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18/MHS01/149
MBBS
PHYSIOLOGY

1. DISCUSS THE LONG -TERM REGULATION OF MEAN ARTERIAL BLOODPRESSURE ?

Previous studies have shown that the blood pressure response to isometric handgrip remains unchanged during reductions in preload induced by lower body negative pressure (LBNP). The purpose of the present study was to assess the beat-by-beat haemodynamic mechanisms allowing for precise control of mean arterial pressure (MAP). We have followed the cardiovascular variables involved in the regulation of MAP during isometric handgrip with and without additional application of LBNP during defined periods of the ongoing contraction. Sixteen subjects participated. Mean arterial blood pressure (MAP), heart rate (HR), stroke volume (SV), cardiac output (CO), blood flow velocity in the brachial artery, acral skin blood flow, as well as total (TPR) and local (LPR) peripheral resistance were continuously recorded/calculated before, during and after 2 min of handgrip both with and without concomitant LBNP. The main finding was that MAP increased at the same rate and to the same absolute level whether or not LBNP was applied. A uniform increase in MAP was observed even though the cardiovascular variables evolved differently in the periods with and without LBNP. At the onset of LBNP at -20 mmHg, there was a transient drop in MAP and a transient increase in HR, but within seconds, MAP was regulated back to the slope caused by the isometric handgrip proper. CO and SV, which were declining gradually, showed an additional marked but gradual reduction upon LBNP application. At the same time, both LPR and TPR increased markedly and continuously. In summary, the increase in MAP during isometric handgrip remained essentially unchanged by LBNP-induced alterations in preload. The increase in MAP was caused by a marked increase in peripheral resistance. This supports the concept of a central set point, continuously regulated upwards as long as the isometric handgrip persists. Furthermore, it reveals a considerable flexibility in the cardiovascular control mechanisms used to achieve the desired arterial pressure.

2. A SHORT NOTE ON THE FOLLOWING
A. PULMONARY CIRCULATION
This is the portion of the circulatory system that carries deoxygenated blood away from the the right ventricle, to the lungs and carries oxygenated blood to the left atrium and ventricleof the heart
B. CIRCLE OF WILLIS
The Circle of Willis is the joining area of several arteries at the bottom (inferior) side of the brain. At the Circle of Willis, the internal carotid

arteries branch into smaller arteries that supply oxygenated blood to over 80% of the cerebrum.

C. SPLANCHNIC CIRCULATION

The splanchnic circulation consists of the blood supply to the gastrointestinal tract, liver, spleen, and pancreas. It consists of two large capillary beds partially in series. The small splanchnic arterial branches supply the capillary beds, and then the efferent venous blood flows into the PV.

D. CORONARY CIRCULATION

Coronary circulation is the circulation of blood in the blood vessels that supply the heart muscle (myocardium). Coronary arteries supply oxygenated blood to the heart muscle, and cardiac veins drain away the blood once it has been deoxygenated.

D. CUTANEOUS CIRCULATION

The cutaneous circulation is the circulation and blood supply of the skin. The skin is not a very metabolically active tissue and has relatively small energy requirements, so its blood supply is different to that of other tissues.

3. DISCUSS THE CARDIOVASCULAR ADJUSTMENT THAT OCCURS DURING EXERCISE

The cardiovascular system provides the link between pulmonary ventilation and oxygen usage at the cellular level. During exercise, efficient delivery of oxygen to working skeletal and cardiac muscles is vital for maintenance of ATP production by aerobic mechanisms. The equine cardiovascular response to increased demand for oxygen delivery during exercise contributes largely to the over 35-fold increases in oxygen uptake that occur during submaximal exercise. Cardiac output during exercise increases greatly owing to the relatively high heart rates that are achieved during exercise. Heart rate increases proportionately with workload until heart rates close to maximal are attained. It is remarkable that exercise heart rates six to seven times resting values are not associated with a fall in stroke volume, which is maintained by splenic contraction, increased venous return, and increased myocardial contractibility. Despite the great changes in cardiac output, increases in blood pressure during exercise are maintained within relatively smaller limits, as both pulmonary and systemic vascular resistance to blood flow is reduced. Redistribution of blood flow to the working muscles during exercise also contributes greatly to the efficient delivery of oxygen to sites of greatest need. Higher work rates and oxygen uptake at submaximal heart rates after training imply an adaptation due to training that enables more efficient oxygen delivery to working muscle. Such an adaptation could be in either blood flow or arteriovenous oxygen content difference. Cardiac output during submaximal exercise does not increase after training, but studies using high-speed treadmills and measurement of cardiac output at maximal heart rates may reveal improvements in maximal oxygen uptake due to increased stroke volumes, as occurs in humans. Improvements in hemoglobin concentrations in blood during exercise after training are recognized, but at maximal exercise, hypoxemia may reduce arterial

oxygen content. More effective redistribution of cardiac output to muscles by increased capillarization and more efficient oxygen diffusion to cells may also be an important means of increasing oxygen uptake after training.