Applied cardiovascular physiology

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Abstract

Maintaining an equilibrium between oxygen supply and demand is a principal function of the cardiovascular system. In times of altered metabolic demand mechanisms exist to maintain the balance between supply and demand. Exercise, haemorrhage and pregnancy all lead to changes in oxygen demand and subsequently modification of cardiac output. The main response to isotonic exercise is an increased cardiac output by autonomically mediated increases in heart rate and venous return. This allows delivery of increased oxygen concentrations to the exercising muscle. Cardiac output increases throughout pregnancy. In the first and second trimesters this rise is mainly due to an increase in stroke volume, however during the later stages of pregnancy stroke volume reaches a plateau and further increase in cardiac output is mediated by a rising heart rate. In contrast, during haemorrhage, decreased venous return leads to a reduction in cardiac output, with a baroreceptor response due to the drop in arterial blood pressure. The tachycardia and vasoconstriction which follows are compensatory mechanisms in an attempt to preserve blood pressure. The Valsalva manoeuvre illustrates several aspects of reflex control of the cardiovascular system and allows non-invasive assessment and quantification of control mechanisms. Changes in stroke volume during the respiratory cycle can be used to predict fluid responsiveness and can be measured as pulse pressure variation or stroke volume variation.

Keywords Autonomic; baroreceptor; cardiac output; exercise; haemorrhage; pregnancy; Valsalva

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Learning objectives

After reading this article you will be able to:

- explain the mechanisms controlling cardiac output during isotonic and isometric exercise
- describe the changes in cardiovascular physiology which occur during pregnancy
- discuss the cardiovascular response seen in acute haemorrhage
- contrast the physiological mechanisms used to increase cardiac output during haemorrhage and exercise
- explain the Valsalva manoeuvre and draw the physiological responses in heart rate and blood pressure corresponding to each phase
- understand how the heart and lungs interact during the respiratory cycle to change stroke volume and predict fluid responsiveness

The autonomic nervous system affects the cardiac output, primarily via heart rate modulation. At rest, sympathetic and parasympathetic nervous systems are in balance. In the parasympathetic nervous system the vagus nerve originates in the medulla and innervates the sinoatrial (SA) and atrioventricular (AV) nodes. Acetylcholine acts as the neurotransmitter. Parasympathetic stimulation leads to decreased activity of the SA and AV nodes, leading to a decreased heart rate. The sympathetic nerves arise from the spinal cord and innervate the SA node and ventricular muscle mass. The nerves release norepinephrine as the neurotransmitter. The response is an increase in heart rate and an increased force of contractility of the ventricles.

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Stroke volume (SV) is governed by end-diastolic volume (preload), afterload, and the strength of ventricular contraction. When end-diastolic volume (the preload) increases, the stroke volume increases. With this increased end-diastolic volume, a slight stretching of the cardiac muscle fibres also occurs, which increases the force of contraction.

Afterload is the left ventricular wall stress during ejection which in most circumstances is a measurement equivalent to the blood pressure (BP) in the aorta. The SV is inversely proportional to the aortic BP.

Exercise

During exercise there is an increased metabolic load in the exercising muscles. In order to maintain the balance between oxygen supply and demand, several physiological mechanisms exist and sympathetic activity predominates. Initially, in anticipation of increased physical activity, there is a cortically mediated 'central command' response. This causes activation of the sympathetic nervous system and decreased parasympathetic outflow leading to an increase in heart rate prior to commencement of exercise. The next phase of the cardiovascular response is dependent upon whether the exercise is isometric (static) or isotonic (dynamic). In isometric exercise, (when muscle fibre length remains static, such as during weight lifting) there is minimal change in stroke volume. Muscle contraction is sustained causing occlusion of its own arterial supply and consequently, a marked increase in peripheral vascular resistance. This increase in vascular resistance raises both systolic and diastolic arterial blood pressures. Because BP is a major determinant of afterload, the left ventricular wall stress, and thus the cardiac workload, is significantly higher during static exercise compared with the cardiac workload achieved during dynamic exercise. In comparison, during isotonic exercise, (when repeated shortening and lengthening of muscle occurs, for example in running or swimming) there is a marked increase in cardiac output via increases in heart rate and stroke volume (Figure 1). Activation of the muscle pump greatly enhances venous return to the heart, which compensates for the reduced

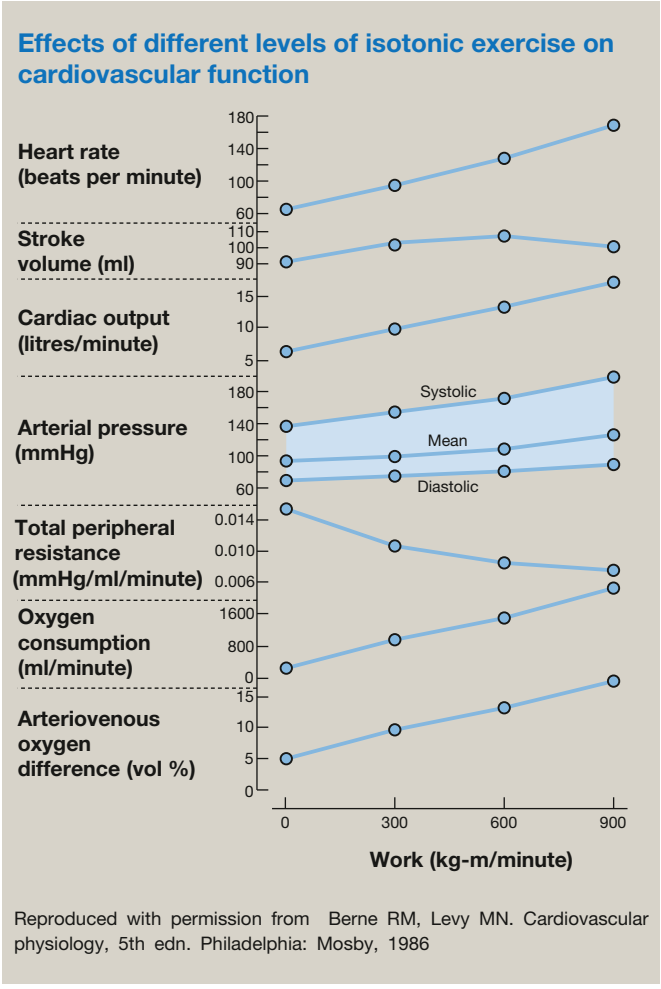

Figure 1

diastolic filling time resulting from the rise in heart rate. A decrease in oxygen concentration sensed by chemoreceptors located in the arch of the aorta and the carotid arteries and escalation of temperature leads to further sympathoactivation to allow increased oxygen delivery and reduction of internal core temperature. Epinephrine and norepinephrine released during exercise increase contractility via increased calcium influx through calcium channels in cardiac muscle membranes. This allows for greater myosin and actin interaction, increasing inotropy. During exercise, the afterload is reduced, which allows for an increase in SV and therefore increased cardiac output.

Active skeletal muscle releases locally vasoactive substances such as adenosine, K^+ and acid metabolites which cause vasodilatation, capillary recruitment and redistribution of blood flow. Blood flow to the exercising muscle may increase more than tenfold. The resultant reduction in muscle vascular resistance is offset by an increased sympathetic drive to other vascular beds. Splanchnic, renal and initially skin blood flow are all decreased. As exercise progresses, cutaneous blood flow increases in order to facilitate heat loss and then falls again as exhaustion is approached. Blood flow to the myocardium is also increased to match metabolic demands.[1] (Table 1).

Blood flow to organ systems during exercise and at rest

Organ system	Blood flow (ml/minute)	
	Exercise	Rest
Brain	750	750
Heart	750	750
Skeletal muscle	12,500	1200
Skin	1900	500
Abdominal viscera	600	1400
Kidneys	600	1100
Other	400	600
Total	17,500	5800

In trained athletes cardiac output can be increased up to seven times its baseline, but stroke volume rarely rises above twice its resting state. With endurance training, there can be an elevation in resting stroke volume and drop in resting heart rate, with increased left ventricular muscle mass. In addition, there is an increase in resting blood volume and red cell count to enhance cardiac reserve. These changes are similar to those mechanisms evident in pregnancy. From Pinnock. *Fundamentals of Anaesthesia.*

Table 1

Cardiopulmonary exercise testing

As workload increases, oxygen consumption (VO2) will eventually exceed oxygen supply. The point at which the rate of VO2 exceeds aerobic capacity is called the anaerobic threshold (AT). ATP required for continued exercise is then generated anaerobically producing an increase in lactic acid and subsequently CO_2 production which can be detected by cardiopulmonary exercise testing (CPET). CO_2 production increases in comparison to O_2 consumption and the respiratory exchange ratio exceeds 1.

Preoperative measurement of the AT has important clinical significance as there is elevated postoperative mortality risk following major surgery when AT is less than 11 ml/kg/minute.[2]

Pregnancy

By 5 weeks post-conception cardiac output increases. This occurs from a combination of increased SV, increased heart rate and a decrease in total peripheral resistance. Cardiac output is increased by approximately 40% at the end of the first trimester. Anatomically the heart is displaced upward and to the left by the gravid uterus. Flow murmurs are quite common due to increases in plasma volume and cardiac output.

The ECG reflects these changes with left axis deviation, ST segment depression and T wave flattening often seen.

A decrease in systemic vascular resistance (SVR) occurs due to the vasodilatory effects of progesterone and the proliferation of low resistance vascular beds in the inter-villous spaces of the placenta. Blood flow to the uterus increases to about 700 ml/minute by term. Blood flow to the kidneys and skin also increases, but the brain and liver blood flow remains constant.

Despite the increase in cardiac output, there is a fall in BP, due to the significant drop in SVR. Diastolic pressure may fall as much as 20% and systolic by around 8%.