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What It Means Heart Failure?

José Fernando Guadalajara-Boo*

Instituto Nacional de Cardiología Ignacio Chávez, Juan Badiano Num. 1 Col. Sección XVI Deleg. Tlalpan 14080, México, D.F.

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INTRODUCTION

Heart failure is a disease in which there are multiple definitions and the concept is adopted arbitrarily by each author, in such a way that the terminology is so confusing now the authors of the mega trials in medical treatment of heart failure, avoid thus refer to the disease to be treated and are only set to indicate that the value of the ejection fraction is <40 or 35%. In this paper, the goal is the promptly revise the concepts of contractility, ventricular function, preload, afterload and heart failure, compensation mechanisms. These definitions and concepts are based on the original contributions of recognized researchers, in an attempt to clarify the concepts, which was born the niomination of heart failure and thus avoid an erroneous interpretation, almost always motivated by inadequate simplification of terms, in order to scientifically explain the concept of ventricular function and heart failure.

Heart function: "The heart is a muscular pump that generates pressure and move volume, whose function is the supply of oxygenated blood to the tissues of the body and send unsaturated blood to oxygenate the lungs to Life sustaining" [1,2].

In the 20th century, there are hundreds of papers of basic and clinical research on left ventricular function and heart failure, made by great Scientist men. At this review, is intended to analyse those studies that can clarify and give scientific support to achieve an understanding of the concept of heart failure.

Contractility: "It is the intrinsic capacity of the myofibril to shorten its length and its shortening speed independent of the pre and afterload" [1].

In the isolated myofibril contractility can be measured by quantifying degree and speed of shortening to stimulate it directly to a constant initial length and without resistance to its shortening (no-load). The intact heart contractility is very difficult to quantify in its function is always subject to a diastolic load (preload) and a force that has to overcome during his emptying (afterload).

Today the closest way to meet the heart's intrinsic contractile State is the end systolic relationship stress/volume or pressure/volume at which generates a curve that is extrapolated to pressure 0 mmHg, this curve has been called "End systolic elastanse" (E_{max}). This implies that a ventricle has more contractility when reduce the systolic volume higher magnitude to one greater afterload than another whose systolic volume is higher for the same afterload, unfortunately the method is little practical and difficult to achieve in clinical settings but has been very useful for basic and clinic research.

It's common for the term "Contractility" is erroneously used interchangeably to refer to the "ventricular function": ventricular function refers to the relationship between contractility and instantaneous hemodynamic load (preload and afterload) and do not mean intrinsic contractile State (heart contractility) [3].

Preload: It is the length of the myofibril at rest, immediately before ventricular contraction. In the intact heart is represented by the diastolic volume than in normal conditions generates a force that increases the area of the myofibril immediately before contraction (diastolic wall stress) [1].

Afterload: is defined as the force per unit sector area that opposes the ventricular contraction during the emptying of the heart towards the great vessels and obeys to Laplace law, so it is quantified by calculating the systolic wall stress.

$S=P \times r/ 2h^3$

Systolic left ventricular function: "Left ventricular function is the result of the simultaneous interaction of

Corresponding author: José Fernando Guadalajara-Boo, Instituto Nacional de Cardiología Ignacio Chávez, Juan Badiano Num. 1 Col. Sección XVI Deleg. Tlalpan 14080, México, D.F., E-mail: guadalajara@cardiologia.org.mx

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contractility with load (preload or afterload) and is quantified by ejection fraction (EF)" [3]. The normal values of EF: 67 ± 8%.

Diastolic function: It is the ability of the heart to receive the systemic and pulmonary venous return and that it is represented by the diastolic volume. This capacity is dependent on isovolumic relaxation and ventricular distensibility (compliance) [2].

Cardiac reserve: "Is the ability of the heart to increase cardiac output".

A. Chronotropic reserve: "is the ability of the heart to increase the cardiac output by increasing heart rate".

B. Diastolic reserve: "is the ability of the heart to increase cardiac output through the Frank-Starling mechanism, and its limit is pulmonary edema".

C. Systolic reserve: "is the ability of the heart to increase cardiac output through increasing its contractility, which depends on the anatomofunctional integrity of the myofibril (shifts upward Starling curve)".

Heart failure: In 1967, Braunwald et al. defined heart failure: "When the heart loses its ability to supply enough blood to meet the metabolic needs of the tissues of the body in a normal physical activity" [1]. This condition in 1970, Mason et al. [4], called "Decompensated Heart Failure". This definition conceptualized with clarity that heart is not able when it ceases to fulfil its vital role, (tissue perfusion) that is why, if it is not corrected in a period of hours or days, ensues death; and they noted that when this picture appears, the organism avoid this lethal condition, using mechanisms that attempt to restore cardiac output and tissue perfusion; these mechanisms they call "compensatory mechanisms"; Which try to restore the vital function of the heart: tissue perfusion.

Compensatory mechanisms

The Frank Starling mechanism increases ventricular diastolic volume and normalises cardiac output. The enlargement of heart (cardiomegaly) and increased diastolic pressure with increase left ventricular wall stress, triggers secretion of brain natriuretic peptide (BNP); by other hand, the increase atrial wall stretching (stress) triggers ANP secretion. These substances really are hormones that function as internal diuretics and vasodilators allow relieve the congestion pulmonary and systemic venous, which gets a hemodynamic State, that while the heart is in failure (decreased EF), the patient maintains cardiac output and therefore tissue perfusion (Compensated Heart Failure) and absence of systemic and pulmonary congestion and this state, allow that the patient is in functional class I, so in these conditions the patient has **Asymptomatic Compensated Heart Failure** [2]. In these cases ACE Inhibitors have proven to be the most effective treatment to prevent the progression to symptomatic heart failure. When heart failure, appears and the Starling mechanism is not able for maintain cardiac output, stimulates adrenergic system and catecholamine secretion increases the heart rate (chronotropic reserve) and the positive inotropic effect increases contractility, shifts upward the Starling curve (inotropic reserve), increasing cardiac output; and also stimulates RAA system and these mechanism increase cardiac output and tissue perfusion. By other hand, the secretion of angiotensin II increase peripheral resistance and maintain the perfusion pressure; aldosterone secretion retains renal Na+ and water increases the intravascular volume, the preload and cardiac output and compensate heart failure but produce clinical consequences: tachycardia, pallor, oliguria, increases of heart size, dyspnea, edema, pulmonary congestion and hepatomegaly. In conclusion, the patient's symptoms are not by themselves due to heart failure, they are consequence to activation of the compensatory mechanisms, but are those who maintain tissue perfusion and life, then this condition corresponds to **Symptomatic Compensated Heart Failure**. That is, compensation does not refer to the patients has symptoms, it refers to **life preservation** [2,4].

When the compensatory mechanisms fails to restore cardiac output appears tissue hypoperfusion (**the heart loses its vital functions**), with corresponds to **cardiogenic shock** (descompensated heart failure) [2,4].

Decompensated heart failure: "Is the inability of the heart to eject sufficient amount of blood, to maintain an adequate blood pressure, to perfuse oxygen to the tissues of the body. This inability is due to ineffective myocardial contraction either by intrinsic damage of the myofibril or excessive hemodynamic overload" [4].

When contractility is depressed in potentially reversible (hibernating myocardium) systolic reserve is lost until the cause (pharmacological, surgical or Interventional coronary reperfusion) is solved and restores tissue perfusion and life is preserved; but when there is extensive destruction of myofibrils by necrosis (infarction) or inflammation (myocarditis), as in cardiogenic shock, systolic reserve is lost and the application of inotropics is not follow of improvement of EF; so it does not increase the cardiac output by this mechanism (loss of the systolic reserve) and appears **Decompensated Heart Failure** [2,4]: cardiogenic shock this concept, explains the reason for the reduction of mortality of cardiogenic shock with early reperfusion, retrieving the viable myocardial not functioning (hibernating myocardium) at risk of necrosis and restores the systolic reserve, the cardiac output and life of the patient.

When there is extensive myocardial damage and extreme downward deviation of ventricular function curve occurs irreversible cardiogenic shock, without effective treatment, leads to death. The clinical manifestations are: weak pulse, blood systolic pressure <80 mm Hg, peripheral vasoconstriction, cold, wet and bluish skin, oliguria (<50

cc/h), mental confusion, metabolic acidosis, are the true symptoms of **Decompensated Heart Failure in other words the inability of the heart to maintain its vital function (tisular perfusion)** [2,4].

Diastolic dysfuction: "The myocardial or extracardiac alterations that produce an impediment of variable degree to the filling of the heart, which can cause to raise the intraventricular diastolic pressure without increasing the diastolic volume and which coincide with a normal ejection fraction" [2].

Patients who have symptoms like shortness of breath and EF>50% (heart failure "with preserved systolic function") have a mortality rate of 7.6% at 10 years [5], while patients who have heart failure in functional class I (40-50% FE) have a mortality rate of 70%, 82% (FE<40%) and 90% in patients functional class II to IV to 10 years; when the ejection fraction is <50% [6] and the difference in mortality between two groups is explained because patients with heart failure and "preserved systolic function" do not activate the neuroendrocrine system.

CONCLUSION

Heart Failure: "It is a condition in which functional or structural diffuse damage of the myofibril (Hibernation, necrosis, apoptosis, inflammation, toxic effects of drugs), or an excessive hemodynamic overload, causes decrease in contractile force in the heart (hence the EF); with increase the ventricular volumes with (decompensated) or without (compensated) reduction of cardiac output" [1,2,4]..

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