



A Brief Note on Pathophysiology of Heart Failure

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Description

A decrease in cardiac muscle efficiency owing to injury or stress is the core aetiology of heart failure. As a result, it can be caused by a variety of conditions, such as myocardial infarction, which occurs when the heart muscle is deprived of oxygen and dies, hypertension, which increases the force of contraction required to pump blood, and amyloidosis, which occurs when misfolded proteins are deposited in the heart muscle, causing it to stiffen.

The heart of a person with heart failure may have a reduced force of contraction due to the overloading of the ventricle. The higher filling of the ventricle results in increased contraction force and hence a rise in cardiac output in a healthy heart. This mechanism fails in heart failure because the ventricle is overloaded with blood, making heart muscle contractions less effective. This is due to the cardiac muscle's diminished capacity to cross-link actin and myosin filaments.

A reduced stroke volume could be caused by the inability of systole, diastole, or both. The most common cause of increased end systolic volume is decreased contractility. Impaired ventricular filling occurs when the ventricle's compliance declines, resulting in decreased end diastolic volume. The amount of cardiac output that might increase in times of elevated oxygen demand is reduced as the heart works harder to meet regular metabolic demands. This adds to the typical symptom of exercise intolerance in the cause of heart failure. This equates to a reduction in cardiac reserve, or the heart's ability to function harder during heavy exercise. The heart is incapable of satisfying the metabolic demands of the body during exercise because it has to work harder to meet normal metabolic demands.

An elevated heart rate is a common observation in people with heart failure, which is caused by increased sympathetic activity in attempt to maintain appropriate cardiac output. This initially helps to compensate for

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heart failure by maintaining blood pressure and perfusion, but also puts additional burden on the myocardial, increasing coronary perfusion requirements and potentially aggravating ischemic heart disease. It's possible that the muscle layer of the heart will grow in size. The overall consequence is decreased cardiac output and higher heart strain. This increases the risk of cardiac arrest and decreases blood flow to the rest of the body. Reduced cardiac output produces a variety of changes in the rest of the body in chronic disease, some of which are physiological compensations and others which are part of the disease process:

- The posterior pituitary secretes vasopressin in response to increased sympathetic activation, causing fluid retention in the kidneys. This causes blood volume and blood pressure to rise.
- Heart failure impairs the kidneys' ability to excrete sodium and water, causing edoema to worsen. Renin, an enzyme that catalyses the formation of the powerful vasopressor angiotensin, is released when blood flow to the kidneys is reduced. Angiotensin and its metabolites generate more vasoconstriction and stimulate the adrenal glands to secrete more of the steroid aldosterone.
- Chronically high levels of circulating neuroendocrine hormones such catecholamines, renin, angiotensin, and aldosterone have a significant impact on the myocardium, leading to long structural abnormalities in the heart. Many of these remodelling effects appear to be mediated by Transforming Growth Factor beta (TGF-beta), a common downstream target of the signal transduction cascade triggered by catecholamines and angiotensin II, as well as Epidermal Growth Factor (EGF), a target of the aldosterone-activated signalling pathway.
- Reduced perfusion of skeletal muscle induces muscle fibre atrophy. This can lead to weakness, fatiguability, and a loss of peak strength, all of which contribute to exercise intolerance.