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Blood pumping system working based on Cardiac Output

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Cardiac output (CO) is the amount of blood pumped by the heart minute and is the mechanism whereby blood flows around the body, especially providing blood flow to the brain and other vital organs. The body's demand for oxygen changes, such as during exercise, and the cardiac output is altered by modulating both heart rate (HR) and stroke volume (SV). As a result, the regulation of cardiac output is subject to a complex mechanism involving the autonomic nervous system, endocrine, and paracrine signalling pathways.

Because every tissue in the body relies on the heart pumping blood for nourishment, any cardiovascular dysfunction has the potential to result in significant morbidity and mortality [1]. Heart disease affects nearly 30 million Americans annually and is the number one cause of death in the United States [2]. The degree of functional impairment can be assessed by a variety of methods that guides diagnosis, prognosis, and treatment. As a clinician, you will come across heart disease in the course of their practice and should be familiar with the basics of cardiac function.

There are a number of clinical methods to measure cardiac output, ranging from direct intracardiac catheterization to non-invasive measurement of the arterial pulse. Each method has advantages and drawbacks. Relative comparison is limited by the absence of a widely accepted "gold standard" measurement. Cardiac output can also be affected significantly by the phase of respiration – intra-thoracic pressure changes influence diastolic filling and therefore cardiac output.

Cardiac output (CO) is the amount of blood pumped out by the heart in one minute. CO can be calculated using a simple equation: the stroke volume (SV) – the volume of blood pumped by the ventricles with each heart beat – multiplied by the heart rate [3]. First, one needs to calculate the SV – the difference between the EDV (the volume of blood left in the ventricles during diastole) and the ESV (the volume of blood remaining in the ventricles after it has contracted). The CO can vary; for example, it will increase in response to metabolic demands such as exercise or pregnancy. In pathological states such as heart failure, the CO may not be sufficient to support simple activities of daily living or to increase in response to demands such as mild-to-moderate exercise (Jarvis and Saman, 2017). The amount of preload determines the volume of blood that can leave the heart (the CO) and influences the stretch and tension on the individual muscle cells which make up the cardiac fibres. The SV increases in response to preload. As a result of the filling, increased pressure in the ventricles increases the stretching of the cardiac muscle fibres [4]. This stretch culminates in increased contractility of the heart and increased CO. Up to a certain physiological limit, the preload and contractility of the heart are positively correlated. This explains how exercise can improve cardiac performance.

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