Review Article

Diastolic dysfunction and atrial fibrillation in coronary heart disease surgery: A literature review

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ABSTRACT

Diastolic dysfunction can cause atrial fibrillation through the following mechanisms: increased atrial afterload, atrial stretch, and atrial wall pressure due to dilatation. Diastolic dysfunction is often overlooked in coronary heart disease than systolic (left ventricular) function, even though diastolic dysfunction can also result in significant morbidity and mortality. Diastolic dysfunction is an independent predictor of atrial fibrillation. Diastolic dysfunction enlarges the left atrium, stretches the insertion site for pulmonary veins, and initiates atrial fibrillation. Atrial remodelling in atrial fibrillation and diastolic dysfunction progresses from metabolic changes (phosphorylation) to gene expression changes (calcium channel downregulation) to hibernation (myolysis, dedifferentiation) and culminates in irreversible changes (fatty changes).

1. Introduction

Coronary heart disease is the leading cause of cardiovascular death. According to CDC data, coronary heart disease caused 360,900 deaths in 2019. Two out of 10 coronary heart disease deaths occur in adults under 65 years old [1]. In addition to optimizing medical therapy, revascularization is essential to managing coronary heart disease. Coronary artery bypass surgery (CABG) still serves as the primary method of revascularization, particularly in patients with multi-vessel disease or left primary disease [2]. Atrial fibrillation is a relatively common complication of CABG. Mathew et al. reported that the incidence of atrial fibrillation after CABG was 27% [3]. Post-operative atrial fibrillation will increase mortality and morbidity, including length of intensive care and hospitalization, hemodynamic disorders, and the risk of congestive heart failure, renal insufficiency, neurological events, stroke and even death [3,4].

Diastolic dysfunction refers to an abnormality of mechanical function during diastole. Diastole happens when the myocardium loses its ability to generate power and shortens and returns to its relaxed length. Diastolic dysfunction occurs when the process is prolonged, slowed, and incomplete [5]. Diastolic dysfunction causes several mechanisms that can support the occurrence of atrial fibrillation. These mechanisms include increased atrial afterload, atrial stretch, and atrial wall pressure

due to dilatation [6]. In coronary heart disease, diastolic dysfunction is often less noticeable than systolic (left ventricular) function, even though diastolic dysfunction also results in significant morbidity and mortality [5].

1.1. Collecting data

We collect our references using the preferred reporting items for systematic reviews and meta-analyses (PRISMA) statement [7,8]. In addition, we also use a measurement tool to assess systematic reviews (AMSTAR 2) to analyze the results of our review [9].

1.2. Diastolic dysfunction

Conceptually, diastole is when the myocardium loses its ability to generate force, shortens, and returns to its relaxed length and force. Diastolic dysfunction occurs when the process is prolonged, slowed, or incomplete [5]. Left ventricular diastolic function abnormalities can be found in most patients with coronary heart disease. This abnormality occurs in decreased left ventricular filling and increased time-to-peak filling rates. This phenomenon is a clinical reflection of ischemia affecting the rate of diastolic relaxation of the papillary muscle, related to the fact that relaxation of the myocardium during early diastole is an

active and energy-dependent process. Abnormalities of diastolic function in patients with coronary heart disease also reflect a lack of increased coronary flow during diastole [10,11].

2. Measurement of diastolic function

2.1. Relaxation

Diastole includes the period in which the myocardium loses its ability to generate force, shortens, and then returns to strength and rest length. Relaxation occurs in a series of energy-consuming steps, starting with the release of calcium from troponin C, the release of actin-myosin cross bridges, phosphorylation of phospholamban, sarcoplasmic reticulum calcium ATPase-induced absorption of calcium into the sarcoplasmic reticulum, sodium/calcium exchanger-induced extrusion of calcium from the cytoplasm, slows the rate of the cross-bridge cycle, and the elongation of the sarcomere to its resting length. This process requires an adequate supply of energy and mechanisms to regenerate at an adequate rate. The degree and extent to which these cellular processes occur determine the degree and extent of active ventricular relaxation. This process decreases left ventricular pressure at the ventricular level at constant volume (isovolumic relaxation). It then fills the left ventricular space with variable left ventricular pressure (auxotonic relaxation). Measurements taken during auxotonic relaxation are affected by both active relaxation and passive stiffness [5,12].

Isovolumic relaxation can be measured by measuring left ventricular pressure with a high-fidelity micromanometer catheter and calculating the instantaneous peak left ventricular pressure drop rate, peak $(-)$ dP/dt, and isovolumic left ventricular pressure drop time constant, τ. When the natural log of LV diastolic pressure is plotted against time, it equals the inverse slope of this linear relationship. The time required for the LV pressure to fall to approximately two-thirds of its initial value is expressed in more conceptual terms. When the isovolumic pressure drop is slowed down, τ is extended, and the numerical value of τ increases. Echocardiographic techniques can make a noninvasive estimate of the total isovolumic relaxation time. No relaxation index (isovolumic or auxotonic) can be considered an "intrinsic" relaxation level index unless the loading conditions (and other modulators) are held constant or at least specified. One practical way to overcome this limitation is to examine the relaxation index over the load range. Afterload can be altered acutely by mechanical or pharmacological methods. Abnormal

relaxation is indicated by a shift in the position of the relaxation-versus-afterload relationship, where relaxation is slowed at each equivalent systolic pressure (Fig. 1) [12].

On the other hand, active relaxation, in the strictest sense, is an early diastolic event, the time of onset of this process depends, at least in part, on a systolic event such as the duration of contraction. In contrast, the timing of the onset of relaxation may modify the systolic event. Therefore, the rate and degree of relaxation, in addition to being dependent on ventricular load, also depend on the duration of systole, the onset of relaxation, and the time during systole during which the load is changed. Suppose the onset of relaxation is delayed (e.g., with an increase in load early in systole). In that case, this can prolong the systole's duration, increase the heart's work during systole, and prolong relaxation. Conversely, if the onset of relaxation occurs earlier (e.g., due to increased load at the end of systole), this may shorten the systole's duration and relaxation. Thus, complex interactions between events traditionally thought to occur during systole may influence the measurement and interpretation of active relaxation. The diastole's auxotonic left ventricular filling phase can be characterized by Doppler echocardiography or radionuclide, conductance, or MRI techniques. Although each technique has advantages and disadvantages, all assess diastolic function by measuring the transient volume index during ventricular filling. However, like all relaxation indices, the auxotonic index must be interpreted based on simultaneous load changes, both afterload and loading (load present during loading). For example, the precise pattern of early and late diastolic transmission flow rates will depend on factors regulating atrial and LV pressures before and after mitral valve opening and the resultant atrial-ventricular pressure gradient (filling load). Thus, it is unsurprising that interventions or pathological conditions that increase left atrial pressure increase the initial transmission flow rate. In contrast, interventions that reduce left atrial pressure reduce the initial filling rate. In order to correctly interpret changes in the transmission flow rate, changes in the loading load must be considered. Additional indices that may be less sensitive and may indicate changes in load are currently being investigated. These include pulmonary venous flow rate, transmission propagation velocity, and tissue Doppler velocity (Fig. 2) [5].

2.2. Myocardial stiffness

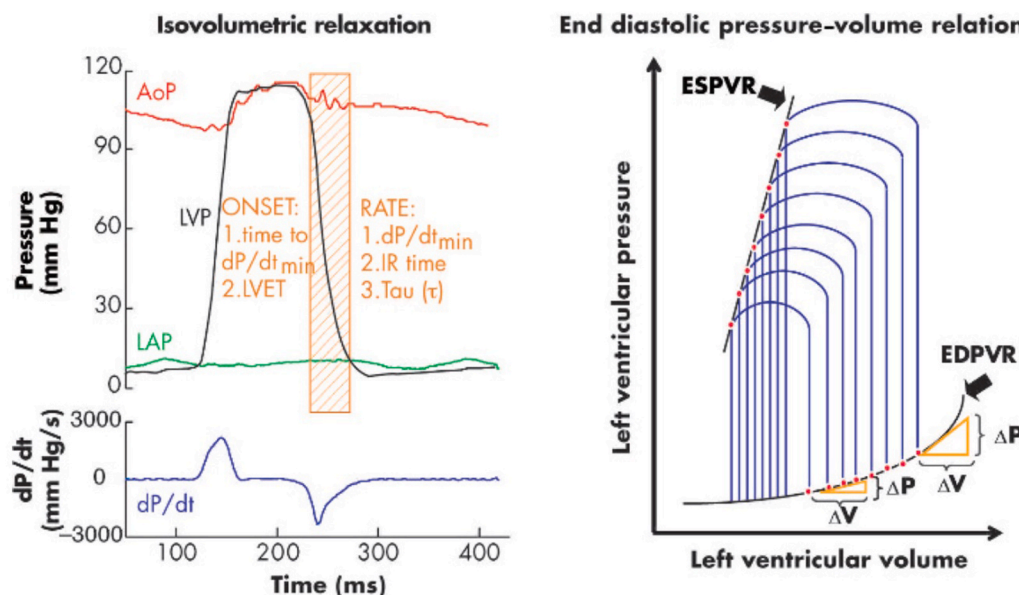In addition to active relaxation, passive viscoelastic properties

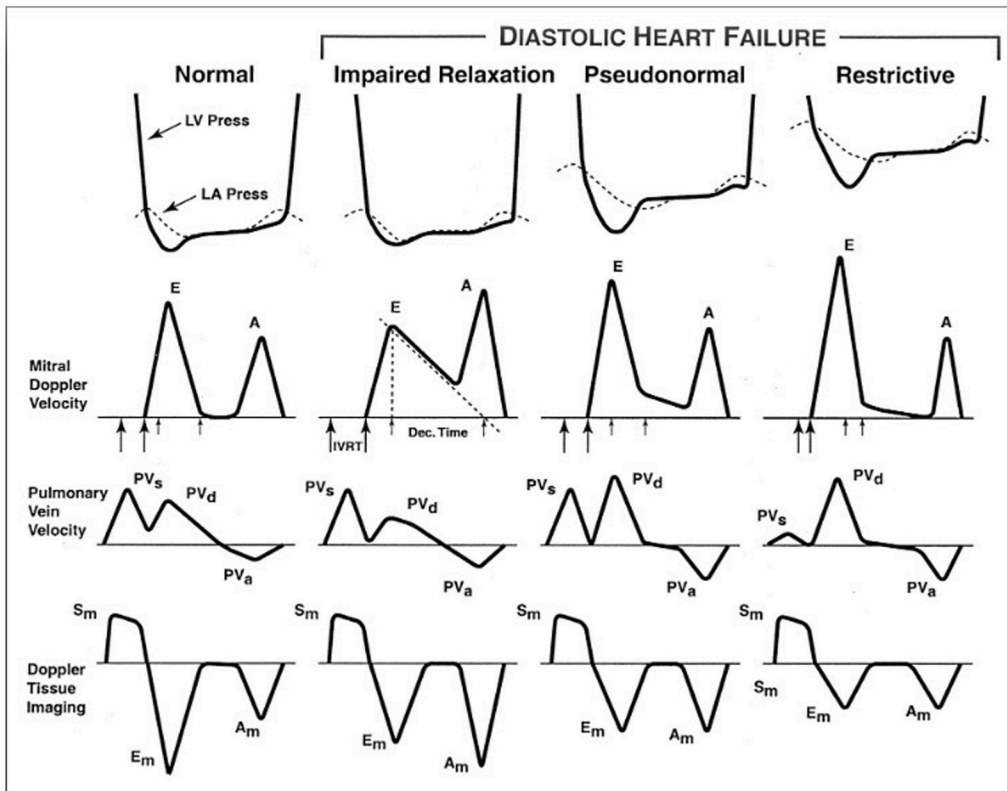**Fig. 1.** Hemodynamic evaluation of diastolic function [12].

Fig. 2. Degree of diastolic dysfunction [5].

contribute to the processes that return the myocardium to its strength and length of rest. This passive viscoelastic property depends on the intracellular and extracellular structures. Changes in ventricular chamber stiffness can be assessed by examining the pressure-volume relationship during diastole. Space stiffness is determined by the myocardial stiffness of its constituents and the LV mass and LV mass/volume ratio. Changes in myocardial stiffness can be assessed by examining myocardial stress, strain, and strain rate relationships during diastole [5].

Space stiffness can be measured by examining the relationship between diastolic pressure and volume. The operating stiffness at any point along a given pressure-volume curve is equal to the slope of the tangent drawn to the curve at that point ($\Delta P/\Delta V$) (Fig. 3). The operational stiffness changes during filling; stiffness is lower at smaller volumes and higher at larger volumes (volume-dependent changes in pressure and diastolic stiffness). Since the relationship between diastolic volume pressure is curvilinear and generally exponential, the relationship between $P/\Delta V$ and pressure is linear; The slope (Kc) is called the space stiffness modulus (or space stiffness constant) and can be used as a single numerical value to measure the space stiffness. As the overall space stiffness increases, the pressure-volume curve shifts to the left, the slope of the $P/\Delta V$-versus-pressure relationship becomes steeper, and Kc increases (volume-independent changes in pressure and diastolic stiffness). Thus, the diastolic pressure can be changed by a volume change depending on the operating stiffness or by a volume change independent of the chamber stiffness [5].

Cardiac muscle behaves as a viscoelastic material, developing a resisting force (stress, σ) as the myocardial length (strain, ε) increases with ventricular filling. A strain is a deformation (increase in length) of a muscle produced by applying a force (increase in stress). Myocardial stiffness can be measured by examining the relationship between stress and myocardial tension during diastole. Myocardial stiffness equals the slope ($d\sigma/d\varepsilon$) of the tangent drawn to the strain-strain relationship at a given strain. Since the stress-strain relationship is curvilinear and exponential, the relationship between d/d and stress is linear, and the
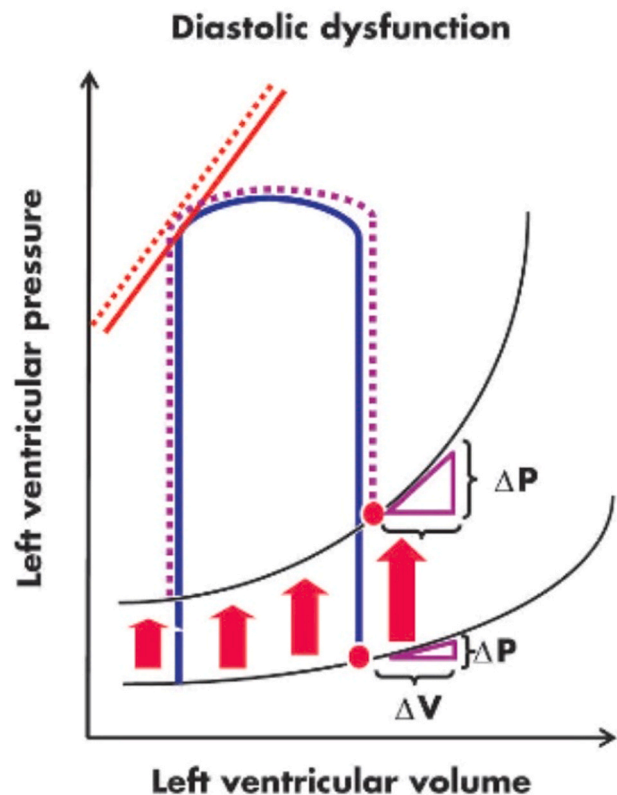

Fig. 3. Volume relationship curves and left ventricular pressure [12].

slope of this relationship, K_m, is the myocardial stiffness modulus (or myocardial stiffness constant). As myocardial stiffness increases, the stress-strain relationship shifts to the left so that for any given change in myocardial length (strain), there is a more significant increase in the force (wall stress) that develops to resist this deformation. In addition, the slope of the $d\sigma/d\varepsilon$-versus-stress relationship becomes steeper, and K_m increases as myocardial stiffness increases [5].

Based on diastolic parameters, diastolic dysfunction is divided into 3 grades, namely grades 0 to 4. Grade 0: normal left ventricular (LV) filling pressure; mitral E/A ratio 0.75–1.5; mitral deceleration time 160–240 ms, e'≥8, E/e' <8, pulmonary vein systolic forward flow rate > diastolic, pulmonary vein atrial reversal duration shorter than mitral A flow duration and left atrial volume index (LAVI) < 34 mL/m². Grade 1: LV filling pressure is normal to slightly elevated or left ventricular relaxation is impaired; mitral E/A ratio <0.75, mitral deceleration time >240 ms, e'<8, E/e' 8, pulmonary vein systolic forward flow rate ≥ diastolic, pulmonary vein atrial reversal duration shorter than mitral A flow duration, and LAVI 34 mL/m². Grade 2: moderately elevated LV filling pressure or pseudonormal left ventricular diastolic filling; Mitral E/A 0.75–1.5 with a decrease of 0.5 with Valsalva manoeuvre, mitral deceleration time 160–240 ms, e'<8, E/e' 9–12, pulmonary vein systolic forward flow rate < diastolic, the duration of pulmonary venous atrial reversal was longer than the duration of mitral A flow (≥30 ms), and LAVI was 34 mL/m². Grade 3: severe increase in LV filling pressure or restriction of LV diastolic filling; Mitral E/A >1.5 with a decrease of 0.5 (reversible) or <0.5 (fixed) with Valsalva manoeuvre, mitral deceleration time <160 ms, e'<8, E/e' vein systolic forward flow rate pulmonary < diastolic, pulmonary vein atrial reversal duration longer than mitral A flow duration (≥30 ms), and LAVI 34 mL/m² [13].

2.3. Coronary artery bypass surgery with cardiopulmonary machine (on pump CABG)

Coronary artery bypass surgery is a form of surgical myocardial revascularization. This action can be conducted with or without a cardiopulmonary machine [14]. In some studies, the advantage of coronary artery bypass surgery with a cardiopulmonary machine is complete revascularization [15,16]. Coronary artery bypass surgery without a cardiopulmonary machine tends to have a higher incidence of repeat revascularization. In addition, coronary artery bypass surgery with a cardiopulmonary machine is preferred over the alternative in emergencies. In some studies, it is also said that mild hypothermia in coronary artery bypass surgery with cardiopulmonary machines is said to be beneficial for cerebral protection so that it can avoid neurophysiological deficits. Another advantage is the familiarity with the technique, related to the coronary artery bypass technique without a cardiopulmonary machine which requires a relative learning curve [14,17].

Disadvantages of coronary heart bypass surgery with cardiopulmonary machines include the impact of inflammation, the risk of kidney dysfunction, and gastrointestinal stress [17]. Exposure to extracorporeal circulation/cardiopulmonary machinery triggers activation of the complement cascade, activation of neutrophils, and release of endotoxins from the mucosal surface, and changes in perfusion may result in ischemia-reperfusion injury. The other impact is the production of oxygen free radicals, cytokines, platelet activation, and inflammatory mediators [18].

2.4. Pathophysiology of coronary artery bypass surgery with cardiopulmonary machine

When the heart is stopped, the cardiopulmonary machine maintains circulation. The cardiopulmonary machine can cause complications in organs such as the heart, lungs, kidneys, and brain. Mechanisms that contribute to this include a contact of the plasma and the surface of the cannula, shear force between the plasma and the cardiopulmonary machine, hypothermia, and ischemia-reperfusion injury [19]. The

pathophysiology of the cardiopulmonary machine occurs through two activation mechanisms: material-dependent and non-material activation. Material-related activation is initiated by blood contact in the cardiopulmonary machine circuit. The contact is related to the humoral defence system. Activation of the humoral defence system will activate the kallikrein, fibrinolytic, and coagulation systems. Activation of these products, either directly or indirectly, will activate leukocytes, platelets, and endothelium. Activation of the complement system also occurs due to blood contact with foreign materials. Complement activation specifically complements C3a, C5a, and TCC (terminal complement complex). The impact of this complement activation will damage the cell membrane [20].

Complement formation also plays an essential role in leukocyte recruitment, upregulation of neutrophil activation markers, and cytokine production. The literature states that the incidence and degree of impaired cardiac, pulmonary, and renal function after cardiopulmonary machinery is related to the plasma concentration of complement fragment 3a. Furthermore, inhibition of TCC production by protease inhibitors was also associated with a significant reduction in the detrimental effect of ischemia-reperfusion on the myocardium after cardiopulmonary machinery [21].

Myocardial edema is one of the effects of coronary artery bypass surgery. This edema can be detrimental because it can affect the systolic and diastolic function of the heart. Cardioplegic fluid is a hypooncotic solution and supports myocardial edema. Lymphatic drainage is reduced when the heart is stopped and contributes to myocardial edema [22]. When the cardiopulmonary machine runs, the heart stops in the diastolic phase. This includes impaired contraction, increased transmicrovascular fluid flow, activation of cellular and humoral mediators, and increased microvascular permeability [14,19].

Ischemia-reperfusion injury is one of the consequences of coronary artery bypass surgery, specifically CABG with a cardiopulmonary machine. Ischemia-reperfusion injury results from temporary occlusion of a coronary artery. Circulatory flow returns with a greater quantity of blood compared to coronary flow before surgery. Mechanisms that could explain ischemia-reperfusion injury are apoptosis due to the accumulation of free radicals through anaerobic metabolism during ischemia and intracellular calcium ion overload. This ischemia-reperfusion injury may manifest as arrhythmias, stunning, low cardiac output, and perioperative myocardial infarction [23].

Electrolyte fluctuations, especially sodium, are also from coronary artery bypass surgery. These fluctuations may occur through antidiuretic hormone-return mechanisms, volume dilution due to surgical field suctioning, hypothermia, and pH offset. Fluctuations in sodium >5meq/L can lead to complications, including arrhythmias. Fluctuations >12 meq/L were associated with impaired mortality in the ICU, whereas fluctuations >15 meq/L were associated with post-operative seizures [24]. Cardiopulmonary machines or extracorporeal circulation use in coronary artery bypass surgery also has various effects that can occur in various body organs (Table 1) [25].

2.5. Atrial fibrillation

Atrial fibrillation is a supraventricular tachyarrhythmia with uncoordinated atrial electrical activation and ineffective atrial contraction. Characteristics of the electrocardiogram (ECG) of atrial fibrillation include irregularly irregular R-R intervals (when atrioventricular conduction is not impaired), absence of marked P waves, and irregular atrial activation. Atrial fibrillation was classified into several categories based on the presentation, duration, and spontaneous termination of spontaneous episodes (Table 2). Based on the clinical type, atrial fibrillation can be classified into AF secondary to structural cardiac disease, focal AF, polygenic AF, post-operative AF, AF in mitral stenosis or prosthetic heart valves, AF in athletes, monogenic AF (Table 3) [26]. In surgery, it is often known as post-operative atrial fibrillation (POAF). The risk factors for post-operative atrial fibrillation by time are preoperative,

Table 1

The impact of the cardiopulmonary machine on the Body's organs.

Organ	Impact
Blood	Decreased platelet count and decreased blood clotting factors.
Brain	Depending on the perfusion pressure and blood pressure, no disturbance occurs when the pressure is maintained sufficiently. Post-operative psychic or neurologic changes are often associated with emboli by fibrin, air, antifoam, or endogenous fragments. Brain edema due to fluid accumulation in the intracellular space can be treated by water sequestration (diuretics, corticosteroids). Hydrops of astrocyte cells or myelin edema indicates fatal conditions.
Lungs	Decreased lung function due to decreased volume and inadequate ventilation. Post-perfusion syndrome may occur in the form of pulmonary edema, pulmonary interstitial hemorrhage, atelectasis, or hemothorax. This can lead to left atrial overdistention leading to pulmonary hypertension and is often fatal.
Kidney	Depression of glomerular filtration rate and renal plasma flow by up to 50%, which usually returns to an average of 1 h after completion of extracorporeal circulation. Administration of diuretics or intraoperative hemodialysis may be considered. No spontaneous return of renal function indicates renal insufficiency. It also depends on the preoperative evaluation of the patient's renal function. Mannitol, which reduces post-glomerular renal vascular resistance, prevents a decrease in renal plasma flows during perfusion without affecting the glomerular filtration rate.
Heart/ myocardium	To prevent left ventricular distension due to the accumulation of blood, it is advisable to install a "vent" in the left ventricle in the old procedures. Myocardial distention can cause sl-myocardial/myocyte edema. Complications that often occur in the myocardium are side effects of cooling or drugs due to the formation and accumulation of lactic acid in the myocardium, loss of blood glycogen and ATP and a decrease in blood pH.

Table 2

Atrial fibrillation classification.

Atrial fibrillation pattern	Definition
First diagnosed/new onset	Previously undiagnosed AF, regardless of duration or presence/severity of AF-related symptoms.
Paroxysmal	AF that ends spontaneously or with intervention within 7 days of onset.
Persistent	AF that persists for more than 7 days, including episodes ending in cardioversion (drug or electrical cardioversion) after 7 days.
Long persistent	Continuous AF of >12 months when deciding to adopt a rhythm control strategy.
Permanent	AF is acceptable to both patient and physician, and no further attempts to restore/maintain sinus rhythm will be made. Permanent AF represents the therapeutic attitude of the patient and physician rather than the pathophysiological attribute inherent in AF. The term should not be used in a rhythm control strategy with antiarrhythmic drug therapy or AF ablation. If a rhythm control strategy is adopted, the arrhythmia will be reclassified as 'long persistent AF'.

Table 3

Clinical type of atrial fibrillation.

AF Type	Clinical Presentation	Pathophysiology Possibilities
Secondary AF to structural heart disease	AF in patients with left ventricular systolic or diastolic dysfunction, longstanding hypertension with left ventricular hypertrophy, and/or structural heart disease. The onset of AF in these patients is frequent enough to lead to hospitalization and is a poor predictor of outcome.	Increased atrial pressure and remodelling of atrial fibrillation with activation of the sympathetic and renin-angiotensin systems.
Focal AF	Patients with recurrent and frequent paroxysmal AF. Often symptomatic, young patients with marked atrial waves (rough AF), atrial ectopy, and/or atrial tachycardia in AF.	In most cases originating in the pulmonary veins, local triggers initiate AF. AF due to one or more re-entrant thrusters is also considered part of this type of AF.
Polygenic AF	AF in carriers of specific gene variants associated with early-onset AF.	Currently under research. The presence of specific gene variants can also affect the outcome of treatment.
Post-surgery AF	New-onset AF (usually self-limiting) after major cardiac surgery in patients with previous sinus rhythm and no history of AF.	Acute factors: inflammation, oxidative stress, high sympathetic tone, electrolyte changes, and volume overload, possible interactions with pre-existing substrates.
AF in patients with mitral stenosis or heart valve prosthesis	AF in patients with mitral stenosis, after mitral valve surgery and, in some cases, other heart valves.	Left atrial pressure and volume loading are the main drivers of atrial enlargement and atrial remodelling in these patients.
AF in athlete	Usually paroxysmal, related to the duration and intensity of exercise	Increased vagal tone and atrial volume
Monogenic AF	AF in patients with hereditary cardiomyopathy, including channelopathies	Arrhythmogenic mechanisms responsible for sudden death may contribute to these patients.

intraoperative, and post-operative (Fig. 4) [27].

2.6. Atrial fibrillation pathophysiology

Risk factors that can cause atrial fibrillation include sex (male), aging, ethnicity (non-Caucasian), genetics, hypertension, diabetes, chronic kidney disease, inflammation, chronic obstructive pulmonary disease, heart valve disease, heart failure, heart disease, coronary disease, acute surgical disease, alcohol consumption, obesity, smoking [26,28]. Various factors cause complex atrial changes, including stretch-induced fibrosis, hypocontractility, fat infiltration, inflammation, vascular remodelling, ischemia, ion channel dysfunction, and calcium instability. All increase ectopy and conduction disturbances, increase the atrial propensity to develop/maintain AF, and facilitate AF-associated hypercoagulable states. Hypocontractility reduces local endothelial shear stress, which increases the expression of plasminogen activator inhibitors, and ischemia-induced inflammation increases the expression of endothelial adhesion molecules or promotes endothelial cell release, resulting in exposure of tissue factors to the bloodstream. AF itself exacerbates many of these mechanisms, which may explain its progressive nature (Fig. 5) [26,29].

2.7. Atrial fibrillation (AF) diagnosis

The clinical presentation of atrial fibrillation can vary from asymptomatic to symptomatic. Complaints include palpitations, shortness of breath, fatigue, tension or chest pain, dizziness, fainting, and sleep disturbances. Hemodynamically, patients can be categorized with atrial fibrillation, hemodynamically stable or unstable. The diagnosis of atrial fibrillation requires documentation of the rhythm with an electrocardiogram (ECG). An episode lasting more than 30 s is a diagnosis of AF [30,31].

2.8. Management

In simple terms, the management of atrial fibrillation is called the ABC holistic pathway or Atrial fibrillation Better Care (ABC). A is anticoagulation/avoid stroke, B is better symptom control, and C is cardiovascular risk factors and concomitant disease: detection and
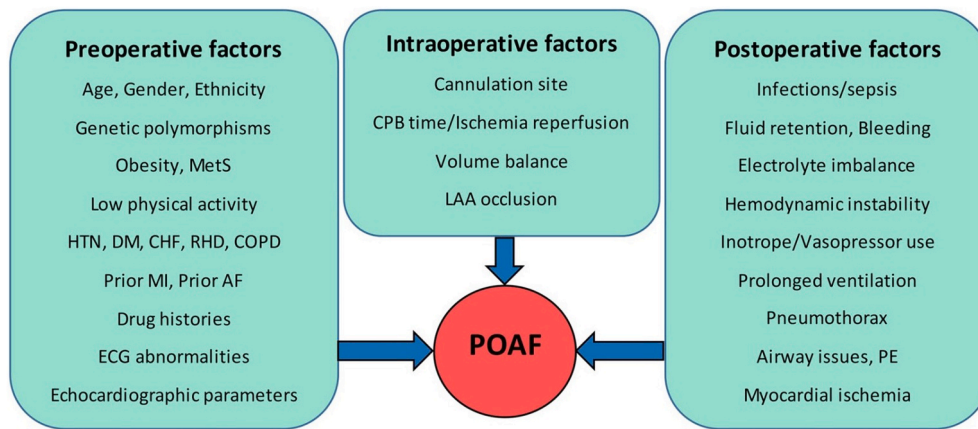
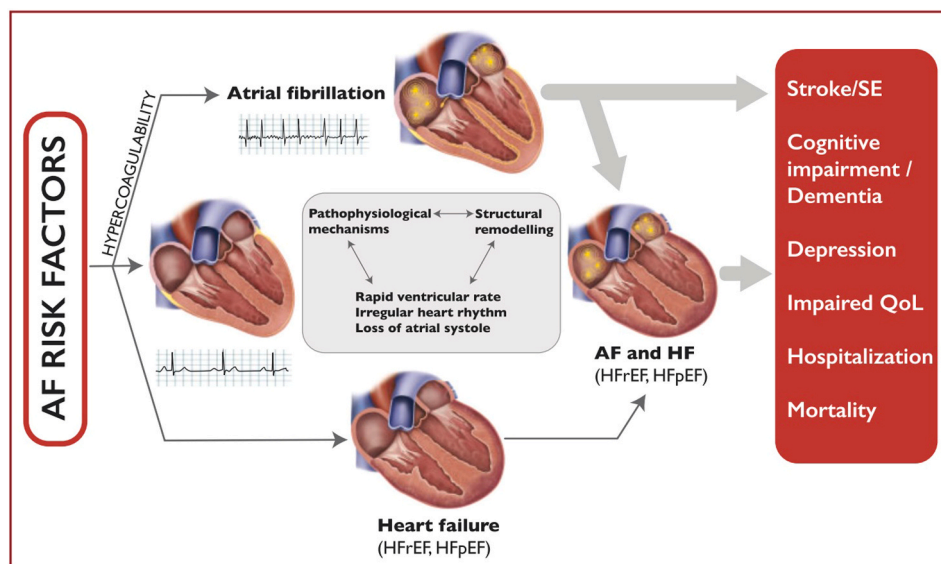**Fig. 4.** Risk factors for post-operative atrial fibrillation [35].

Fig. 5. Atrial fibrillation (AF) pathophysiology [26].

management. The principles of anticoagulation management and stroke avoidance are achieved by assessing stroke risk by scoring, bleeding risk assessment, and stroke prevention therapy. The principle of management of both atrial fibrillation or B (better symptom control) is achieved by rate control either medically with beta-blockers, non-dihydropyridine calcium channel blockers, digoxin, amiodarone, node ablation atrioventricular, rhythm control, cardioversion (electrical or pharmacological), catheter ablation, to surgery. The third management principle is C or cardiovascular risk factors and concomitant disease: detection and management are achieved by controlling risk factors such as lifestyle modifications such as weight reduction, use of caffeine, alcohol, physical activity, and management of specific diseases or comorbidities such as heart failure, heart disease, coronary, diabetes mellitus [26,32].

2.9. Diastolic dysfunction and atrial fibrillation

From a ventricular perspective, diastolic dysfunction includes 3 fundamental problems that occur during ventricular diastole, including impaired ventricular relaxation, decreased ventricular compliance, and increased atrial filling pressures. Although longitudinal studies showing progression through the various phases of diastolic function have been limited, one study did show that individuals who demonstrated worsening diastolic dysfunction in follow-up studies had an increased risk of

death. A classification scheme based on the echocardiographic pattern observed in patients with diastolic dysfunction has been developed. It includes class I (relaxation disorder), class II (pseudonormalization), class III (restrictive filling, reversible), and class IV (irreversible restrictive filling pattern; Fig. 1) [12].

Among the earliest changes in diastolic function are impaired relaxation, which can be caused by abnormal active relaxation, such as from impaired calcium handling and contractile fiber cycle, or abnormal passive recoil due to poor ventricular systolic or deficient structural proteins, such as titin. However, if ventricular relaxation is impaired, less ventricular filling occurs in early diastole, causing more ventricular filling later due to atrial contraction (hence, more giant emitting E and A waves). The impact of these changes on atrial function is less predictable, although based on PV velocity studies, it appears that the atria face a situation of increased preload (pretrial volume) and possibly increased atrial ejection fraction. Under these circumstances, the work performed by the atria increases, which in theory, increases the energy requirements of atrial myocytes and could have significant consequences for future remodelling. One issue of interest is whether this is considered normal in elderly patients, where a peak E/A ratio of <1 is typical, or whether relaxation disorders are part of natural cardiac aging and a significant risk factor for the development of AF [6].

Diastolic dysfunction is an independent predictor of atrial fibrillation

[33]. Diastolic dysfunction enlarges the left atrium, stretches the insertion site for the pulmonary veins, and initiates atrial fibrillation [34]. Atrial remodelling in atrial fibrillation and diastolic dysfunction progresses from metabolic changes (phosphorylation) to gene expression changes (calcium channel downregulation) to hibernation (myolysis, de-differentiation) and culminates in irreversible changes (fibrosis, fatty changes) [30].

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Author contribution

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Registration of research studies

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Declaration of competing interest

The authors declare that they have no conflict of interest.

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