

Another randomized trial used cardiac MRI to assess RV function comparing on and off pump CABG patients [64]. Antegrade cold crystalloid cardioplegia was used in on-pump cases. Early after surgery, RV stroke volume index decreased in both groups whether the RCA was grafted or not.

By six months, there was recovery of RV function in both the groups. The early reduction of function was due to a decrease in the RV end diastolic volume index (RVEDVI) while the RV end-systolic volume index remained unchanged. The authors suggest that even minor inflammation, effusion or hematoma associated with surgery could selectively impair RVEDVI.

Analysis of longitudinal displacement from the A4C view in a patient before (a) and after (b) cardiac surgery. a) Before surgery, the sLRP is located at the apex of the LV (in both biventricular and isolated LV analysis). All segments are moving towards the apex in systole. b) After the surgery, the sLRP is in the middle of RV free wall and septum (represented by the blue line) so that the apical segments of the RV free wall and septum invert their displacement (represented by the green line); the basal segments RV free wall and septum reduce displacement and apical segment VI (red) increases displacement pulled by RV. At the bottom, a scheme of this behavior (brown in diastole and white in systole) shows the relationship of cardiac structures and the anterior chest [32].

CONCLUSION

PSM is often seen after all types of cardiac surgery, including on or off-pump CABG, minimally invasive or robotic surgery and with valve surgery [63-65]. It sometimes resolves quickly or takes months to resolve or remains permanent. Prior studies suggest that PSM after uncomplicated CS is a consequence of altered regional RV contractile patterns and not a primary septal event as there is no loss of septal perfusion or thickening. After CS, there is a decrease in longitudinal shortening and an increase in transverse shortening of the endocardial and epicardial right ventricular muscle fibers, respectively. Two mechanisms have been proposed to explain these changes in fiber shortening. Several authors have reported that decreased RV longitudinal strain (deformation) results in reduced longitudinal shortening [32, 45, 46, 52], whereas other studies have reported decreased longitudinal displacement to be responsible for reduced longitudinal shortening [53].

Some investigators only reported longitudinal shortening and did not discuss strain or displacement [33, 48]. Increased RV transverse displacement has been observed and recorded by most investigators to explain the increased transverse shortening [32, 33, 48, 53], although decreased longitudinal strain and simultaneous increased transverse strain have been described [52]. Speckle tracking-derived different deviations in the magnitude and direction of longitudinal and transverse shortening suggest that these changes represent alterations in native muscle fiber contractility rather than RV tethering or global cardiac translation.

The medium segment of the RV free wall remains fixed and immobile after CS [19, 32]. This “anchor-like” effect produces changes in cardiac dynamics, reduced systolic tricuspid annular excursion, traction of the RV apical segment, LV apex, and septum (PSM) [32]. It is reasonable to speculate that the increased shortening of the epicardial transverse fibers (RV free wall to septum) may influence or reverse systolic septal motion (PSM) as a compensating mechanism to preserve global RV function, considering the RV free wall is fixed. Perhaps RV performance is augmented by a left ventricular-septal contractile contribution: the mechanical displacement of the septum into the RV that defines PSM [66]. Irrespective of this mechanistic hypothesis, changes in right ventricular regional contractile patterns after CS are temporally related to and responsible for PSM.

CONSENT FOR PUBLICATION

Not applicable.

FUNDING

None.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

ACKNOWLEDGEMENTS

Declared none.

REFERENCES

[1] Burggraf GW, Craige E. Echocardiographic studies of left ventricular wall motion and dimensions after valvular heart surgery. Am J Cardiol 1975; 35(4): 473-80. http://dx.doi.org/10.1016/0002-9149(75)90829-2 PMID: 1119397

[2] Force T, Bloomfield P, O’Boyle JE, *et al.* Quantitative two-dimensional echocardiographic analysis of motion and thickening of the interventricular septum after cardiac surgery. Circulation 1983; 68(5): 1013-20. http://dx.doi.org/10.1161/01.CIR.68.5.1013 PMID: 6311455

[3] Lehmann KG, Lee FA, McKenzie WB, *et al.* Onset of altered interventricular septal motion during cardiac surgery. Assessment by continuous intraoperative transesophageal echocardiography. Circulation 1990; 82(4): 1325-34. http://dx.doi.org/10.1161/01.CIR.82.4.1325 PMID: 2401066

[4] Reynolds HR, Tunick PA, Grossi EA, Dilmanian H, Colvin SB, Kronzon I. Paradoxical septal motion after cardiac surgery: A review of 3,292 cases. Clin Cardiol 2007; 30(12): 621-3. http://dx.doi.org/10.1002/clc.20201 PMID: 18069678

[5] Schroeder E, Marchandise B, Schoevaerdts JC, Kremer R. Paradoxical ventricular septal motion after cardiac surgery. Analysis of M-mode echocardiograms and follow-up in 324 patients. Acta Cardiol 1985; 40(3): 315-24. PMID: 3875201

[6] Grines CL, Bashore TM, Boudoulas H, Olson S, Shafer P, Wooley CF. Functional abnormalities in isolated left bundle branch block. The effect of interventricular asynchrony. Circulation 1989; 79(4): 845-53. http://dx.doi.org/10.1161/01.CIR.79.4.845 PMID: 2924415

[7] Roeleveld RJ, Marcus JT, Faes TJ, *et al.* Interventricular septal configuration at mr imaging and pulmonary arterial pressure in pulmonary hypertension. Radiology 2005; 234(3): 710-7. http://dx.doi.org/10.1148/radiol.2343040151 PMID: 15634939

[8] Ryan T, Petrovic O, Dillon JC, Feigenbaum H, Conley MJ, Armstrong WF. An echocardiographic index for separation of right ventricular volume and pressure overload. J Am Coll Cardiol 1985; 5(4): 918-27. http://dx.doi.org/10.1016/S0735-1097(85)80433-2 PMID: 3973294

[9] Voelkel AG, Pietro DA, Folland ED, Fisher ML, Parisi AF. Echocardiographic features of constrictive pericarditis. Circulation 1978; 58(5): 871-5. http://dx.doi.org/10.1161/01.CIR.58.5.871 PMID: 699252

[10] Weyman AE, Wann S, Feigenbaum H, Dillon JC. Mechanism of abnormal septal motion in patients with right ventricular volume overload: A cross-sectional echocardiographic study. Circulation 1976; 54(2): 179-86. http://dx.doi.org/10.1161/01.CIR.54.2.179 PMID: 939018

[11] Roshanali F, Yousefnia MA, Mandegar MH, Rayatzadeh H, Alinejad S. Decreased right ventricular function after coronary artery bypass grafting. Tex Heart Inst J 2008; 35(3): 250-5. PMID: 18941594

[12] Kang MK, Chang HJ, Cho IJ, *et al.* Echocardiographic investigation of the mechanism underlying abnormal interventricular septal motion after open heart surgery. J Cardiovasc Ultrasound 2014; 22(1): 8-13. http://dx.doi.org/10.4250/jcu.2014.22.1.8 PMID: 24753803

[13] Hedman A, Alam M, Zuber E, Nordlander R, Samad BA. Decreased right ventricular function after coronary artery bypass grafting and its relation to exercise capacity: A tricuspid annular motion-based study. J Am Soc Echocardiogr 2004; 17(2): 126-31. http://dx.doi.org/10.1016/j.echo.2003.10.023 PMID: 14752486

[14] Eslami B, Roitman D, Karp RB, Sheffield LT. The echocardiogram after pericardiectomy. Jpn Heart J 1979; 20(1): 1-5. http://dx.doi.org/10.1536/ihj.20.1 PMID: 449039

[15] Unsworth B, Casula RP, Kyriacou AA, *et al.* The right ventricular annular velocity reduction caused by coronary artery bypass graft surgery occurs at the moment of pericardial incision. Am Heart J 2010; 159(2): 314-22. http://dx.doi.org/10.1016/j.ahj.2009.11.013 PMID: 20152232

[16] Righetti A, Crawford MH, O'rourke RA, Schelbert H, Daily PO, Ross J Jr. Interventricular septal motion and left ventricular function after coronary bypass surgery: Evaluation with echocardiography and radionuclide angiography. Am J Cardiol 1977; 39(3): 372-7. http://dx.doi.org/10.1016/S0002-9149(77)80091-X PMID: 139103

[17] Feneley M, Kearney L, Farnsworth A, Shanahan M, Chang V. Mechanisms of the development and resolution of paradoxical interventricular septal motion after uncomplicated cardiac surgery. Am Heart J 1987; 114(1 Pt 1): 106-14. http://dx.doi.org/10.1016/0002-8703(87)90314-0 PMID: 3496774

[18] Waggoner AD, Shah AA, Schuessler JS, *et al.* Effect of cardiac surgery on ventricular septal motion: Assessment by intraoperative echocardiography and cross-sectional two-dimensional echocardiography. Am Heart J 1982; 104(6): 1271-8. http://dx.doi.org/10.1016/0002-8703(82)90156-9 PMID: 7148645

[19] Joshi SB, Salah AK, Mendoza DD, Goldstein SA, Fuisz AR, Lindsay J. Mechanism of paradoxical ventricular septal motion after coronary artery bypass grafting. Am J Cardiol 2009; 103(2): 212-5. http://dx.doi.org/10.1016/j.amjcard.2008.08.067 PMID: 19121438

[20] Buckberg G, Athanasuleas C, Saleh S. Septal myocardial protection during cardiac surgery for prevention of right ventricular dysfunction. Anadolu Kardiyol Derg 2008; 8 (Suppl. 2): 108-16. PMID: 19028643

[21] Choi SH, Choi SI, Chun EJ, *et al.* Abnormal motion of the interventricular septum after coronary artery bypass graft surgery: Comprehensive evaluation with MR imaging. Korean J Radiol 2010; 11(6): 627-31. http://dx.doi.org/10.3348/kjr.2010.11.6.627 PMID: 21076588

[22] Chouraqui P, Rabinowitz B, Livschitz S, Horoszowsky D, Kaplinsky E, Smolinsky A. Effects of antegrade *versus* combined antegrade/retrograde cardioplegia on postoperative septal wall motion in patients undergoing open heart surgery. Cardiology 1997; 88(6): 526-9. http://dx.doi.org/10.1159/000177403 PMID: 9397306

[23] Giubbini R, Rossini P, Bertagna F, *et al.* Value of gated SPECT in the analysis of regional wall motion of the interventricular septum after coronary artery bypass grafting. Eur J Nucl Med Mol Imaging 2004; 31(10): 1371-7. http://dx.doi.org/10.1007/s00259-004-1569-y PMID: 15221295

[24] Ozdemir S, Yener AÜ, Barutcu A, Tan YZ, Çelik F. The assessment of septal wall motion in patients undergoing CABG by myocardial perfusion-gated SPECT. Nucl Med Commun 2015; 36(7): 738-46. http://dx.doi.org/10.1097/MNM.0000000000000309 PMID: 25816362

[25] Akins CW, Boucher CA, Pohost GM. Preservation of interventricular septal function in patients having coronary artery bypass grafts without cardiopulmonary bypass. Am Heart J 1984; 107(2): 304-9. http://dx.doi.org/10.1016/0002-8703(84)90379-X PMID: 6607664

[26] Wranne B, Pinto FJ, Siegel LC, Miller DC, Schnittger I. Abnormal postoperative interventricular motion: New intraoperative transesophageal echocardiographic evidence supports a novel hypothesis. Am Heart J 1993; 126(1): 161-7. http://dx.doi.org/10.1016/S0002-8703(07)80024-X PMID: 8322660

[27] Jennings RB, Reimer KA. The cell biology of acute myocardial ischemia. Annu Rev Med 1991; 42: 225-46. http://dx.doi.org/10.1146/annurev.me.42.020191.001301 PMID: 2035969

[28] Turer AT, Hill JA. Pathogenesis of myocardial ischemia-reperfusion injury and rationale for therapy. Am J Cardiol 2010; 106(3): 360-8. http://dx.doi.org/10.1016/j.amjcard.2010.03.032 PMID: 20643246

[29] Codreanu I, Pegg TJ, Selvanayagam JB, *et al.* Details of left ventricular remodeling and the mechanism of paradoxical ventricular septal motion after coronary artery bypass graft surgery. J Invasive Cardiol 2011; 23(7): 276-82. PMID: 21725122

[30] Siddiqui MM, Jalal A, Sherwani M, Ahmad MZ. Right ventricular dysfunction after coronary artery bypass grafting is a reality of unknown cause and significance. Heart Surg Forum 2012; 15(4): E185-8. http://dx.doi.org/10.1532/HSF98.20111142 PMID: 22917821

[31] Yadav H, Unsworth B, Fontana M, *et al.* Selective right ventricular impairment following coronary artery bypass graft surgery. Eur J Cardiothorac Surg 2010; 37(2): 393-8. http://dx.doi.org/10.1016/j.ejcts.2009.08.004 PMID: 19758814

[32] Moya Mur JL, García Martín A, García Lledó A, *et al.* Geometrical and functional cardiac changes after cardiac surgery: A phisiopatological explanation based on speckle tracking. Int J Cardiovasc Imaging 2018; 34(12): 1905-15. http://dx.doi.org/10.1007/s10554-018-1424-z PMID: 30073630

[33] Raina A, Vaidya A, Gertz ZM, Susan Chambers , Forfia PR. Marked changes in right ventricular contractile pattern after cardiothoracic surgery: Implications for post-surgical assessment of right ventricular function. J Heart Lung Transplant 2013; 32(8): 777-83. http://dx.doi.org/10.1016/j.healun.2013.05.004 PMID: 23773508

[34] Rösner A, Avenarius D, Malm S, *et al.* Changes in right ventricular shape and deformation following coronary artery bypass surgery-insights from echocardiography with strain rate and magnetic resonance imaging. Echocardiography 2015; 32(12): 1809-20. http://dx.doi.org/10.1111/echo.12973 PMID: 26010320

[35] Fransen EJ, Maessen JG, Hermens WT, Glatz JF. Demonstration of ischemia-reperfusion injury separate from postoperative infarction in coronary artery bypass graft patients. Ann Thorac Surg 1998; 65(1): 48-53. http://dx.doi.org/10.1016/S0003-4975(97)01036-9 PMID: 9456094

[36] Kovács A, Lakatos B, Tokodi M, Merkely B. Right ventricular mechanical pattern in health and disease: beyond longitudinal shortening. Heart Fail Rev 2019; 24(4): 511-20. http://dx.doi.org/10.1007/s10741-019-09778-1 PMID: 30852772

[37] Brown SB, Raina A, Katz D, Szerlip M, Wiegers SE, Forfia PR. Longitudinal shortening accounts for the majority of right ventricular contraction and improves after pulmonary vasodilator therapy in normal subjects and patients with pulmonary arterial hypertension. Chest 2011; 140(1): 27-33. http://dx.doi.org/10.1378/chest.10-1136 PMID: 21106653

[38] Haber I, Metaxas DN, Geva T, Axel L. Three-dimensional systolic kinematics of the right ventricle. Am J Physiol Heart Circ Physiol 2005; 289(5): H1826-33. http://dx.doi.org/10.1152/ajpheart.00442.2005 PMID: 15964922

[39] Kaul S, Tei C, Hopkins JM, Shah PM. Assessment of right ventricular function using two-dimensional echocardiography. Am Heart

J 1984; 107(3): 526-31.
http://dx.doi.org/10.1016/0002-8703(84)90095-4 PMID: 6695697

[40] Meluzín J, Spinarová L, Bakala J, *et al.* Pulsed doppler tissue imaging of the velocity of tricuspid annular systolic motion; a new, rapid, and non-invasive method of evaluating right ventricular systolic function. Eur Heart J 2001; 22(4): 340-8.
http://dx.doi.org/10.1053/euhj.2000.2296 PMID: 11161953

[41] Saxena N, Rajagopalan N, Edelman K, López-Candales A. Tricuspid annular systolic velocity: A useful measurement in determining right ventricular systolic function regardless of pulmonary artery pressures. Echocardiography 2006; 23(9): 750-5.
http://dx.doi.org/10.1111/j.1540-8175.2006.00305.x PMID: 16999693

[42] David JS, Tousignant CP, Bowry R. Tricuspid annular velocity in patients undergoing cardiac operation using transesophageal echocardiography. J Am Soc Echocardiogr 2006; 19(3): 329-34.
http://dx.doi.org/10.1016/j.echo.2005.09.013 PMID: 16500497

[43] Alam M, Hedman A, Nordlander R, Samad B. Right ventricular function before and after an uncomplicated coronary artery bypass graft as assessed by pulsed wave Doppler tissue imaging of the tricuspid annulus. Am Heart J 2003; 146(3): 520-6.
http://dx.doi.org/10.1016/S0002-8703(03)00313-2 PMID: 12947373

[44] Bitcon CJ, Tousignant C. The effect of pericardial incision on right ventricular systolic function: A prospective observational study. Can J Anaesth 2017; 64(12): 1194-201.
http://dx.doi.org/10.1007/s12630-017-0972-3 PMID: 28983853

[45] Singh A, Huang X, Dai L, *et al.* Right ventricular function is reduced during cardiac surgery independent of procedural characteristics, reoperative status, or pericardiotomy. J Thorac Cardiovasc Surg 2020; 159(4): 1430-1438.e4.
http://dx.doi.org/10.1016/j.jtcvs.2019.04.035 PMID: 31133354

[46] Rong LQ, Yum B, Abouzeid C, *et al.* Echocardiographic predictors of intraoperative right ventricular dysfunction: A 2D and speckle tracking echocardiography study. Cardiovasc Ultrasound 2019; 17(1): 11.
http://dx.doi.org/10.1186/s12947-019-0161-3 PMID: 31174537

[47] Tamborini G, Muratori M, Brusoni D, *et al.* Is right ventricular systolic function reduced after cardiac surgery? A two- and three--dimensional echocardiographic study. Eur J Echocardiogr 2009; 10(5): 630-4.
http://dx.doi.org/10.1093/ejechocard/jep015 PMID: 19252190

[48] Keyl C, Schneider J, Beyersdorf F, *et al.* Right ventricular function after aortic valve replacement: A pilot study comparing surgical and transcatheter procedures using 3D echocardiography. Eur J Cardiothorac Surg 2016; 49(3): 966-71.
http://dx.doi.org/10.1093/ejcts/ezv227 PMID: 26130077

[49] Maffessanti F, Gripari P, Tamborini G, *et al.* Evaluation of right ventricular systolic function after mitral valve repair: A two-dimensional Doppler, speckle-tracking, and three-dimensional echocardiographic study. J Am Soc Echocardiogr 2012; 25(7): 701-8.
http://dx.doi.org/10.1016/j.echo.2012.03.017 PMID: 22542273

[50] Grønlykke L, Korshin A, Holmgaard F, *et al.* Marked reduction in right ventricular longitudinal contraction after cardiopulmonary bypass in patients with preserved right ventricular output. J Cardiothorac Vasc Anesth 2019; 33: S117-9.
http://dx.doi.org/10.1053/j.jvca.2019.07.071

[51] Abdelmoneum MS, Elmeligy , Eldarky EA, Mohamad MM. Evaluation of the effects of coronary bypass grafting on the right ventricular function using speckle tracking echocardiography. J Cardiol Cardiovasc Med 2019; 4: 236-41.
http://dx.doi.org/10.29328/journal.jccm.1001075

[52] Donauer M, Schneider J, Jander N, Beyersdorf F, Keyl C. Perioperative changes of right ventricular function in cardiac surgical patients assessed by myocardial deformation analysis and 3-dimensional echocardiography. J Cardiothorac Vasc Anesth 2020; 34(3): 708-18.
http://dx.doi.org/10.1053/j.jvca.2019.08.026 PMID: 31540753

[53] Korshin A, Grønlykke L, Holmgaard F, *et al.* Right ventricular transverse displacement increases following cardiac surgery: Possibly compensating loss in tricuspid annular plane systolic excursion (TAPSE). J Clin Monit Comput 2020; 34(6): 1139-48.
http://dx.doi.org/10.1007/s10877-020-00466-2 PMID: 31983013

[54] Salvo GD, Pergola V, Fadel B, Bulbul ZA, Caso P. Strain echocardiography and myocardial mechanics: from basics to clinical applications. J Cardiovasc Echogr 2015; 25(1): 1-8.
http://dx.doi.org/10.4103/2211-4122.158415 PMID: 28465921

[55] Toyoda T, Akasaka T, Watanabe N, *et al.* Evaluation of abnormal motion of interventricular septum after coronary artery bypass grafting operation: Assessment by ultrasonic strain rate imaging. J Am Soc Echocardiogr 2004; 17(7): 711-6.
http://dx.doi.org/10.1016/j.echo.2004.03.033 PMID: 15220894

[56] Zanobini M, Loardi C, Poggio P, *et al.* The impact of pericardial approach and myocardial protection onto postoperative right ventricle function reduction. J Cardiothorac Surg 2018; 13(1): 55.
http://dx.doi.org/10.1186/s13019-018-0726-5 PMID: 29866151

[57] Bhaya M, Sudhakar S, Sadat K, *et al.* Effects of antegrade *versus* integrated blood cardioplegia on left ventricular function evaluated by echocardiographic real-time 3-dimensional speckle tracking. J Thorac Cardiovasc Surg 2015; 149(3): 877-84.e1-5.

[58] Becker M, Robbers L, Brouwer WP, *et al.* A cardiovascular magnetic resonance study on the short and long-term effects of coronary artery bypass graft surgery on the right systolic function. Madridge J Cardiol 2017; 2(1): 14-20.
http://dx.doi.org/10.18689/mjc-1000105

[59] Unsworth B, Casula RP, Yadav H, *et al.* Contrasting effect of different cardiothoracic operations on echocardiographic right ventricular long axis velocities, and implications for interpretation of post-operative values. Int J Cardiol 2013; 165(1): 151-60.
http://dx.doi.org/10.1016/j.ijcard.2011.08.031 PMID: 21917325

[60] Dalén M, Oliveira Da Silva C, Sartipy U, *et al.* Comparison of right ventricular function after ministernotomy and full sternotomy aortic valve replacement: A randomized study. Interact Cardiovasc Thorac Surg 2018; 26(5): 790-7.
http://dx.doi.org/10.1093/icvts/ivx422 PMID: 29325064

[61] Lindqvist P, Holmgren A, Zhao Y, Henein MY. Effect of pericardial repair after aortic valve replacement on septal and right ventricular function. Int J Cardiol 2012; 155(3): 388-93.
http://dx.doi.org/10.1016/j.ijcard.2010.10.049 PMID: 21112100

[62] Michaux I, Filipovic M, Skarvan K, *et al.* A randomized comparison of right ventricular function after on-pump *versus* off-pump coronary artery bypass graft surgery. J Thorac Cardiovasc Surg 2011; 141(2): 361-7.
http://dx.doi.org/10.1016/j.jtcvs.2010.02.023 PMID: 20381082

[63] Khani M, Hosseintash M, Foroughi M, Naderian M, Khaheshi I. Assessment of the effect of off-pump coronary artery bypass (OPCAB) surgery on right ventricle function using strain and strain rate imaging. Cardiovasc Diagn Ther 2016; 6(2): 138-43.
http://dx.doi.org/10.21037/cdt.2016.02.03 PMID: 27054103

[64] Pegg TJ, Selvanayagam JB, Karamitsos TD, *et al.* Effects of off-pump *versus* on-pump coronary artery bypass grafting on early and late right ventricular function. Circulation 2008; 117(17): 2202-10.
http://dx.doi.org/10.1161/CIRCULATIONAHA.107.735621 PMID: 18413501

[65] Michaux I, Filipovic M, Skarvan K, *et al.* Effects of on-pump *versus* off-pump coronary artery bypass graft surgery on right ventricular function. J Thorac Cardiovasc Surg 2006; 131(6): 1281-8.
http://dx.doi.org/10.1016/j.jtcvs.2006.01.035 PMID: 16733158

[66] Goldstein JA, Tweddell JS, Barzilai B, Yagi Y, Jaffe AS, Cox JL. Importance of left ventricular function and systolic ventricular interaction to right ventricular performance during acute right heart ischemia. J Am Coll Cardiol 1992; 19(3): 704-11.
http://dx.doi.org/10.1016/S0735-1097(10)80296-7 PMID: 1538031