ISSN: 2474 - 9206

Research Article

Journal of Anesthesia & Pain Medicine

Diastolic Ventricular Insufficiency and Use of Anesthetics

Sofía Doria Castro [1], Nixon Martin Burbano Erazo [2], Luisa Fernanda Mesa Rodríguez [3], María Fernanda Landinez Cordero [4], Camilo Andrés Benavides Pai [5], Santiago Andrés Tapia Mora [6], Francisco Alberto Rodríguez Lee [7], Yudy Andrea Chicaiza Guerrero [8]

[1]*Fourth Year Anesthesiology Resident, Universidad San Carlos de Guatemala*

[2]*General Physician, Universidad Tecnológica de Pereira, Colombia*

[3]*General Physician, Fundación Universitaria de Ciencias de la Salud FUCS, Bogotá*

[4]*General Physician, Universidad Autónoma de Bucaramanga, Colombia*

[5]*General Physician, Universidad Cooperativa de Colombia, Headquarters Pasto*

[6]*General Physician, Universidad de Cuenca, Ecuador*

[7]*General Physician, Universidad Autónoma de Guadalajara, México*

[8]*General Physician, Universidad Cooperativa de Colombia*

***Corresponding author**
Sofía Doria Castro, Fourth Year Anesthesiology Resident, Universidad San Carlos de Guatemala, Colombia

Submitted: 14 Nov 2021; **Accepted**: 24 Nov 2021; **Published**: 18 Jan 2022

citation: *Sofía Doria Castro, Nixon Martin Burbano Erazo, Luisa Fernanda Mesa Rodríguez, Maria Fernanda Landinez Cordero, Camilo Andrés Benavides Pai, Santiago Andrés Tapia Mora, Francisco Alberto Rodríguez Lee, Yudy Andrea Chicaiza Guerrero (2022) Diastolic Ventricular Insufficiency and Use of Anesthetics. J Anesth Pain Med 7(1): 04-10.*

Abstract

Heart failure is a pathology that generates a progressive deterioration that little by little compromises the functional capacity of the individual who suffers from it, affecting the ability of the heart to pump blood to the body adequately. Regarding its prevalence in general, it can affect from 1% to 14% of the population in the United States and Europe. In Colombia, according to a study published in 2012, heart failure has a prevalence in the general population of 2.3%, with 67.5% being heart failure with preserved EF. This is classified according to the ejection fraction of the patients, which may be preserved or decreased. Diastolic ventricular dysfunction is of the type with normal ejection fraction. Its incidence varies in critically ill patients between 40 and 80%, the use of anesthetics in these patients has not been much studied, and however, some very important considerations can be taken into account when applying them.

Methodology

A narrative review was carried out, in which different databases were used such as Scielo, PubMed, Sciencedirect, academic google, among others. The selection of articles was made through indexed journals in English and Spanish languages from 2010 to 2021 that met the selection criteria.

Results

Diastolic dysfunction includes an alteration in diastolic ventricular compliance; that is, it affects cardiac filling, when the

ventricle is relaxed. It is a condition in which, to sustain normal cardiac output, the filling pressure is increased. Patients with diastolic ventricular failure. Most anesthetics reduce sympathetic tone, which decreases venous return due to an increase in the compliance of the venous system, arterial vasodilation, and a decrease in blood pressure, thus generating a greater alteration in diastole, and consequently in preload.

Conclusions

The use of anesthetics in diastolic insufficiency should be further studied due to the implications it has at the cardiovascular level when inducing a patient. Being these more sensitive to hemodynamic changes, and cavitary pressures that generates a high risk of imminent decompensation during the procedures to which these patients undergo regardless of whether they are cardiac or not.

Keywords: Cardiac, Anesthetics, Heart Failure, Diastolic Dysfunction, Systole, Preload, Afterload.

Introduction

Heart failure is a multifactorial disease, which represents a great challenge in public health. Heart failure is defined as the inability of the heart to pump blood and supply oxygen to the body according to its metabolic needs and requirements; which consists of a functional and structural alteration, which generates a modification in the cardiovascular physiology that is manifested clinically in the patient, triggering at the pathophysiological level a progressive wear of the myocytes, decreasing their contractile capacity and consequently the cardiac output, therefore that patients suffering from this disease manifest a systemic compromise that little by little leads to a deficit in functional capacity (figure 1) [1].

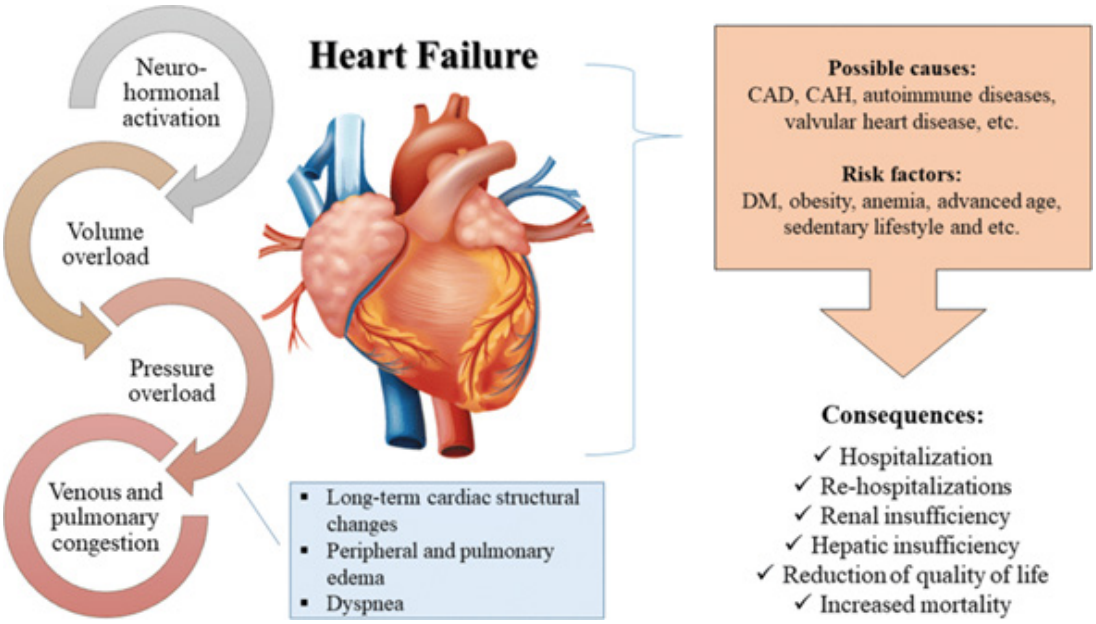

Figure 1: Heart failure

The heart is an organ made up of muscle tissue and a vascular system that connects the rest of the tissues and keeps the body oxygenated. Among the cardinal symptoms of the disease are dyspnea and fatigue, however, other manifestations are: edema, cough, rales, jugular engorgement, acute pulmonary edema, hepatomegaly, weight loss, among others. In the cardiac cycle, a series of physiological events occur that determine preload and afterload. These comprise an alternation between systole and diastole. Systole is that stage of ventricular contraction in which the heart ejects blood into the body, while diastole consists of the filling stage, in which the ventricle relaxes, allowing a sufficient load of blood that will consequently be distributed. Thus repeating the cycle, during each heartbeat. The number of heart beats generated in one minute corresponds to the heart rate and the heart rate times the volume ejected in systole corresponds to the cardiac output (figure 2) [1-2].

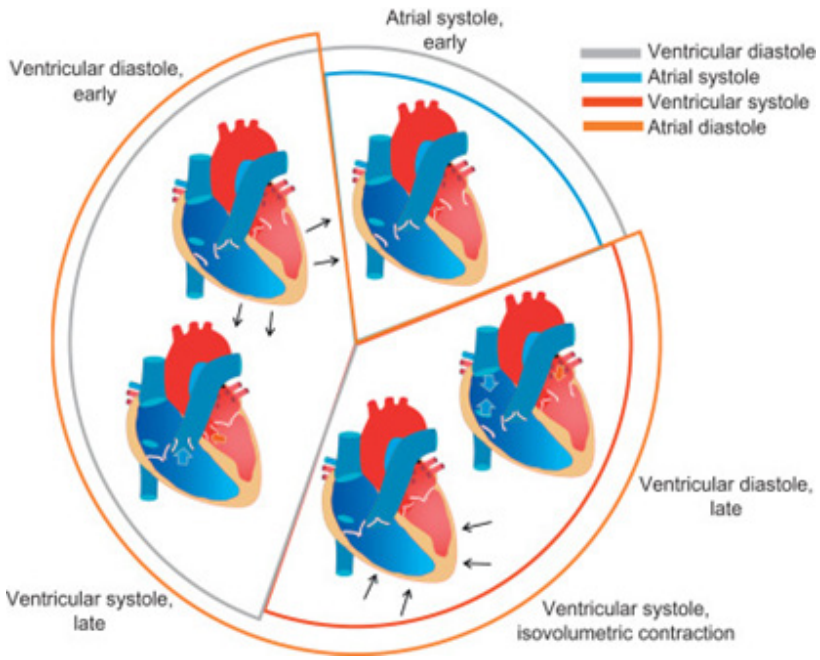

Figure 2: Cardiac cycle

Methodology

A narrative review was carried out, in which different databases were used such as Scielo, PubMed, Sciencedirect, academic google, among others. The selection of articles was made through indexed journals in English and Spanish languages from the years 2010 to 2021. As key words, the following terms were used according to the terms DeCs and MeSH: cardiac, anesthetics, heart failure, diastolic dysfunction, systole, preload, afterload. In this review, 26 original and review publications related to the subject under study were identified, of which 16 met the inclusion criteria used. The inclusion criteria included articles on cardiac physiology, heart failure and the use of anesthetics in diastolic dysfunction, which at the time of the search allowed the abstract to be read and which were within the established years. Exclusion criteria: That their publication date was less than 2010 and that it did not allow the summary to be viewed.

Results

Physiology

In order for the systolic function of the left ventricle to take place, and to achieve adequate ejection of blood into the aorta, a correct contraction phase must be present. The contractile capacity of the heart depends on the myocardial sarcomere, which is the smallest functional unit of the muscle fiber, which is shortened thanks to the formation of bridges between the actin and myosin filaments and the sliding of these filaments between them. Initially, the entry of the calcium ion into the cell is generated by the calcium L channels during cell depolarization, which activates the release from the sarcoplasmic reticulum to the cytosol of a large amount of calcium, which when binding to troponin in the Thin myofilament activates the formation of bridges and their replacement. Preload is an estimate of ventricular volume at the end of diastole, and depends on the stretching of the heart muscle prior to contraction. That is, as the ventricle dilates at the same time, it distends the sarcomere, which generates a higher rate of turnover, with increased expulsive volume and cardiac output. The more distension there is in the filling, the greater the contractile force, which is known as Starling's law. Preload can be affected by venous return, diastolic dysfunction, or cardiac constriction. Afterload, it is that force that the heart must make to counteract the resistance opposed to the expulsion of blood. This opposition generated by systemic vascular resistance, aortic stenosis, and at the level of the pulmonary circulation such as pulmonary embolism [3].

Diastole is divided into 4 phases, which occur during the cardiac cycle. Isovolumic relaxation, fast filling, slow filling or diastasis and atrial systole. The first diastolic phase constitutes the isovolumic relaxation period, this consists of the relaxation of the ventricles, with the consequent decrease in intraventricular pressure so that the filling period occurs. Then the opening of the mitral valve begins, by decreasing the intraventricular pressure below the value of the atrial pressure. The opening of the tricuspid and mitral valves allows the consequent filling of the ventricles with a slight increase in pressure (around 5 mmHg), this filling is rapid. Later, the flow slows down from the atria to the ventricles due to the slight increase in pressure in the ventricles. As atrial pressure increases, between about 4-8 mmHg, it contracts, preparing the ventricles for the onset of systole. Approximately 70% of the blood volume flows directly from the atria to the ventricles. Left ventricular diastole begins from the time of aortic valve closure until the mitral valve closure [2].

Diastolic Dysfunction

During the ventricular diastole period, it would be normal for myocardial relaxation to be rapid and complete; and the ventricle receives blood from the venous system in an amount adequate to maintain the stroke volume. However, when there is dysfunction, this includes an alteration in diastolic ventricular compliance; that

is, it affects cardiac filling, when the ventricle is relaxed. It is a condition in which, to sustain normal cardiac output, the filling pressure is increased [4].

The incidence of diastolic dysfunction in selected populations of critically ill patients is estimated to range from 40% to 80%. It is difficult to differentiate systolic or diastolic dysfunction clinically, since the signs and symptoms are the same, so other methods are necessary to guide the diagnosis, such as cardiac catheterization, echocardiography, and natriuretic peptide. Cardiac catheterization remains the gold standard for the diagnosis of diastolic dysfunction, but the consequences of its invasiveness and the risk of using it as a routine tool must be taken into account. The echocardiogram is non-invasive and is increasingly available in ICUs [5].

At the pathophysiological level, in diastolic dysfunction there is a dissociation of the actin-myosin bridges that occurs when there is a decrease in ATP hydrolysis and less calcium uptake by the sarcoplasmic reticulum, which generates a delay and a prolongation in the expansion of the sarcomere. In simpler words, ventricular filling pressure increases, due to the inability of the cardiac chamber to relax and fill with blood, before the next ventricular contraction [6].

Conditions such as chronic arterial hypertension, myocardial ischemia, diabetes, restrictive heart disease, aortic stenosis, hypertrophic and dilated cardiomyopathy, senility, among others. The decrease in compliance is also affected by infiltrative processes of the myocardium, amyloidosis, hemochromatosis, and myocardial fibrosis. Finally, it all comes down to diastolic involvement and, consequently, preload [9].

Diastolic Heart Failure

The classification of heart failure is based mainly on the left ventricular ejection fraction (FeVi), which can be preserved, which is normal, or reduced, being <40%. Diastolic heart failure corresponds to those patients with signs or symptoms of insufficiency who present preserved FeVi plus diastolic dysfunction. This is normal when it is above 50%. It is determined by echocardiography, and it is the amount of blood that the heart expels each time it contracts [4, 7, 9].

Other tools that are useful are the chest x-ray where cardiomegaly or signs of pulmonary congestion can be evidenced and the electrocardiogram, where the findings are nonspecific, such as atrial fibrillation or signs of old ischemia, however, these must always be correlated with the patient's clinic. At the laboratory level there are natriuretic peptides, their values are lower than in patients with heart failure and reduced ventricular ejection fraction, although if they are elevated they are associated with adverse cardiovascular outcomes. Epidemiologically, its prevalence in general can affect from 1% to 14% of the population in the United States and Europe. In Colombia, according to a study published in 2012, heart failure has a prevalence in the general population of 2.3%, with 67.5% being heart failure with preserved EF. Heart failure with preserved ejection fraction is not only common today, but its incidence is increasing at a rate of 1% per year; so its study should be of increasing interest [6].

Causes of diastolic dysfunction
Arterial hypertension
Diabetes
Coronary ischemic disease
Hypertrophic heart disease
Restrictive heart disease
Dilated heart disease
senility

Graph 1: Causes of diastolic dysfunction

Anesthetics in Diastolic Insufficiency

The choice of anesthetic technique is very important in perioperative management; An integration of the information given by the cardiologist, internist, surgeon and those in charge of postoperative care should be made to develop an individualized perioperative management plan, led by the anesthesiologist who will make the respective analysis and make the final decision. The choice of anesthetic technique should be analyzed, deciding between general anesthesia or neuraxial techniques, and deciding whether to anesthesia with halogenated volatile liquids or intravenous anesthetics. All this in order to reduce perioperative morbidity and mortality.

In general, most anesthetic techniques reduce sympathetic tone, leading to a decrease in venous return due to an increase in the compliance of the venous system, arterial vasodilation and finally a decrease in blood pressure; This is why anesthetic management must guarantee an adequate organic perfusion pressure. There is no exact target value for blood pressure, however, patients with diastolic ventricular failure are more sensitive to hemodynamic changes, so cardiac output must be preserved and myocardial work minimized. The evidence is contradictory, in the case of ischemic heart disease, it is suggested that inhaled agents provide greater cardioprotection than intravenous ones. Intravenous and inhaled anesthetics are associated with cardiovascular depression that can occur due to alteration of the sympathetic system, the contractile function of the myocardium, or the modification of cardiovascular control mechanisms [16].

Anesthetic Considerations

As in diastolic dysfunction there is an affectation in the filling, the heart must have the necessary time to fill the chamber, reason why the increase of the heart rate must be avoided since it reduces this time. In addition, in the case of arrhythmias such as atrial fibrillation, these should be prevented since the diastolic phase depends on the physiological atrial contraction that occurs during the cardiac cycle, as it is not performed properly, it can lead to an affectation of preload and output cardiac. The choice of regional versus general anesthesia is still debated and there are no definitive recommendations in any way. In addition, for general anesthesia, intravenous induction and maintenance with balanced anesthesia of volatile agents and opioids is recommended. What speaks more of a use of multiple anesthetics, but not of the superiority or preference of one or the other. Control of systolic blood pressure should be within 10-20% of baseline value and should be kept below diastolic BP, to prevent increased ventricular wall stress and not in-

crease cardiac workload [10].

Etomidate and ketamine are very useful anesthetics in unstable patients, however, in ventricular failure their use has not been clarified. For anesthetic induction, etomidate shows to be better than Propofol in terms of greater hemodynamic stability. This is relevant since a patient with heart failure often has more severe comorbidities, which aggravate its condition, so there is a great risk of post-induction hypotension, and present increased morbidity and mortality. What makes etomidate a less reliable drug is its adrenal suppressing effect, although the evidence is not clear, it must be taken into account, when evaluating the risks vs benefits. On the other hand, this drug is an inducer that does not inhibit sympathetic tone, therefore, it does not affect cardiac function significantly and is associated with minimal changes in heart rate and blood pressure, without affecting cardiac contractility in patients with impaired ventricular relaxation [12, 14].

Propofol is one of the most widely used anesthetics, it is a hypnotic that is widely used during the induction and maintenance of anesthesia. Its advantages are that it has minimal side effects, a controllable anesthetic state, a rapid onset, and a rapid exit of the patient from general anesthesia. However, it is associated with cardiovascular depression, which manifests itself with a reduction in systemic blood pressure as a result of decreased sympathetic tone and systemic vascular resistance. Another aspect to take into account is its negative inotropic effect, mediated by a decrease in calcium reuptake by the sarcoplasmic reticulum, and which modulates the phosphorylation of myofibrils, mechanisms directly involved in myocardial muscle relaxation. Cases of Propofol have also been documented as an antiarrhythmic, this is important since arrhythmia is one of the aggravating factors of diastolic dysfunction. Kannan and Sherwood reported a case in which a 68-year-old man with a previous myocardial infarction experienced supraventricular tachycardia, with the administration of adenosine or carotid sinus massage there was no significant effect, but propofol managed to convert the supraventricular arrhythmia into sinus rhythm before of electrical cardioversion. Calcium channels are involved in cardiac function that trigger excitation-contraction coupling, modulate the shape of the action potential, and participate in cardiac arrhythmia, so the negative inotropic effect of propofol could be explained by its inhibition [13].

Ketamine increases sympathomimetic activity by central stimulation of the autonomic nervous system and by inhibiting the reuptake of intra and extraneuronal catecholamines, it has a positive inotropic effect, which implies an increase in blood pressure, heart rate and cardiac output. This indicates that ketamine is not an appropriate anesthetic in a patient with diastolic dysfunction, since it does not sustain adequate hemodynamic stability. Since there is the risk of tachycardia, which would worsen the patient's condition, since there is an increase in heartbeat, at the same time the ventricular filling time decreases, there is greater oxygen consumption by the myocardium, which increases the energy demand, there is an increase in systemic vascular resistance, so the heart is exposed to myocardial stress and overexertion since the opposing force to the ejection of the blood will be increased. The positive inotropic effect of ketamine is also accompanied by a greater release of calcium from the sarcoplasmic reticulum with an increase in its concentration in the cytosol, aggravating ventricular relaxation [10, 15].

As for opioids, such as fentanyl, they do not cause depression, have cardioprotective effects, antiarrhythmic activity, prolong depolarization time, can produce peripheral vasodilation, both venous and arterial, the use of these in patients with diastolic dysfunction should prevent the decrease in diastolic pressure that may affect coronary perfusion pressure [10].

Inhaled anesthetics reduce myocardial contractility in a dose-dependent manner by altering calcium homeostasis, with deterioration of contractile function that manifests hemodynamically with alteration of the left ventricular end systolic pressure-left ventricular volume ratio. It prolongs isovolumic relaxation and reduces the rate of early left ventricular filling, without affecting the elastic properties of the myocardium, that is, they are associated with a negative inotropic effect but not a negative lusiotropic effect (relaxation). Another effect more related to volatile anesthetics is the reduction of the atrial contribution [11].

Conclusion

The use of anesthetics in diastolic heart failure should be further studied due to the implications it has at the cardiovascular level when inducing a patient. These are more sensitive to hemodynamic changes and intracavitary pressures, which generates a high risk of imminent decompensation during the surgical procedures these patients undergo regardless of whether they are cardiac or not. Propofol is one of the most used anesthetics, it allows adequate induction and anesthetic recovery. However, in patients with general diastolic dysfunction, caution should be exercised as it can lead to cardiovascular depression, which manifests itself with a reduction in systemic blood pressure as a result of decreased sympathetic tone and systemic vascular resistance. Ketamine has a positive inotropic effect, so that in patients with diastolic dysfunction it represents an increased risk since it does not sustain hemodynamic stability. Inhaled anesthetics reduce myocardial contractility in a dose-dependent manner by altering calcium homeostasis, reducing contractile activity and prolonging isovolumic relaxation without affecting the elastic properties of the myocardium, and can also be used as co-ductors. Anesthetic management in patients with diastolic heart failure is complex and high risk, which is why it must be studied in depth in a multidisciplinary manner, the conditions of the patient must be known as a whole and the considerations that may put their lives at risk patients.

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