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Review Article

Need for Term Definition in Cardiology-Cardiac Failure and Cardiac Insufficiency: A Short Review

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Abstract

Background: Cardiac failure is often used interchangeably with cardiac insufficiency. This creates issues in patient approach and management outcome.

Objectives: The aim of this Short Review is to highlight the distinction and relationship between cardiac failure and cardiac insufficiency.

Methods: Data were extracted by literature search on PubMed, Google scholar citation, Medline and other repositories.

Results: Cardiac failure is a different form of cardiac insufficiency mainly by the fact that the later has no myocardial damage or contractility failure. There are also no clinical or radiographic features of cardiomegaly in cardiac insufficiency.

Keywords: Cardiac failure; Cardiac insufficiency; Children.

Introduction

Congestive heart failure is considered to be characterized by heterogeneity in presentation according to age group and causative factors[1]. These presumed diverse presentations complicate the assessment, clinical definition, diagnosis, and treatment given to this group of patients with "heart failure" [1,2]. "Heart failure" in children, though etiologically diverse, results in decrease in systemic oxygen transport (SOT) which is usually the unifying pathophysiologic mechanism [3]. However, this unifying pathophysiologic mechanism has failed to critically differentiate between actual heart failure and cardiac insufficiency Pediatric heart failure was often previously classified as heart failure syndromes in order to take into considerations the various clinical manifestations and etiologies that may cause the decrease in systemic oxygen transport [3]. Thus, heart failure syndrome was said to be present when the cardiac output is insufficient to meet the metabolic demands of the body [1,3].

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Cardiac insufficiency refers to inadequate cardiac output to maintain metabolic demand of the body in a background of structurally normal heart muscles while cardiac failure is an abnormal cardiac pumping of blood due to structurally and functionally defective heart muscle [4]. Hence the term heart failure as a general term is a misnomer except when used for inadequate cardiac output due to myocardial pump failure [5]. Both situations will invariably give rise to peripheral circulatory collapse from the resultant reduction in SOT.

Methods

A search for published publications on cardiac failure and cardiac insufficiency was done through several repositories such as PubMed, systematic reviews , google scholar citation and meta-analysis. Keywords such as cardiac failure, cardiac insufficiency, children, chest radiograph, pulmonary hypertension and echocardiography were used.

Pathophysiology

The heart is a pump whose output is proportional to its filling volume and inversely proportional to the resistance against which it pumps [2]. However, as the ventricular end diastolic volume increases, the healthy heart increases cardiac output until a maximum is reached and no further increase in cardiac output can be obtained despite the metabolic demand [3]. This, and other states of circulatory stress and cardiac injury then result in SOT reduction and consequently the body will evoke some compensatory responses.

The trigger for these pathways is the unmet tissue demands which can result from the heart either due to excessive preload, excessive after load, abnormal rhythm, decrease in myocardial contractility or from the tissue damage itself due to excessive demand or increased rate of metabolism [3,4]. The compensatory mechanism so evoked can be beneficial in the short term but if prolonged, that becomes deleterious and worsen the heart failure [2]. These mechanisms include the sympathetic nervous system, the activation of the of renin-aldosterone angiotensin system, cytokine-induced inflammation and signaling cascades that trigger cachexia [2]. Adequacy of SOT which is the goal of these compensatory mechanisms is a product of cardiac output and systemic oxygen content. The formulae below illustrate this [3,4].

$SOT=CO \times [SO_2]$

$CO=SV \times HR$

$SV=VR \times MYC$

$(SO_2)=Hb\ V/P$

$SOT=1/PR \times VR \times MYC \times HR \times Hb \times V/P$

Where SOT=Systemic oxygen transport, CO=Cardiac output, SV=Stroke volume, PR=Peripheral resistance, HR=Heart rate, VR=Venous Return, MYC=Myocardial contractility, So2=Systemic oxygen content. Hb=Haemoglobin,V/P=Ventillation/Perfusion mismatch [3].

From the equation, it is clear that affectations of any or a combination of the components on the right side of the equation will affect SOT and clearly, these affectations are germane. However, it is only the myocardial contractility from a heart muscle derangement that is clearly about the pump, the heart. While some of the others can

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impair myocardial contractility there is however no myocardial damage in them, at least initially. It should also be noted that there are times when increased metabolic demands elicit changes in the determinants of SOT without affectation of the myocardial contractility and which if prolonged, may lead to normal or increased cardiac output that may also be insufficient to meet this increased demand. The mechanisms for these vary as VF, PR, HR or Hb may be involved singly or in combination [2,3]. These conditions are usually referred to as high output cardiac failure [3,4].

Though as noted, SOT can be variously affected directly by the factors on the right side of the equation other than myocardial contractility, the prolonged states however may also lead to ultimate myocardial failure if urgent steps to arrest the acute stages are not instituted. This is because of the non-salutary effect of the various compensatory mechanisms that are activated consequent on a defective SOT, and these include: the increase in sympathetic tone which is aimed to increase heart rate and myocardial contractility which when prolonged causes tachycardia and increase in peripheral vasoconstriction, the after load, with increase in oxygen consumption of the myocardium [3,4].

Clinical features

Clinical features of cardiac failure are age and disease process dependent [1,2]. The degree of manifestation of these features depends on the degree of the child's cardiac reserve [2]. A child who has exhausted his compensatory mechanism response will manifest symptoms at rest . This has been demonstrated by Ross, et al., [6]. In congestive cardiac failure, there is present, myocardial failure with an associated palpable cardiomegaly (demonstrable radiologically or by echocardiogram) and sometimes a gallop rhythm. These distinguish congestive cardiac failure from cardiac insufficiency where there is also a peripheral circulatory collapse but without other features as in myocardial failure.

The critical question is should these forms of heart failure syndromes be considered as cardiac failure when there is no clear clinical or radiological evidence of myocardial pump failure?

The manifestations of the compensatory mechanisms are usually important to the clinician in order to differentiate deranged SOTs from insufficient cardiac output (cardiac insufficiency) and those due to acute increase in tissue demand.

Due to the various causes (from the equation),these findings include tachycardia, signs of respiratory distress, diaphoresis, edema, irritability, orthopnea, and wheezing. When there is a myocardial contractility failure however, cardiomegaly, detectable clinically by displaced apex with audible heart sounds and often with a gallop, a raised and oscillating JVP with the cardiomegaly, confirmed radiologically or with echocardiography are invariably present in such patients, consequent on a compensatory dilatation and hypertrophy of the ventricles. This is the state that is often distinctly referred to as cardiac failure. However, in other such acute states of deranged SOT, cardiac insufficiency states, with activated compensatory mechanisms, cardiomegaly and other mentioned features are absent [3]. Expectantly, there will be neither a

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demonstrable displaced apex beat nor radiological increase in cardiothoracic ratio in cardiac insufficiency [6-8]. In other words, cardiac failure is a distinct entity that can be diagnosed but it should be borne in mind that, in itself, is also a cause of cardiac insufficiency where the other causes can be its differential diagnoses and it can also be caused by prolonged states of the other cardiac insufficiency causalities. Again, the states referred to as "high output cardiac failures" are also a consequence of considering states of inadequate SOTs as "cardiac failure". From the above, it is clear that these also do not meet the cardiac failure criteria and should correctly be seen as states of high output cardiac insufficiency. Furthermore, management of cardiac failure is usually aimed at maximizing cardiac output and this is uniquely done by the use of digitalis but in other situations of SOT derangement and activated compensatory mechanisms, there is no alteration of myocardial contractility, thus the use of digitalis is not key but other management modalities.

Conclusion

From above, it is obvious that the use of the term cardiac failure will be best reserved for clinical conditions where SOT is defective with evident resultant compensatory mechanisms activated and where there is raised and oscillating JVP if appreciated, proven cardiomegaly (clinical, radiological and by echocardiogram), possible presence of gallop rhythm and consideration for use of inotropes targeted at the heart muscles in managing them while cardiac insufficiency will be reserved for other situations where SOT with compensatory mechanisms are present but without the features as listed for cardiac failure or need for digitalis in treating them.

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