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Unraveling main perioperative management principles for patients with diastolic dysfunction

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Abstract

Background. The assessment of diastolic function is often overshadowed by the evaluation of significant valve anomalies and systolic function, which is determined by ejection fraction. To accurately predict a surgical procedure's outcome, it is beneficial to consider the diastolic evaluation findings alongside the conventional perioperative risk assessment.

Aim. This article aims to provide a thorough summary of the diagnostic methods, treatment options, and how the perioperative pathway can be optimized for patients with diastolic dysfunction (DD).

Methods. The literature used for this review was selected using "Elsevier", "Pubmed", and "UptoDate" databases. The search used the following keywords and their combinations: diastolic dysfunction perioperatively, diastolic heart failure, diastolic dysfunction and anaesthesia, perioperative hypertension, and diastolic dysfunction ESC.

Results. Echocardiography is a crucial diagnostic tool for assessing DD, providing detailed information about the structure and function of the heart. Talking about the choice of anesthetic technique, sevoflurane better preserves diastolic relaxation during spontaneous ventilation than propofol and isoflurane has no adverse lusitropic effects and do not exacerbate preexisting DD. Several studies have demonstrated that reducing afterload, isoflurane, sevoflurane, and desflurane can enhance left ventricular relaxation. Moreover, adequate blood pressure, heart rate, rhythm, fluid status, and avoidance of noticeable variations in hemodynamics and volume are the cornerstones of the perioperative care of DD.

Conclusion. Anesthesia plans should be individualized to minimize the potential impact on diastolic function and maintain optimal hemodynamic stability. Close monitoring and vigilant management of fluid balance, heart rate, and blood pressure are paramount intraoperatively.

Keywords: diastolic heart failure, diastolic dysfunction, anaesthesia, diastolic dysfunction perioperatively.

1. Introduction

The foundation of anesthesiology practice is preoperative cardiac assessment. Despite its importance, the assessment of diastolic function is often overshadowed by the evaluation of significant valve anomalies and systolic function, which is determined by ejection fraction. 21% of the population exhibits asymptomatic mild left ventricle diastolic dysfunction, while 7% have moderate or severe diastolic dysfunction. Identifying patients at risk for diastolic dysfunction can prevent increased risk for perioperative morbidity and mortality. Hence, to accurately predict a surgical procedure's outcome, it is beneficial to consider the diastolic evaluation findings alongside the conventional perioperative risk assessment. It is obligatory to control hypertension, avoid tachycardia and myocardial ischemia, preserve sinus rhythm and normovolemia, and keep the heart rate below diastolic blood pressure. This article aims to provide a thorough summary of the diagnostic methods, treatment options, and how the perioperative pathway can be optimized for patients with diastolic dysfunction.

2. Methods

The literature used for this review was selected using “Elsevier”, “Pubmed”, and “UptoDate” databases. The search used the following keywords and their combinations: diastolic dysfunction perioperatively, diastolic heart failure, diastolic dysfunction, anaesthesia, perioperative hypertension, and diastolic dysfunction ESC. Inclusion criteria: 1) full-text articles; 2) the article analyzes diastolic dysfunction etiology, pathogenesis, clinical symptoms, diagnostics, treatment and links with perioperative care or anaesthesia; 3) articles in English. Criteria for rejecting articles: 1) the article is not related to diastolic dysfunction and perioperative care or anesthesia; 2) the article was published earlier than 2006. The selected publications were written in English and published in 2006 - 2023 years. The majority of the articles were published in the last 10 years. This literature review included 33 articles published in peer-reviewed journals that met the inclusion and exclusion criteria.

3. Results

3.1 Ethiology

DD refers to an impairment in the relaxation phase of the heart's cardiac cycle, specifically during diastole when the heart is filling with blood [1]. One primary contributor is hypertension, as sustained high blood pressure can lead to structural changes in the heart muscle, making it less compliant and impairing its ability to relax [2]. Other cardio-vascular factors include myocardial ischemia, where inadequate blood supply compromises heart muscle function [3]. Additionally, conditions such as hypertrophic cardiomyopathy and restrictive cardiomyopathy, characterized by abnormal thickening or stiffening of the heart walls, can contribute to DD [4]. Non-cardiovascular causes may include diabetes, obesity, and ageing, all of which can affect the elasticity and function of the heart muscle. Some authors report that approximately 34 % of diabetic patients experience DD [5].

3.2 Diastolic dysfunction and atrial fibrillation

DD can significantly influence the development and progression of atrial fibrillation (AF), a common and potentially severe cardiac arrhythmia [6]. As the impaired relaxation of the heart during diastole occurs, there is an elevated pressure within the left atrium, leading to increased strain on the atrial walls [7]. This persistent pressure overload can contribute to atrial structural remodeling, promoting fibrosis and hypertrophy. DD is particularly relevant in the

context of AF, as it often results in elevated left ventricular filling pressures and atrial dilation, both of which are known triggers for arrhythmia [8]. Moreover, the altered hemodynamics associated with DD can lead to abnormal atrial stretch, triggering electrical abnormalities in the atrial tissue [6,8]. The combination of impaired relaxation during diastole and irregular heart rhythm in AF can reduce cardiac output. As the heart struggles to fill and pump blood efficiently, the body may experience inadequate perfusion, contributing to symptoms of diastolic heart failure [9–11]. The presence of exercise intolerance, exertional dyspnea, and pulmonary edema with average ejection fraction, alongside symptoms and signs resembling chronic obstructive pulmonary disease, may suggest the company of diastolic heart failure which is a symptomatic form of DD [12,13]. This hypothesis can be further supported by echocardiographic or angiographic data (table 1).

Table 1. *Criteria for diastolic heart failure. All three measures are required for the diagnosis of diastolic heart failure [9].*

Criteria for diastolic heart failure	
1. Signs and symptoms of congestive heart failure	Pulmonary edema, effort dyspnea, orthopnea
2. Normal or mildly reduced ejection fraction	Ejection fraction >50 %, normal left ventricle end diastolic volume
3. Abnormal left ventricle relaxation; echocardiographic tissue Doppler (E/E' >15); left ventricular end-diastolic pressure >16 mmHg on cardiac catheterization; Biomarkers NT-proBNP >220 pg/ml or BNP >200 pg/ml.	

NT-proBNP: N-terminal pro-B-type Natriuretic Peptide, BNP: brain natriuretic peptide (B-type Natriuretic Peptide)

3.3 Grading diastolic dysfunction

DD is often graded based on the severity of impairment in the relaxation and filling of the heart during diastole. During the initial phase (Grade I), there is a reduction in early filling and an early transmitral pressure gradient due to decreased left ventricular relaxation, frequently resulting from hypertrophy [4,14]. Another stage often involves increased left atrial pressure due to delayed or incomplete relaxation to compensate for the usual stroke volume. The left atrium remodels to restore the pressure gradient and fill against the abnormal left ventricular end-diastolic pressure (Grade II) [4]. The term "pseudonormal" refers to a condition in which left atrial pressure increases and early diastolic filling becomes prominent again. In echocardiography, this diastolic filling seems identical to normal diastolic function [15]. Nevertheless, when the compliance of the left ventricle decreases even more, the initial filling of the left ventricle can only occur when there is an extremely high-pressure gradient across the transmitral valve [16,17]. However, this filling quickly ceases as the pressures in the two chambers become equal. Consequently, a restrictive diastolic filling pattern emerges, which can be classified as grade III or IV depending on whether the restricted filling can be reversed by reducing preload (such as during the Valsalva maneuver). It is important to note that an irreversible restrictive DD is associated with a significantly unfavorable prognosis in terms of mortality [4].

3.4 Central venous and pulmonary artery catheters

Some authors recommend a central venous catheter for vasopressors, inotropes, and other drugs that

cannot be given peripherally [18–20]. However, others believe that using a central venous catheter carries some risk, fluid responsiveness is difficult to predict and is not very beneficial for perioperative care of individuals with DD [18–20].

There is still much disagreement in the literature on the utility of a pulmonary artery catheter as a monitoring tool. Using a pulmonary artery catheter is sometimes criticized for not changing patient outcomes. Also, it is frequently utilized as a salvage or rescue therapy [21]. Other authors believe a pulmonary artery catheter may be justified perioperatively under the right circumstances, especially in individuals with severe DD [9]. A pulmonary artery catheter enables the evaluation of volume responsiveness through sequential assessments of cardiac output following an intervention. This valuable tool equips the attending physician with essential data to aid in selecting appropriate inotropes and vasopressors [22]. However, the literature also describes the misinterpretation of data associated with using pulmonary artery catheters. Consequently, if one lacks proficiency in the placement, utilization, and interpretation of such catheters, it is advisable to refrain from their use [18].

3.4.1 Echocardiography

Echocardiography is a crucial diagnostic tool for assessing DD, providing detailed information about the structure and function of the heart during the relaxation and filling phases of the cardiac cycle [16].

By evaluating the chamber pressure–volume relationship, invasive cardiac catheterization is the gold standard for defining DD. Nonetheless, echocardiography has mostly replaced cardiac catheterisation as the preferred method for quickly detecting DD due to its non-invasiveness and accessibility in the hospital [23]. Echocardiographic assessment begins by evaluating the overall morphology of the left atrium and left ventricle. This evaluation is most effectively conducted using the mid esophageal 4-chamber view in transesophageal echocardiography or the apical 4-chamber view in transthoracic echocardiography [17]. Left atrial dilation may indicate remodelling caused by elevated pressure within the left atrium, whereas left ventricle hypertrophy may indicate reduced compliance of the left ventricle [7,24]. Long-term hypertension is one of the leading causes of DD, and between 50 and 66 % of patients will have thicker walls. The four most often applied methods are colour M-mode propagation velocity, Doppler tissue imaging of the mitral annulus, transmitral inflow, and pulmonary venous inflow [25].

In critically ill patients, echocardiography surpasses measurements obtained from a pulmonary artery catheter as a means for assessing volume responsiveness [21]. By examining mitral inflow patterns and utilizing 2-dimensional measurements of the left ventricular end-diastolic area, echocardiography can accurately predict changes in cardiac output. Before surgery, a transthoracic echocardiography (TTE) evaluation can be performed [26]. However, the utility of transesophageal echocardiography (TEE) during intraoperative procedures may be limited, especially in cases involving significant volume shifts within the chest or abdominal cavity, which can make TTE challenging [13]. In such instances, TEE may be the preferred modality. Not only does TEE assist in guiding volume administration and addressing the common clinical questions of whether more volume is needed or if enough has been given, but it also enables real-time assessment of diastolic function by monitoring dynamic changes in pre-load and after-load [17,27]. Therefore, it is recommended to use TEE in complex cases with severe diseases and when substantial volume shifts are expected [23,25].

3.4.2 Choice of anesthetic technique

The potential benefits of avoiding general anesthesia and using neuraxial or regional anesthetic techniques in patients with DD are still uncertain due to the lack of literature supporting any specific recommendations [9,26].

T. Ryu et al. claim that spinal anesthesia is less favorable than epidural anesthesia. Epidural anesthesia induces slower hemodynamic changes due to its gradual onset and the gradual reduction of sympathetic tone [9,26]. Furthermore, patients with DD may have additional medical conditions such as coronary artery disease or AF, which may necessitate systemic anticoagulation [6,24]. In such cases, it is crucial for clinicians to thoroughly review the patient's medications and determine whether any should be discontinued to avoid vertebral canal haematoma [28].

Patients with poor functional capacity are at a higher risk when undergoing general anesthesia [14]. It is advisable to opt for intravenous induction and maintenance using a combination of balanced anesthesia with volatile agents and opioids [9,29]. The impact of inhaled anesthetics on left ventricular compliance and relaxation has been the subject of several investigations. Using Doppler echocardiography in individuals with established DD, one study discovered that sevoflurane administration significantly enhanced early left ventricular relaxation (measured by E' velocity) [13]. Also, sevoflurane better preserves diastolic relaxation during spontaneous ventilation than propofol [9]. Neuhauser et al. showed that isoflurane had no adverse lusitropic effects and did not exacerbate preexisting DD [30]. Several studies have demonstrated that reducing afterload, isoflurane, sevoflurane, and desflurane can enhance left ventricular relaxation [4].

3.4.3 Treatment

No significant multicenter randomized controlled study has been conducted to analyze the perioperative DD management particularly. American Heart Association guidelines suggest taking care of patients with DD by lowering pulmonary venous pressure, sustaining synchronous atrial contraction and extending diastole time by heart rate regulation [31]. Reducing diastolic volume and diastolic pressure can be accomplished by lowering central blood volume with nitrates, restricting fluid and sodium intake or using diuretics, and downregulating the renin-angiotensin-aldosterone system with angiotensin-converting enzyme inhibitors or angiotensin receptor blockers [32].

The systolic blood pressure should remain within 10-20 % of the baseline value, while the pulse pressure should be kept below the diastolic blood pressure. The combination of low doses of nitroglycerin and phenylephrine titration could be helpful for pulse pressure control, but the presence of either agent individually can potentially worsen hemodynamics [9].

Two crucial aspects of acute perioperative treatment for individuals with DD are the avoidance of tachycardia and the preservation of sinus rhythm. Tachycardia shortens the coronary perfusion time and raises myocardial oxygen demand. Also, tachycardia preferentially reduces diastole in the cardiac cycle, which can result in a decline in left ventricle filling, stroke volume, and cardiac output [2,13,33]. Regarding reducing heart rate, rate control medications like beta and calcium channel blockers are great options. They may be helpful for both acute and long-term treatment [14,26]. Atrial arrhythmias such as AF have a noticeable impact on hemodynamic stability as these patients frequently have poor passive filling and primarily depend on atrial contraction for cardiac output. Anesthesiologists must act fast to identify this and consider the early use of cardioversion to restore sinus rhythm compared to patients without DD [12,14].

In the postoperative period, hypoxemia, pulmonary edema, and AF are the most common complications due to decompensation. These events can happen very fast, even for patients who seem to be stable, therefore careful monitoring is essential. Also, it is crucial to avoid perioperative risk factors that can lead from DD to diastolic heart failure: shivering, anemia, hypoxia, electrolytic imbalance, deterioration in DD, myocardial ischemia, hypovolemia or hypervolemia (extreme volume shifting), tachycardia, rhythms other than sinus, postoperative sympathetic stimulation and hypertensive crisis [9].

In conclusion, adequate blood pressure, heart rate, rhythm, fluid status, and avoidance of noticeable variations in hemodynamics and volume are the cornerstones of the perioperative care of DD [9].

4. Conclusions

Anesthesiologists in perioperative settings may face significant clinical difficulties because of the complicated and growing issue of DD. As explored throughout this article, DD presents unique challenges and considerations for anesthesiologists when caring for patients undergoing surgical procedures or medical interventions. Careful preoperative assessment is essential, including a thorough understanding of the patient's medical history, echocardiographic findings, and overall cardiovascular status. Anesthesia plans should be individualized to minimize the potential impact on diastolic function and maintain optimal hemodynamic stability. Echocardiography can be a beneficial tool in determining the degree of dysfunction and volume status, which can influence clinical decision-making. Close monitoring and vigilant management of fluid balance, heart rate, and blood pressure are paramount intraoperatively. Techniques such as goal-directed fluid therapy and advanced monitoring tools can aid in achieving and maintaining hemodynamic goals while avoiding exacerbation of DD.

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