Academiejaar 2013 – 2014

A pathophysiological approach towards right ventricular function and failure

Michael Vandenheuvel

Promotor: Prof. dr. P. Wouters

Co-promotor: Prof. dr. S. De Hert

Masterproef voorgedragen in de master in de specialistische geneeskunde anesthesie en reanimatie

UNIVERSITEIT GENT

FACULTEIT GENEESKUNDE EN GEZONDHEIDSWETENSCHAPPEN

Academiejaar 2013 – 2014

A pathophysiological approach towards right ventricular function and failure

Michael Vandenheuvel

Promotor: Prof. dr. P. Wouters

Co-promotor: Prof. dr. S. De Hert

Masterproef voorgedragen in de master in de specialistische geneeskunde anesthesie en reanimatie

Unstructured Abstract

The scope of this review is to provide a pathophysiologic summary on perioperative right ventricular function and failure. In recent decades, the importance of right ventricular function in the perioperative period has been established. However, much of our current knowledge on the management of this clinical entity is based on extrapolation of results from left ventricular research, although biventricular physiology is known to be markedly different in many aspects. Here, based on a thorough literature search, we review theoretical as well as practical aspects of perioperative right ventricular failure. After underlining the importance of this topic, we review basic right ventricular anatomy and physiology, with an emphasis on the role of ventricular interaction. Next, potential causes of perioperative right ventricular failure are discussed. The emphasis of this review is on the perioperative anaesthetic considerations, ranging from preoperative assessment over intraoperative monitoring to specific contemporary therapeutic options of perioperative right ventricular failure.

Keywords

Right ventricle, review, right ventricular failure, physiology, pathophysiology, perioperative management.

Introduction

The primary function of the right ventricle (RV) is to facilitate blood flow through the lung. In order to achieve this, the right ventricle is anatomically and physiologically designed as a volume pump, which keeps central venous pressures low. Until recently, its importance was frequently minimised. Studies from the 1950's seemed to indicate that cauterisation of the RV free wall resulted in only modest changes in cardiac output and central venous pressure.[1,2] In 1971, Brooks et al. demonstrated that isolated RV ischaemia had virtually no impact on RV developed pressure, left ventricular (LV) developed pressure or cardiac output, because a slightly elevated central venous pressure provided the driving force needed to create sufficient blood flow through the lungs.[3] This phenomenon is applied in many congenital cardiac heart surgery strategies, such as for instance the Fontan sequence. Brooks however already noted that in the presence of an even small elevation in pulmonary artery pressures, cardiac output could not be maintained.[3] Thus, if LV filling pressure or pulmonary vascular resistance is too high, central venous pressure will not be able to provide adequate pulmonary arterial flow, and a normal RV function becomes critically important.

Over the last decades, numerous studies have underscored the importance of RV function. It was shown that RV dysfunction is an important predictor of overall survival and morbidity in various clinical situations.[4-7] Right heart failure is the main cause of death in pulmonary hypertension.[8] Perioperative mortality is even higher in RV failure then in LV failure and it is important to note that progressive RV failure has a similar incidence as LV failure.[9-11] In a French national survey, overall prevalence of pulmonary arterial hypertension has been determined to 15 per million.[12] As such, RV research was proclaimed a cardiovascular research priority in 2006 by the USA National Institutes of Health.[13]

RV failure is an underdiagnosed entity, also in the non-cardiac surgery perioperative setting.[9-11] Here, we present a review of multiple specific aspects of perioperative RV failure, especially aimed at the non-cardiac anaesthesiologist who may be confronted with RV failure. We discuss the anatomic and physiologic peculiarities, practical assessment and specific perioperative measures useful in perioperative RV failure. We searched pubmed, embase and web of science databases using (combinations of) the following search terms: ‘right ventricular function’, ‘right ventricular failure’, ‘perioperative’, ‘anatomy’, ‘physiology’, ‘epidemiology’, ‘ventricular interaction’, ‘volume overload’, ‘pressure overload’, ‘ischaemia’, ‘perioperative assessment’, ‘echocardiography’, ‘pulmonary artery catheter’, ‘pulse pressure variation’, ‘afterload reduction’, ‘mechanical ventilation’, ‘inotropic support’ and ‘vasopression’.

Basic anatomy and physiology

Anatomy

Compared to the LV, the right sided myocardium is thin. The lower RV mass to volume ratio is a hallmark of its physiologic function. On cross sectional view, one can appreciate the crescent shape of the right ventricle, as opposed to the circular shape of the thick-walled LV. As is apparent on three dimensional imaging, the RV is partially wrapped around the left ventricle, which is of importance in systolic ventricular interaction (figure 1).[14] RV anatomy is typically divided in the trabeculated apical component and a separate in- and outflow (infundibulum) region.[15] This results in the typical peristaltic RV ejection pattern.[16] Because of less prominent circumferential fibers, the RV ejection relies more on longitudinal shortening than in the LV.[17] Under normal conditions, the interventricular septum is concave towards the LV during the entire cardiac cycle.

Arterial perfusion of the right ventricular free wall is mainly provided by the right coronary artery. Blood flow to the apex and the interventricular septum is mainly provided by the left anterior descending artery (anterior two thirds), while the posterior third of the interventricular septum is perfused by the right coronary artery. The right ventricular veins drain into the anterior cardiac veins that empty individually into the right atrium just above the tricuspid valve.[18]

Physiology

Pressure-volume loops provide a suitable framework to discuss the determinants of RV function. If instantaneous pressures and volumes are plotted throughout the cardiac cycle, the characteristic triangular shape of the RV pressure-volume loop can be observed.[19] This framework allows different haemodynamic variables to be identified (figure 2). The RV is characterised by a high diastolic compliance. Its unique thin-walled architecture allows for a great variation in accommodation to venous return, without large changes in end-diastolic pressures.[20] The RV end-diastolic pressure volume curve is thus less steep than its left sided counterpart (figure 2). The drawback of the highly compliant RV free wall is that it results in a high afterload dependence. Even small elevations in pulmonary artery pressures lead to a marked reduction in RV stroke work.[20] As the heart is tightly coupled to the lungs, and mechanical ventilation has profound effects on intrathoracic pressures, heart-lung interactions are an important phenomenon. These are described in more detail below (see perioperative management). In contrast to the LV, right coronary flow is maintained throughout systole and diastole, as a result of the lower intraventricular pressures. Thus, the effects of the drop in diastolic perfusion time due to tachycardia are less important for the RV than for in the LV.[18]

The importance of ventricular interaction is well established. Up to 40 percent of systolic RV function may be attributable to LV systolic function through this mechanism.[21] Ventricular interdependence is defined as any interaction between the LV and the RV, with the exclusion of neuronal and humoral effects. It can be divided in a *direct* and an *indirect* component. The *direct* ventricular interdependence is mostly mediated through the interventricular septum and pericardium. This can be further divided into a direct diastolic and a direct systolic interaction. The *indirect* component is the result of the normal closed loop circulation, where the RV output equals the LV input.[22] In the volume overloaded RV, septal flattening occurs only in diastole.[23,24] In contrast, in the pressure overloaded RV the septal flattening is maintained from diastole into systole. Thus, in this situation, RV systolic function cannot be aided as much by the septum, and RV dilatation will initiate a negative spiral with a drop in RV ejection, a compressed LV and deteriorating LV function.[25-28] Originally, Slinker et al found in an open chest dog model that parallel diastolic and systolic interaction is reduced after pericardiotomy.[29] However, more recent studies conclude that the precise effect of the pericardium on ventricular interaction is still controversial.[30-31]

Causes of RV failure

It is useful to classify clinical RV failure based on the underlying pathophysiological mechanisms. Due to its anatomic and physiologic properties, the single most common cause of RV systolic failure is afterload augmentation. The 2008 updated World Health Organisation clinical classification of pulmonary hypertension defines the different aetiologies.[32] Modest pressure overload will at first mostly lead to increased RV contractility. From animal models it appears that this is first accomplished by the Anrep phenomenon (homeometric autoregulation) by which an adequate ventriculo-arterial coupling can be maintained.[33] As pulmonary afterload rises, an additional catecholamine release allows for an increased inotropic state.[34] Finally, a rise in end- diastolic volume is observed, and the Frank-Starling mechanism is addressed.[33] However, after prolonged and sustained RV pressure overload - even in the absence of ischaemia - RV contractility becomes downregulated.[35-37] Once the RV decompensates, systemic pressures and cardiac output suddenly drop. RV dilation results in a leftward septal diastolic shift, with decreased LV compliance and eventually systemic hypotension. This starts a downward spiral, with a further reduction in biventricular function. Because in RV pressure overload septal reversal is maintained from diastole into systole, the RV can make less use of the normal systolic ventricular interaction, which under normal circumstances accounts for 20 to 40% of RV systolic function.[21]

A second cause of RV dysfunction and failure is RV volume overload. Typical examples are tricuspid or pulmonary valve regurgitation and atrial or ventricular septal defects.[4] Due to its anatomical features to act as a volume pump, the RV can more easily accommodate to volume then to pressure overload. In contrast to the situation of pressure overload, chronic volume overloaded right hearts primarily use the Frank-Starling mechanism.[11] Of note, in RV isolated volume overload the RV can still make use of ventricular interaction. Although the septum will be shifted towards the left in diastole, in systole the septum returns to its normal position, thereby adding substantially to RV systolic function.[23,24] Inflow limitation by e.g. tricuspid or caval vein stenosis, is also a potential aetiology of right heart failure.

RV ischaemia can lead to failure directly, as in RV myocardial infarction, or indirectly as a result of systemic hypotension. Under normal circumstances, the RV is less prone to ischaemia than the LV, mostly because of the thinner myocardium, the more continuous perfusion throughout diastole and systole, and the lower resting oxygen extraction of the RV.[18] Under resting conditions the RV has an oxygen extraction ratio of only 50%.[38] An exercise induced increased oxygen demand will in the RV first be provided by increasing oxygen extraction ratio and only secondary by an increased right coronary blood flow.[38] However, the extent to which ischaemia is important in RV dysfunction and failure is controversial. Canine experiments, where the RV and the LV were mechanically uncoupled and right coronary perfusion pressure or flow was manipulated, suggest that aortic cross clamping improves RV function not through its effect on coronary perfusion pressure or flow but through ventricular interaction.[39-41] The relative importance of coronary perfusion pressure versus other mechanisms contributing to the development of RV failure remains to be fully elucidated. Numerous intrinsic causes, such as sepsis or dysplasia after longstanding RV arrhythmias, and constrictive pericarditis can also lead to RV dysfunction and/or failure. The many complex congenital causes are beyond the scope of this review.

Typical causes of perioperative RV failure include all mechanical as well as metabolic factors associated with increased RV afterload, such as volaemia disorders, pulmonary emboli, hypoxia, sepsis, (inappropriate) mechanical ventilation and acute RV ischaemia. Acute-on-chronic deterioration of RV function can be common in the perioperative setting. An overview of the pathophysiologic vicious circle of RV dysfunction is provided in figure 3. Patients with LV assist devices are particularly prone for developing RV failure: initiation of univentricular mechanical assist to support the failing LV often unmasks a latent RV dysfunction that was hidden in the clinical picture of LV dysfunction. Furthermore, LV assist devices operate by off-loading the LV during systole and diastole, hence eliminating ventriculovascular interaction while exposing the RV to full venous return and by challenging it to resume its function at the level of normal or supranormal cardiac output.

Perioperative anaesthetic considerations

Preoperative assessment

The symptoms and signs of right ventricular failure have been extensively discussed elsewhere.[33,42] Apart from typical clinical symptoms and signs such as dyspnea, hypotension, right upper quadrant discomfort and jugular vein distension, several electrocardiographic and radiographic clues should trigger further investigations.[42] Electrocardiographic specificities may include sinus tachycardia, T-wave inversions in III and aVF or in the precordial leads V1 to V4, right bundle branch blocks and rightward axis. Right sided precordial leads can help in diagnosing RV pathology.[42] Chest radiography in patients with RV failure can reveal dilation of the proximal pulmonary arteries and RV enlargement (with filling of the retrosternal space) or right atrial enlargement. Dilation of the inferior caval vein can be noticed, and pleural effusions are possible.[42] Ideally, all technical investigations should be performed sufficiently in advance preoperatively, in order to allow sufficient time for potential therapeutic optimization. Troponin levels are of importance in the diagnosis of pulmonary emboli and suspected RV myocardial ischaemia, although the low mass of the RV may cause only a slight elevation in troponin levels.[43] B-type natriuretic peptide, which is secreted by the myocardium in the case of increased shear stress and dilatation, can be used to differentiate cardiac and pulmonary acute dyspnea. Also, in many settings of pulmonary hypertension increasing levels of B-type natriuretic peptide correlate with the degree of RV dysfunction.[44,45] To date, the usefulness of B-type natriuretic peptide in the acute setting of RV failure remains unclear.[46]

Because echocardiography is non-invasive and available at the bedside, it has become the most important tool in the evaluation of RV dysfunction and its associated conditions. Care has to be taken to minimise its intrinsic and operator dependent limitations. Important findings include chamber dilatation, increased wall thickness (in chronic pulmonary hypertension) and wall motion abnormalities.[42] RV dilatation can be defined as a ratio of RV end-diastolic area to LV end-diastolic area of >0.6.[47] The McConnell sign, defined as RV free wall hypokinesis with apical sparing, is seen in acute pulmonary emboli.[48] Normal RV ejection fraction is between 35-45%. This lower value compared to the LV is the result of the larger end-diastolic volumes with comparable stroke volumes.[49] As RV contraction is predominantly longitudinal in nature, tricuspid annular plane systolic excursion is a well defined measure of RV function. The total displacement from apex to tricuspid annulus during systole is measured.[50] Doppler echocardiography allows assessing the severity of pulmonary hypertension. Pulmonary artery pressures can be estimated by adding a surrogate of right atrial pressures (e.g. collapsibility of the inferior caval vein) to the calculated peak pressure

gradient. For an extensive review of right heart echocardiography, we refer the reader to an excellent review.[51]

Cardiac magnetic resonance imaging may yield more reproducible data than echocardiography, but its use is currently still limited. Similar morphological findings as in echocardiography can be evaluated, and RV mass and volume measurements can be made. With the use of gadolinium, cardiac magnetic resonance imaging can detect ischaemia, and delayed images can differentiate this from infarction, even in patients with enzyme negative unstable angina.[52]

Right heart catheterisation with end-expiratory measurements of pulmonary artery, right and left sided pressures remains the gold standard for the diagnosis of pulmonary hypertension. These provide prognostic markers of survival.[53] The latest Dana Point 4th World Symposium on Pulmonary Hypertension (2008) defined pulmonary hypertension as a resting mean pulmonary artery pressure of ≥25mmHg. It needs to be kept in mind that, in the case of severe RV heart failure, decreased output can lead to decreasing pulmonary artery pressures.[4,54] We believe that preoperative invasive pulmonary artery catheterisation is indicated especially in cases where moderate to severe right ventricular dysfunction is combined with severe pulmonary hypertension.[62] To further specify diagnosis and reversibility, vasoreactivity testing can be useful in subgroups of pulmonary hypertension patients. Here, the haemodynamic effects of a short-acting vasodilator (e.g. epoprostenol or inhaled nitric oxide) are examined.[55]

Preoperative consultation and premedication

Preoperative consultation should include a thorough anamnestic and clinical search for right ventricular dysfunction. Individualised anxiolysis to avoid tachycardia and increased pulmonary resistances may be provided with benzodiazepines - great care should be taken to avoid respiratory depression. Preoperative oral sildenafil, calcium channel blockers, inhaled nitric oxide and nebulised iloprost can be used to reduce pulmonary artery pressures. It is important that these chronic therapies are not interrupted in the perioperative period. Maintenance of sinus rhythm (e.g. electrical cardioversion of new onset atrial fibrillation) and optimal ventricular rate should be targeted to prevent RV dilatation and optimizing ejection. It is often accomplished at a minimum of 90 beats per minute.[56] When using pharmaceuticals to maintain sinus rhythm, associated negative inotropic and systemic vasodilatory side effects should be kept in mind.

In the setting of RV failure due to left heart disease, optimisation of left heart function and corrective valve surgery can result in a regression of the associated pulmonary

hypertension.[57] Percutaneous coronary interventions can be indicated in the case of acute myocardial infarction. In pulmonary hypertension due to trombo-embolic events, trombolysis, trombectomy or pulmonary endarterectomy can be considered. Patients with RV failure due to severe tricuspid regurgitation may need tricuspid valve surgery.[58] More aggressive surgical end-stage management may include atrial septostomy, in which the atrial shunt decompresses the right side, at the cost of decreased oxygenation. Due to the rise in cardiac index, however, oxygen delivery appears to improve.[59] Total RV exclusion procedures have been used in the setting of RV volume overload, and assist devices or cardiac transplantation can be indicated in selected patients.[60,61]

Monitoring requirements

Although an individualised approach is of paramount importance, some recommend a cascade of invasiveness of perioperative monitoring based on the severity of RV dysfunction and pulmonary hypertension, intraoperative mode of ventilation and type of procedure.[62] Central venous line insertion is useful in the case of positive pressure ventilation and invasive blood pressure monitoring in severe cases of RV failure. Some authors suggest the use of a pulmonary artery catheter should be reserved for patients with severe RV failure with pulmonary hypertension, but it must be remembered that pressure measurements alone provide less information than pulmonary vascular resistance measurements. In our opinion, transesophageal echocardiography is mandatory in any case of RV failure and should only be omitted when an absolute contraindication is present.

Perioperative management

Anesthetic management in RV failure patients can be challenging. At all times, haemodynamic management should focus on maintaining the ratio of systemic to pulmonary arterial pressures at the pre-induction level, as a decrease in this ratio often predicts an imminent collapse.[63] Of note, a higher sympathetic tone is present in the setting of pulmonary artery hypertension.[64] Theoretically, etomidate or ketamine may be superior induction agents to avoid post induction hypotension while providing sufficiently deep anesthesia to prevent large sympathetic responses.[62] Although no studies are available, propofol and inhalation induction have been used as well. All volatile anaesthetics can reduce preload and contractility. Use of desflurane and nitrous oxide is discouraged, as it augments pulmonary vascular resistance.[65,66] No severe adverse effects of opiates on RV function have been described.[62] Lumbar neuraxial anesthesia can be used if preload decrease and subsequent hypotension are anticipated for.[62] However, thoracic epidural anesthesia has been shown to inhibit the reflectory inotropic raise to increased RV afterload in animals – the clinical impact being currently unclear.[67]

- **Homeostasis**

The management of RV failure should focus on restoring RV function with the underlying aetiology kept in mind. At all times, disruption of metabolic homeostasis (hypoxia and hypercarbia) and haemodynamic stressors (in particular in children with reactive pulmonary hypertension) should be avoided. Optimisation of RV preload, afterload and contractility, as well as of ventricular interdependence has to be achieved. It has been suggested that in primary RV failure the first step is to lower RV afterload, whereas in RV failure secondary to LV dysfunction, treatment should focus on the LV.[68] Chronic pulmonary hypertension leads to a RV with a much higher contractile reserve, but this may also result in reduced coronary flow reserve and impaired diastolic function.[68] As we review in the following paragraphs, multiple approaches are feasible – however, the impact of such supportive measures on long term outcome has not yet been clearly established.

- **Preload**

Judicious volume management should optimise RV preload. Although the RV is preload dependent up to a certain point, overfilling can cause RV dilatation and tricuspid valve insufficiency, as will be the case in most RV failure patients.[69] Resulting increased RV wall stress and drop in LV compliance can result in diminished cardiac output and in further RV dilatation. Care must be taken to estimate the position on the Starling curve of the particular patient, which is dependent on volume status as well as on cardiac function and RV afterload. Fluid withdrawal can be accomplished with diuretics or ultrafiltration. Empirically, a frequently used cut-off point is a transmural right atrial pressure of 15 mmHg. At all times, it needs to be kept in mind that any pressure based indicator of a volume measurement is subject to inherent limitations. We therefore prefer volume based (e.g. echocardiographic) variables to assess RV preload and fluid responsiveness. Although generally a useful indicator of fluid responsiveness,[70,71] pulse pressure variation cannot be used reliably in the setting of RV failure, and the lack of a decrease of a high pulse pressure variations after fluid administration has been suggested as a diagnostic tool for RV failure.[72-74] Echocardiographically guided volume challenges may provide an adequate therapeutic approach, e.g. by measuring caval vein diameters.

- **Afterload**

Active attempts to decrease RV afterload must be undertaken. Preferential use of spontaneous breathing or low ventilating pressures, use of a high inspiratory oxygen fraction and mild hyperventilation can lower RV afterload. Recruitment maneuvers and appropriate positive end expiratory pressures should minimise atelectasis formation.[62] The use of

negative pressure ventilation could be of interest. Intrathoracic pressure regulators, devices that allow application of a negative intrathoracic pressure in between positive pressure tidal volumes, have been shown to increase pulmonary artery pressures and cardiac output in animal hypovolaemic and shock models and in normovolaemic anaesthetised patients.[75-77] Their long term effects on RV afterload (by potentially creating atelectasis), have yet to be established. Intravenous vasodilators should only be used with extreme caution, because the resulting systemic hypotension may counter any advantage on pulmonary vasculature.[68] Inhaled pulmonary vasodilators such as inhaled nitric oxide or nebulised iloprost are preferably used. Because of its local action and its short half-life, inhaled nitric oxide causes no systemic hypotension. It only causes vasodilation in ventilated areas, and thus improves ventilation/perfusion mismatch. In acute respiratory distress syndrome patients, it proved to decrease pulmonary vascular resistance and increase RV ejection fraction.[78] However, despite its haemodynamic benefits, no reduced mortality was found in acute RV failure patients.[79] Rebound pulmonary hypertension following sudden withdrawal has been described.[80] Prostacycline (or prostaglandin I2) causes pulmonary vasodilation and improves right ventriculo-arterial coupling in afterload-induced right ventricular failure.[81] It may worsen ventilation-perfusion mismatch, and can worsen pulmonary capillary wedge pressure in the setting of LV dysfunction. With inhaled use, no rebound pulmonary hypertension has been reported. It has no known toxic metabolites. Inhaled iloprost is an analogue with a significantly longer duration of action, which has similar shown haemodynamic benefits.[82] Inhaled milrinone has also been shown to be effective.[83] Acute sildenafil treatment has been shown on magnetic resonance studies to promote RV relaxation.[84] It decreases RV afterload without significantly affecting systemic haemodynamics and decreases RV hypertrophy.[85-86] First line oral endothelin antagonist treatment showed improved survival in a study in primary pulmonary hypertension.[87] Because of their different modes of action (inhaled nitric oxide activates soluble guanylate cyclase, prostacyclin activates adenylate cyclase, phospodiesterase inhibitors work directly on the enzyme type 3 or 5 subfamilies, endothelin antagonist inhibits the vasoconstrictor) combinations are suggested to be synergetic.[62,68] We suggest that after a timely preoperative consultation, prompt referral of patients with chronic pulmonary hypertension to an experienced cardiology department should allow for optimalisation of chronic therapy.

- **Vasopression**

Vasopressors raise systemic arterial pressure, and thus coronary perfusion pressures. The importance of the absolute value of right coronary perfusion pressure in the maintenance of RV function has been debated. Experimental studies uncoupling systemic pressure from the right coronary perfusion pressure suggest that the benefit from a raised systemic pressure

may rather result from increased ventricular systolic interdependence.[39,41] At all times, the benefit of systemic vasoconstriction (with increased perfusion pressures) has to be balanced against the impact of pulmonary vasoconstriction.[88] When acute RV pressure overload is caused by mechanical factors, it was suggested that vasopressors may be beneficial, as systemic tension rises with little additional augmentation of RV afterload.[68] With isolated phenylephrine use in patients with chronic pulmonary hypertension, the net effect was a drop in cardiac output because of a raised pulmonary arterial elastance.[89] Low dose vasopressin was shown to be effective in reversing hypotension in chronic pulmonary hypertension.[90]

- **Inotropics**

In contrast to the left ventricle, the right ventricle (that has not been exposed to chronic pressure overload) has limited contractile reserve. Systemic hypotension is a risk, and thus care has to be taken with inodilators in isolated, primary right ventricular failure. With all inotrope use, care should be taken to avoid arrhythmia. Norepinephrine may be indicated to increase systemic tension in the setting of low cardiac output.[91] Epinephrine and high dose dopamine have also been used to increase RV contractility, but there is no evidence for superiority and side effects are imminent. Intravenous milrinone reduces mean pulmonary artery pressures and improves right heart performance after cardiac surgery.[92] As such, phosphodiesterase III inhibitors can be beneficial especially in the setting where left ventricular backward failure is the major cause. If systemic hypotension develops, however, a vasopressor must be associated. Increased LV contractility can also result in an increased RV systolic function through ventricular interdependence. In a randomised trial in patients with advanced LV systolic dysfunction and associated RV failure, echocardiographic measures of RV function were shown to benefit from levosimedan as compared to placebo.[94] It also reduced pulmonary vascular resistance in animals and patients with decompensated heart failure and improved RV-pulmonary artery coupling more then dobutamine in an experimental acute RV failure setting.[95-98] Clinically, these effects have been shown in multiple settings, such as in RV ischaemia, acute respiratory distress syndrome and after mitral valve replacement.[99,100]

Conclusions

This review provides an overview of the pathophysiology and clinical management of right ventricular function and failure in the perioperative setting. We underscore the specific functionality of the right ventricle. Accordingly, the need for distinct measures of assessment as opposed to the left ventricle is emphasised. Several available therapeutic options for the management of right ventricular failure are discussed. Additional basic physiologic as well as

clinical therapeutic research seems mandatory to get a better insight in the pathophysiology of perioperative RV failure this in order to provide better care to our patients.

Conflicts of interest and sources of funding

This work was supported by the Department of Anaesthesiology, Ghent University Hospital, Ghent, Belgium.

No conflicts of interest are declared.

References

1 Starr I, Jeffers W and Meade R. The absence of conspicuous increments of venous pressure after severe damage to the right ventricle of the dog, with a discussion of the relation between clinical congestive failure and heart disease. *Am Heart J* 1943; **26**:291.

2 Kagan A. Dynamic Responses of the Right Ventricle Following Extensive Damage by Cauterization. *Circulation* 1952; **5**:816–823.

3 Brooks H, Kirk ES, Vokonas PS et al. Performance of the right ventricle under stress: relation to right coronary flow. *J Clin Invest 1971*; **50**:2176–2183.

4 Haddad F, Doyle R, Murphy DJ and Hunt SA. Right Ventricular Function in Cardiovascular Disease, Part II: Pathophysiology, Clinical Importance, and Management of Right Ventricular Failure. *Circulation* 2008; **117**:1717–1731.

5 Gavazzi A, Berzuini C, Campana C et al. Value of right ventricular ejection fraction in predicting short-term prognosis of patients with severe chronic heart failure. *J Heart Lung Transplant* 1997; **16**:774-785.

6 Monchi M, Bellenfant F, Cariou A et al. Early predictive factors of survival in the acute respiratory distress syndrome. A multivariate analysis. *Am J Respir Crit Care Med* 1998; **158**:1076-1081.

7 Stoltzfus D. Right ventricular function and failure in the perioperative period. *Crit Care Med Trans* 1997; **15**:797-822.

8 Galiè N, Hoeper MM, Humbert M et al. Guidelines for the diagnosis and treatment of pulmonary hypertension. *Eur Heart J* 2009; **30**:2493.

9 Reichert CL, Visser CA, van den Brink RB et al. Prognostic value of biventricular function in hypotensive patients after cardiac surgery as assessed by transesophageal echocardiography. *J Cardiothorac Vasc Anesth* 1992; **6**:429-432.

10 Maslow AD, Regan MM, Panzica P, et al. Precardiopulmonary bypass right ventricular function is associated with poor outcome after coronary artery bypass grafting in patients with severe left ventricular systolic dysfunction. *Anesth Analg* 2002; **95**:1507-1518.

11 Szabó G, Soós P, Bährle S, et al. Adaptation of the Right Ventricle to an Increased Afterload in the Chronically Volume Overloaded Heart. *Ann Thorac Surg* 2006; **82**:989-995.

12 Humbert M, Sitbon O, Chaouat A, et al. Pulmonary arterial hypertension in France: results from a national registry. *Am J Respir Crit Care Med* 2006; **173**:1023-1030.

13 Voelkel NF, Quaife RA, Leinwand LA, et al. Right Ventricular Function and Failure: Report of a National Heart, Lung, and Blood Institute Working Group on Cellular and Molecular Mechanisms of Right Heart Failure. *Circulation* 2006; **114**: 1883-1891.

14 Sheehan F, Redington A. The right ventricle: anatomy, physiology and clinical imaging. *Heart* 2008; **94**:1510-1515.

15 Goor DA, Lillehei CW. Congenital malformations of the heart. In: *Congenital malformations of the heart: embryology, anatomy, and operative considerations.* 1st ed. New York, NY: Grune & Stratton; 1975,1-37.

16 Haddad F, Hunt SA, Rosenthal DN, Murphy DJ. Right Ventricular Function in Cardiovascular Disease, Part I: Anatomy, Physiology, Aging, and Functional Assessment of the Right Ventricle. *Circulation* 2008; **117**:1436–1448.

17 Leather HA, AMA R, Missant C, Rex C, Rademakers FE, Wouters PF. Longitudinal but not circumferential deformation reflects global contractile function in the right ventricle with open pericardium. *Am J Physiol Heart Circ Physiol* 2006; **290**:H2369-2375.

18 Barash P, Cullen B, Stoelting R, Cahalan M, Stock M, eds. In: *Clinical Anesthesia, 6th edition.* Lippincott Williams and Wilkins 2009; 222.

19 Maughan WL, Shoukas AA, Sagawa K, Weisfeldt ML. Instantaneous pressure-volume relationship of the canine right ventricle. *Circ Res* 1979; **44**:309-315.

20 Chan CM, Klinger JR. The right ventricle in sepsis. *Clin Chest Med* 2008; **29**:661-676, ix.

21 Yamaguchi S, Harasawa H, Li KS, Zhu D, Santamore WP. Comparative significance in systolic ventricular interaction. *Cardiovasc Res* 1991; **25**:774-83.

22 Santamore WP, Dell'Italia LJ. Ventricular interdependence: significant left ventricular contributions to right ventricular systolic function. *Prog Cardiovasc Dis* 1998; **40**:289-308.

23 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**:179-186.

24 Santamore WP, Meier GD, Bove AA. Effects of Hemodynamic Alterations on Wall Motion in the Canine Right Ventricle. *Am J Physiol Heart Circ Physiol* 1979; **236**:H254–H262.

25 AMA R, Leather HA, Segers P, Vandermeersch E, Wouters PF. Acute pulmonary hypertension causes depression of left ventricular contractility and relaxation. *Eur J Anaesthesiol* 2006; **23**:824-831.

26 Brinker JA, Weiss JL, Lappe DL et al. Leftward septal displacement during right ventricular loading in man. *Circulation* 1980; **61**:626-633.

27 Jardin F, Dubourg O, Guéret P, Delorme G, Bourdarias J-P. Quantitative two-dimensional echocardiography in massive pulmonary embolism: Emphasis on ventricular interdependence and leftward septal displacement. *J Am Coll Cardiol* 1987; **10**:1201-1206.

28 Louie EK, Lin SS, Reynertson SI, Brundage BH, Levitsky S, Rich S. Pressure and Volume Loading of the Right Ventricle Have Opposite Effects on Left Ventricular Ejection Fraction. *Circulation* 1995; **92**:819-824.

29 Slinker BK, Glantz SA. End-Systolic and End-Diastolic Ventricular Interaction. *Am J Physiol Heart Circ Physiol* 1986; **251**:H1062-H1075.

30 Schertz C, Pinsky MR. Effect of the pericardium on systolic ventricular interdependence in the dog. *J Crit Care* 1993; **8**:17–23.

31 Farrar DJ, Chow E, Brown CD. Isolated Systolic and Diastolic Ventricular Interactions in Pacing-Induced Dilated Cardiomyopathy and Effects of Volume Loading and Pericardium. *Circulation* 1995; **92**:1284-1290.

32 Simonneau G, Robbins IM, Beghetti M et al. Updated Clinical Classification of Pulmonary Hypertension. *J Am Coll Cardiol* 2009; **54**:S43–S54.

33 Greyson CR. Pathophysiology of right ventricular failure. *Crit Care Med* 2008; **36**:S57–S65.

34 Nootens M, Kaufmann E, Rector T et al. Neurohormonal activation in patients with right ventricular failure from pulmonary hypertension: Relation to hemodynamic variables and endothelin levels. *J Am Coll Cardiol* 1995; **26**:1581-1585.

35 Kerbaul F, Rondelet B, Motte S et al. Effects of norepinephrine and dobutamine on pressure load-induced right ventricular failure. *Crit Care Med* 2004; **32**:1035-40.

36 Greyson C, Xu Y, Cohen J, G. Schwartz G. Right Ventricular Dysfunction Persists Following Brief Right Ventricular Pressure Overload. *Cardiovasc Res* 1997; **34**:281–8.

37 Greyson C, Xu Y, Lu L, Schwartz GG. Right Ventricular Pressure and Dilation During Pressure Overload Determine Dysfunction After Pressure Overload. *Am J Physiol Heart Circ Physiol* 2000; **278**:H1414-H1420.

38 Zong P, Tune JD, Downey HF. Mechanisms of Oxygen Demand/Supply Balance in the Right Ventricle. *Exp Biol Med* 2005;**230**:507-519.

39 Page RD, Harringer W, Hodakowski GT, et al. Determinants of maximal right ventricular function. *J Heart Lung Transplant* 1992; **11**:90-98.

40 Belenkie I, Horne SG, Dani R, Smith ER, Tyberg JV. Effects of Aortic Constriction During Experimental Acute Right Ventricular Pressure Loading: Further Insights Into Diastolic and Systolic Ventricular Interaction. *Circulation* 1995; **92**:546-554.

41 Klima UP, Lee M-Y, Guerrero JL et al. Determinants of maximal right ventricular function: Role of septal shift. *J Thorac Cardiovasc Surg* 2002; **123**:72-80.

42 Piazza G. The Acutely Decompensated Right Ventricle: Pathways for Diagnosis and Management. *Chest* 2005; **128**:1836-1852.

43 Konstantinides S, Geibel A, Olschewski M et al. Importance of Cardiac Troponins I and T in Risk Stratification of Patients With Acute Pulmonary Embolism. *Circulation* 2002; **106**:1263-1268.

44 Nagaya N, Nishikimi T, Okano Y et al. Plasma Brain Natriuretic Peptide Levels Increase in Proportion to the Extent of Right Ventricular Dysfunction in Pulmonary Hypertension. *J Am Coll Cardiol* 1998; **31**:202-208.

45 Nagaya N, Nishikimi T, Uematsu M et al. Plasma Brain Natriuretic Peptide as a Prognostic Indicator in Patients With Primary Pulmonary Hypertension. *Circulation* 2000; **102**:865-870.

46 Kaczyńska A, Kostrubiec M, Ciurzynski M and Pruszczyk P. B-type natriuretic peptide in acute pulmonary embolism. *Clin Chim Acta* 2008; **398**: 1-4.

47 Vieillard-Baron A, Prin S, Chergui K, Dubourg O, Jardin F. Echo–Doppler Demonstration of Acute Cor Pulmonale at the Bedside in the Medical Intensive Care Unit. Am. J. Respir. *Crit Care Med* 2002; **166**:1310-1319.

48 McConnell MV, Solomon SD, Rayan ME, Come PC, Goldhaber SZ, Lee RT. Regional right ventricular dysfunction detected by echocardiography in acute pulmonary embolism. *Am J Cardiol* 1996; **78**:469-473.

49 Hemnes AR, Forfia PR, Champion HC. Assessment of pulmonary vasculature and right heart by invasive haemodynamics and echocardiography. *Int J Clin Pract Suppl* 2009; **162**:4-19.

50 Kaul S, Tei C, Hopkins JM, Shah PM. Assessment of right ventricular function using two-dimensional echocardiography. *Am Heart J* 1984;**107**: 526-531.

51 Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, Solomon SD, Louie EK and Schiller NB. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr* 2010; **23**: 685-713.

52 Kwong RY, Schussheim AE, Rekhraj S et al. Detecting acute coronary syndrome in the emergency department with cardiac magnetic resonance imaging. *Circulation* 2003; **107**:531-537.

53 D'Alonzo GE, Barst RJ, Ayres SM et al. Survival in patients with primary pulmonary hypertension. Results from a national prospective registry. *Ann Intern Med* 1991; **115**:343-349.

54 Badesch DB, Champion HC, Sanchez MAG et al. Diagnosis and assessment of pulmonary arterial hypertension. *J Am Coll Cardiol* 2009; **54**:S55-66.

55 McLaughlin VV, Archer SL, Badesch DB et al. ACCF/AHA 2009 expert consensus document on pulmonary hypertension: a report of the American College of Cardiology Foundation Task Force on Expert Consensus Documents and the American Heart Association: developed in collaboration with the American College of Chest Physicians, American Thoracic Society, Inc., and the Pulmonary Hypertension Association. *Circulation* 2009; **119**:2250-2294.

56 Puhlman M. Continious-flow left ventricular assist device and the right ventricle. *AACN Adv Crit Care* 2012; **23**:86-90.

57 Haddad F, Kudelko K, Mercier O, Vrtovec B, Zamanian RT, de Jesus Perez V. Pulmonary hypertension associated with left heart disease: characteristics, emerging concepts, and treatment strategies. *Prog Cardiovasc Dis* 2011; **54**:154-167.

58 Aklog L, Williams CS, Byrne JG, Goldhaber SZ. Acute Pulmonary Embolectomy A Contemporary Approach. *Circulation* 2002; **105**:1416-1419.

59 McNeil K, Dunning J, Morrell N. The pulmonary physician in critical care • 13: The pulmonary circulation and right ventricular failure in the ITU. *Thorax* 2003; **58**:157-162.

60 Sano S, Ishino K, Kawada M et al. Total right ventricular exclusion procedure: An operation for isolated congestive right ventricular failure. *J Thorac Cardiovasc Surg* 2002; **123**:640-647.

61 Takagaki M, Ishino K, Kawada M et al. Total Right Ventricular Exclusion Improves Left Ventricular Function in Patients With End-Stage Congestive Right Ventricular Failure. *Circulation* 2003; **108**:II–226-229.

62 Forrest P. Anaesthesia and right ventricular failure. *Anaesth intensive care* 2009: **37**:370-385.

63 Hohn L, Schweizer A, Morel DR, Spiliopoulos A, Licker M. Circulatory failure after anesthesia induction in a patient with severe primary pulmonary hypertension. Anesthesiology 1999; **91**:1943-1945.

64 Velez-Roa S, Ciarka A, Najem B, Vachiery J-L, Naeije R and van de Borne P. Increased Sympathetic Nerve Activity in Pulmonary Artery Hypertension. Circulation. 2004; **110**:1308-1312.

65 Pagel P, Fu J, Damask M. Desflurane and isoflurane produce similar alteration in systemic and pulmonary hemodynamics in patients undergoing one lung ventilation during thoracotomy. *Anesth Analg* 1998; **87**:800-807.

66 Schulle-Sasse U, Hess W, Tarnow J. Pulmonary vascular responses to nitrous oxide in patients with normal and high pulmonary vascular resistance. *Anesthesiology* 1982; **57**:9-13.

67 Rex S, Missant C, Segers P, Wouters P. Thoracic epidural anesthesia impairs the hemodynamic response to acute pulmonary hypertension by deteriorating right ventricular-pulmonary arterial coupling. *Crit Care Med* 2007; **35**:222-9.

68 Wouters P, Rex S and Missant C. *Pharmacological support of the failing right ventricle*. In: Vincent J-L: 2008 Year book of intensive care and emergency medicine. 2008, 88-100.

69 Mebazaa A, Karpati P, Renaud E, Algotsson L. Acute right ventricular failure--from pathophysiology to new treatments. *Intensive Care Med* 2004; **30**:185-196.

70 Michard F. Changes in arterial pressure during mechanical ventilation. *Anesthesiology* 2005; **103**: 419-428.

71 Pinksy MR and Payen D. Functional hemodynamic monitoring. *Crit Care* 2005; **9**: 566-572.

72 Daudel F, Tüller D, Krähenbühl S et al. Pulse pressure variation and volume responsiveness during acutely increased pulmonary artery pressure: an experimental study. *Crit Care* 2010; **14**:122.

73 von Ballmoos MW, Takala J, Roeck M et al. Pulse-pressure variation and hemodynamic response in patients with elevated pulmonary artery pressure: a clinical study. *Crit Care* 2010; **14**:111.

74 Michard F, Richards G, Biais M, Lopes M, Auler JO. Using pulse pressure variation or stroke volume variation to diagnose right ventricular failure? *Crit Care* 2010; **14**:451.

75 Yannopoulos D, Metzger A, McKnite S et al. Intrathoracic pressure regulation improves vital organ perfusion pressures in normovolemic and hypovolemic pigs. *Resuscitation* 2006; **70**:445-453.

76 Yannopoulos D, McKnite S, Metzger A, Lurie KG. Intrathoracic Pressure Regulation Improves 24-Hour Survival in a Porcine Model of Hypovolemic Shock. *Anesth Analg* 2007; **104**:157-162.

77 Huffmyer JL, Groves DS, Scalzo DC et al. The Effect of the Intrathoracic Pressure Regulator on Hemodynamics and Cardiac Output. *Shock* 2011; **35**:114-116.

78 Fierobe L, Brunet F, Dhainaut JF et al. Effect of Inhaled Nitric Oxide on Right Ventricular Function in Adult Respiratory Distress Syndrome. *Am J Respir Crit Care Med* 1995; **151**:1414-1419.

79 Bhorade S, Christenson J, O'connor M, Lavoie A, Pohlman A, Hall JB. Response to Inhaled Nitric Oxide in Patients with Acute Right Heart Syndrome. *Am J Respir Crit Care Med* 1999; **159**:571-579.

80 Christenson J, Lavoie A, O'connor M, Bhorade S, Pohlman A, Hall JB. The Incidence and Pathogenesis of Cardiopulmonary Deterioration After Abrupt Withdrawal of Inhaled Nitric Oxide. *Am J Respir Crit Care Med* 2000; **161**:1443-1449.

81 Kerbaul F, Brimioulle S, Rondelet B, Dewachter C, Hubloue I, Naeije R. How Prostacyclin Improves Cardiac Output in Right Heart Failure in Conjunction with Pulmonary Hypertension. *Am J Respir Crit Care Med* 2007; **175**:846-850.

82 Rex S, Schaelte G, Metzelder S et al. Inhaled iloprost to control pulmonary artery hypertension in patients undergoing mitral valve surgery: a prospective, randomized-controlled trial. *Acta Anaesthesiol Scand* 2008; **52**:65-72.

83 Denault AY, Lamarche Y, Couture P et al. Inhaled Milrinone: A New Alternative in Cardiac Surgery? *Semin Cardiothorac Vasc Anesth* 2006; **10**:346-360.

84 Gan CT-J, Holverda S, Marcus JT et al. Right Ventricular Diastolic Dysfunction and the Acute Effects of Sildenafil in Pulmonary Hypertension Patients. *Chest* 2007; **132**:11-17.

85 Nagendran J, Stewart K, Hoskinson M, Archer SL. An anesthesiologist's guide to hypoxic pulmonary vasoconstriction: implications for managing single-lung anesthesia and atelectasis. *Curr Opin Anaesthesiol* 2006; **19**:34-43.

86 Nagendran J, Archer SL, Soliman D et al. Phosphodiesterase Type 5 Is Highly Expressed in the Hypertrophied Human Right Ventricle, and Acute Inhibition of Phosphodiesterase Type 5 Improves Contractility. *Circulation* 2007; **116**:238-248.

87 McLaughlin VV, Genthner DE, Panella MM, Rich S. Reduction in pulmonary vascular resistance with long-term epoprostenol (prostacyclin) therapy in primary pulmonary hypertension. *N Engl J Med* 1998; **338**:273-277.

88 Gold JA, Cullinane S, Chen J, Oz MC, Oliver JA, Landry DW. Vasopressin as an alternative to norepinephrine in the treatment of milrinone-induced hypotension. *Crit Care Med* 2000; **28**:249-252.

89 Rich S, Gubin S, Hart K. The effects of phenylephrine on right ventricular performance in patients with pulmonary hypertension. *Chest* 1990; **98**:1102-1106.

90 Price LC, Forrest P, Sodhi V et al. Use of vasopressin after caesarean section in idiopathic pulmonary arterial hypertension. *Br J Anaesth* 2007; **99**:552-555.

91 Layish DT and Tapson VF. Pharmacologic hemodynamic support in massive pulmonary embolism. *Chest* 1997; **111**:218.

92 Gillies M, Bellomo R, Doolan L and Buxton B. Bench-to-bedside review: Inotropic drug therapy after adult cardiac surgery – a systematic literature review. *Crit Care* 2005; **9**:266-279.

93 Stobierska-Dzierzek B, Awad H, Michler RE. The evolving management of acute right-sided heart failure in cardiac transplant recipients. *J Am Coll Cardiol* 2001; **38**:923-931.

94 Parissis J, Paraskevaidis I, Bistola V et al. Effects of Levosimendan on Right Ventricular Function in Patients With Advanced Heart Failure. *Am J Cardiol* 2006; **98**:1489-1492.

95 Missant C, Rex S, Segers P and Wouters PF. Levosimendan improves right ventriculovascular coupling in a porcine model of right ventricular dysfunction. *Crit Care Med* 2007; **35**:707-15.

96 Slawsky MT, Colucci WS, Gottlieb SS et al. Acute Hemodynamic and Clinical Effects of Levosimendan in Patients With Severe Heart Failure. *Circulation* 2000; **102**: 2222-2227.

97 Leather HA, Ver Eycken K, Segers P, Herijgers P, Vandermeersch E, Wouters PF. Effects of levosimendan on right ventricular function and ventriculovascular coupling in open chest pigs. *Crit Care Med* 2003; **31**:2339-2343.

98 Kerbaul F, Rondelet B, Demester J-P et al. Effects of levosimendan versus dobutamine on pressure load-induced right ventricular failure. *Crit Care Med* 2006; **34**:2814-2819.

99 Price LC, Wort SJ, Finney SJ, Marino PS, Brett SJ. Pulmonary vascular and right ventricular dysfunction in adult critical care: current and emerging options for management: a systematic literature review. *Crit Care* 2010; **14**:R169.

100 Morais RJ. Levosimendan in Severe Right Ventricular Failure Following Mitral Valve Replacement. *J Cardiothoracic Vasc Anesth* 2006; **20**:82-84.

Figures

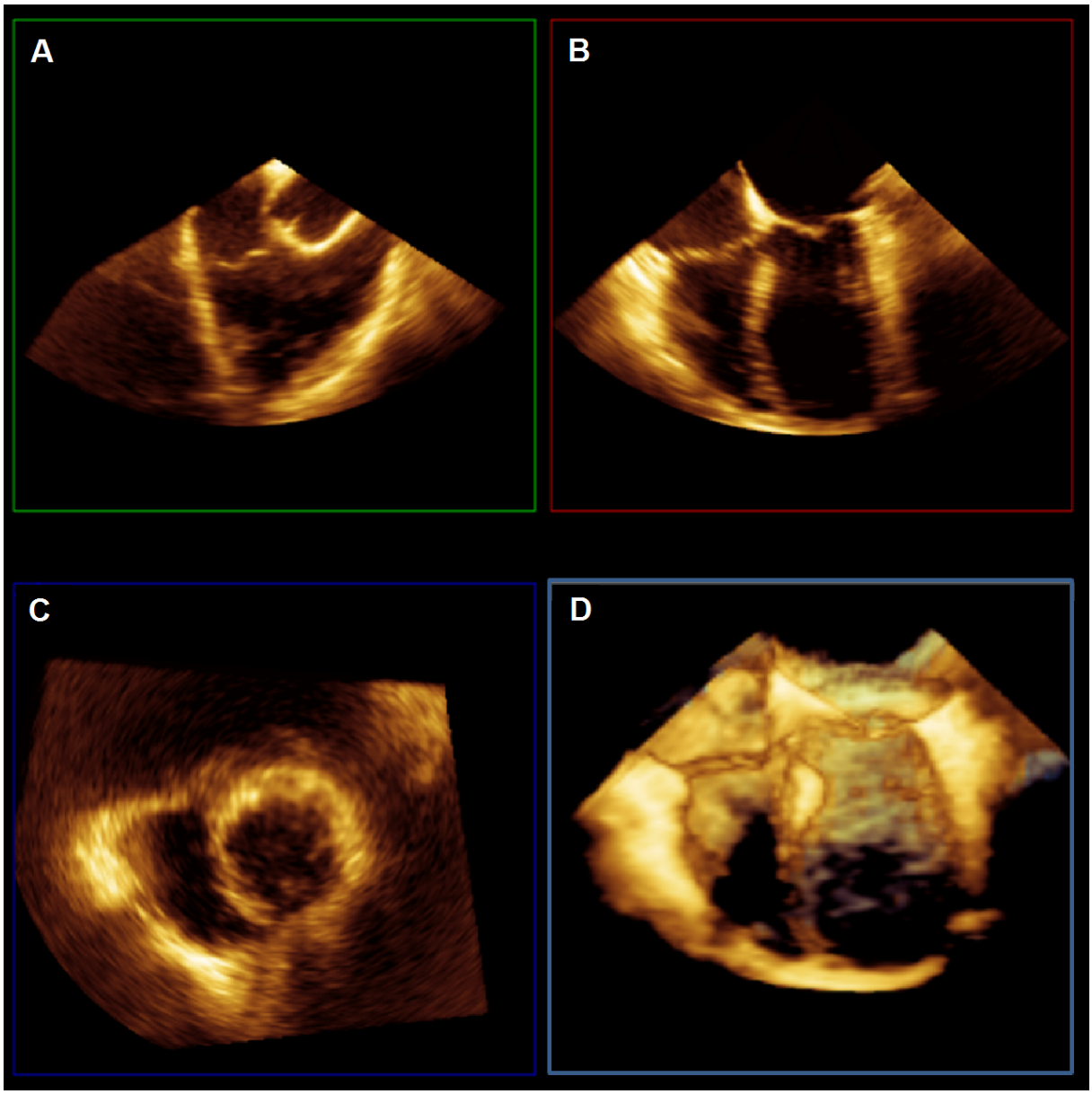

Figure 1 - Three dimensional imaging

Simultaneous display of three planes through a three dimensional full volume data set. Using built-in software, the image planes can be adjusted to obtain two dimensional RV views. The mid esophageal right ventricular inflow-outflow view (A) allows appreciation of the right ventricular free wall as well as of tricuspid and pulmonary valve function. (Longitudinal) contractility and septal position can be visualized in this mid esophageal four chamber view (B). On a transgastric mid papillary short axis view (C) the crescent shape of the right ventricle, as it is wrapped around the left ventricle, is apparent. Three dimensional full volume dataset cropped to provide mid esophageal four chamber view (D).

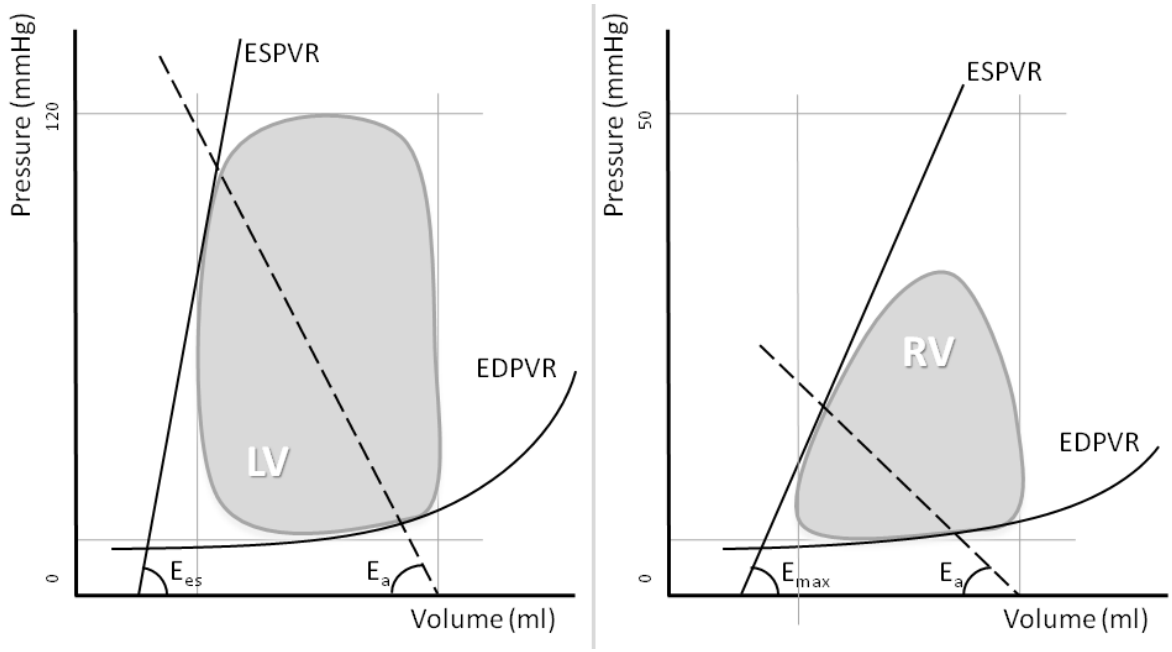

Figure 2 - Pressure-volume loops

Pressure-volume loops are constructed by plotting intraventricular pressures and volumes throughout the cardiac circle. The isovolumetric contraction comprises the onset of systole. Due to the relatively low pulmonary artery pressures, this phase is very short in the RV. This explains the triangular shape of the RV pressure-volume loops, as opposed to the more rectangular LV loops. The less steep shape of the right ventricular of the end diastolic pressure volume relationship (EDPVR) correlates with its higher compliance. The angle of the end systolic pressure volume relationship (ESPVR) with the volume axis is the maximal (E_{max} in the RV) or end systolic (E_{es} in the LV), an index of ventricular contractility. The arterial elastance (E_a) is a measure of ventricular afterload.

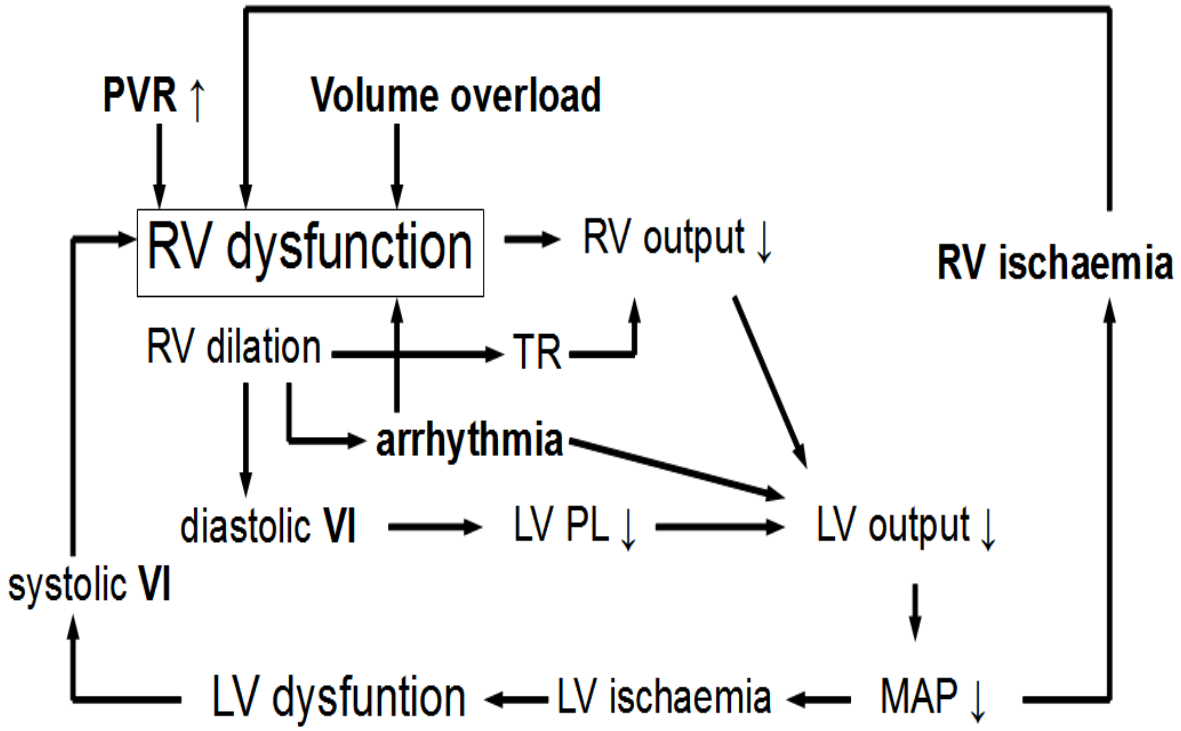

Figure 3 - Right ventricular dysfunction: a pathophysiologic vicious circle

The principal pathways towards right ventricular dysfunction are presented in bold: pressure overload, volume overload, ischaemia, arrhythmia and alterations in ventricular interaction. Diminished biventricular output gives rise to a vicious circle in which haemodynamics continue to worsen. (LV: left ventricle, RV: right ventricle, PL: preload, PVR: pulmonary vascular resistance, TR: tricuspid regurgitation, VI: ventricular interdependence)

Referentie van de publicatie

Eur J Anaesthesiol. 2013 Jul;30(7):386-94. doi: 10.1097/EJA.0b013e3283607a2d.

A pathophysiological approach towards right ventricular function and failure.

Vandenheuvel MA[1], Bouchez S, Wouters PF, De Hert SG.

Nederlandse samenvatting

De primaire functie van het rechter ventrikel (RV) is om de bloedstroom door de longen te ondersteunen. Om dit te bereiken is het ventrikel anatomisch en fysiologisch ontworpen als een debietpomp, die de centrale veneuze druk laag kan houden. Tot voor kort werd het belang van het rechter ventrikel vaak geminimaliseerd. In de afgelopen decennia hebben talrijke studies echter het belang van een normale RV functie onderstreept. Er werd aangetoond dat RV dysfunctie een belangrijke voorspeller is van overleving en van de morbiditeit in verschillende klinische situaties. Zo is rechter hartfalen is de belangrijkste doodsoorzaak bij pulmonale hypertensie. Perioperatieve sterfte is nog hoger in RV falen dan in linker hartfalen en het is belangrijk op te merken dat progressief RV falen een vergelijkbare incidentie heeft als linker hartfalen.

Het doel van dit werkstuk is om een pathofysiologische samenvatting te maken van de perioperatieve rechter ventrikel functie en dysfunctie. Zoals eerder vermeld werd het belang van de rechter ventrikel functie in de peri-operatieve periode reeds in verschillende domeinen vastgesteld. Toch is een groot deel van onze huidige kennis over de aanpak van rechter ventrikel falen slechts gebaseerd op een extrapolatie van de resultaten van linker hartkamer onderzoek , hoewel van de biventriculaire fysiologie bekend is dat er duidelijke verschillen zijn in vele aspecten. Er werd een grondige literatuurstudie uitgevoerd: we doorzochten de Pubmed, Embase en Web of Science databases met behulp van (combinaties van) de volgende (engelstalige) zoektermen: ' rechter ventrikel functie ' , ' rechter ventrikel falen ' , ' perioperatief ' , ' anatomie ' , ' fysiologie ' , ' epidemiologie ' , ' ventriculaire interactie ' , ' volume overbelasting ' , ' druk overbelasting ' , ' ischemie ' , ' echocardiografie ' , ' longslagadercatheter ' , ' polsdruk variatie ' , ' nabelasting reductie ' , ' mechanische ventilatie ' , ' inotrope ondersteuning 'en' vasopressine'.

We hebben zowel theoretische als praktische aspecten van perioperatief rechter ventrikel falen beoordeeld. Na het onderstrepen van het belang van dit onderwerp , bespreken we de rechter ventrikel anatomie en fysiologie, met de nadruk op de rol van de ventriculaire interactie. Vervolgens worden mogelijke oorzaken van perioperatief rechter ventrikel falen besproken en opgelijst. De klemtoon van het artikel ligt op de specifieke perioperatieve anesthetische overwegingen, gaande van de preoperatieve evaluatie en oppuntstelling tot de intra-operatieve monitoringstechnieken en specifieke hedendaagse therapeutische mogelijkheden van perioperatief rechter ventrikel falen. Hierbij worden, naast een veelvoud aan ventilatoire en andere ingrepen die een invloed kunnen hebben op de functie van het rechter ventrikel, intraveneuze en inhalatie pharmacologische ingrepen besproken. De

bedoeling is om de anesthesist te voorzien van een uitgebreid overzicht van perioperatieve rechter ventrikel functie en falen.