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MATRIC NO: 18/MHS01/174

DEPARTMENT: MEDICINE AND SURGERY

PHYSIOLOGY ASSIGNMENT

1. Healthy State

   In healthy individuals, increases in ECF volume result in a proportional increase in the total blood volume. As described in our discussion of the vascular function curve, an increase in total blood volume will enhance the "Mean Systemic Pressure" which in turn increases the venous return and thus the cardiac preload. Courtesy of the Frank-Starling Relationship, increased preload on the heart will enhance the cardiac output. Finally, as discussed in systemic arterial pressure regulation, an increased cardiac output will boost the systemic arterial pressure so long as the SVR remains constant. In this way, an increase in ECF volume results in an increase in the arterial pressure; conversely, a decrease in ECF volume will yield a decline in arterial pressure.

Diseased State

In certain disease states, increases in ECF volume does not result in proportional increases in the total blood volume. This occurs in contexts of deranged Starling Forces in which fluid leaks out of the vasculature and thus does not contribute to the total blood volume but instead contributes to states of generalized edema, peripheral edema, or ascites. In such cases, the renal mechanisms of long-term arterial pressure regulation in fact exacerbate these edematous states. This occurs because the kidneys continue to resorb salt and water in an attempt to boost the systemic arterial pressure; however, instead of contributing to correcting the deficient arterial pressure, the additional fluid volume simply ends up in the interstitial fluid and thus aggravates the edema. Frequently, such edematous states are corrected by using diuretics, which modulate renal physiology in such a way that the kidneys excrete large volumes of salt and water, thus decreasing the ineffective additional ECF volume and correcting the edema

2. **Pulmonary circulation**, system of blood vessels that forms a closed circuit between the heart and the lungs, as distinguished from the systemic circulation between the heart and

all other body tissues. On the evolutionary cycle, pulmonary circulation first occurs in lungfishes and amphibians, the first animals to acquire a three-chambered heart. The pulmonary circulation becomes totally separate in crocodilians, birds, and mammals, when the ventricle is divided into two chambers, producing a four-chambered heart. In these forms the pulmonary circuit begins with the right ventricle, which pumps deoxygenated blood through the pulmonary artery. This artery divides above the heart into two branches, to the right and left lungs, where the arteries further subdivide into smaller and smaller branches until the capillaries in the pulmonary air sacs (alveoli) are reached. In the capillaries the blood takes up oxygen from the air breathed into the air sacs and releases carbon dioxide. It then flows into larger and larger vessels until the pulmonary veins (usually four in number, each serving a whole lobe of the lung) are reached. The pulmonary veins open into the left atrium of the heart.

B. The Circle of Willis is a structure located at the base of the brain (around eye level) encircling around the brainstem and the parts of the mid-brain, that provides a blood supply to the brain and neighboring structures.

More specifically, it’s a circulatory anastomosis (i.e., a connection between two blood vessels, such as between arteries, veins, or between an artery and a vein (arterio-venous anastomosis)) that encircles the stalk of the pituitary gland and allows distribution of blood to the brain and nearby structures.

C. The splanchnic circulation is composed of gastric, small intestinal, colonic, pancreatic, hepatic, and splenic circulations, arranged in parallel with one another. The three major arteries that supply the splanchnic organs, cellac and superior and inferior mesenteric, give rise to smaller arteries that anastomose extensively. The circulation of some splanchnic organs is complicated by the existence of an intramural circulation. Redistribution of total blood flow between intramural vascular circuits may be as important as total blood flow. Numerous extrinsic and intrinsic factors influence the splanchnic circulation. Extrinsic factors include general hemodynamic conditions of the cardiovascular system, autonomic nervous system, and circulating neurohumoral agents. Intrinsic mechanisms include special properties of the vasculature, local metabolites, intrinsic nerves, paracrine substances, and local hormones. The existence of a multiplicity of regulatory mechanisms provides overlapping controls and restricts radical changes in tissue perfusion.

D. Coronary circulation, part of the systemic circulatory system that supplies blood to and provides drainage from the tissues of the heart. In the human heart, two coronary arteries arise from the aorta just beyond the semilunar valves; during diastole, the increased aortic pressure above the valves forces blood into the coronary arteries and thence into the musculature of the heart. Deoxygenated blood is returned to the chambers of the heart via coronary veins; most of these converge to form the coronary venous sinus, which drains into the right atrium. The heart normally extracts 70 to 75 percent of the available oxygen from the blood in coronary circulation, which is much more than the amount extracted by other organs from their circulations—e.g., 40 percent by resting skeletal muscle and 20 percent by the liver. Obstruction of a coronary artery, depriving the heart tissue of oxygen-rich blood, leads to death of part of the heart muscle (myocardial infarction) in severe cases, and total heart failure and death may ensue.

E. 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. Some of the circulating blood volume in the skin will flow through will flow through arteriovenous anastomoses (AVAs) instead of capillaries. AVAs serve a role in temperature regulation. In this article we shall consider the different adaptations of the cutaneous circulation, and its role in body temperature control.

3. The integrated response to severe exercise involves fourfold to fivefold increases in cardiac output, which are due primarily to increases in cardiac rate and to a lesser extent to augmentation of stroke volume. The increase in stroke volume is partly due to an increase in end-diastolic cardiac size (Frank-Starling mechanism) and secondarily due to a reduction in end-systolic cardiac size. The full role of the Frank-Starling mechanism is masked by the concomitant tachycardia. The reduction in end-systolic dimensions can be related to increased contractility, mediated by beta adrenergic stimulation. Beta adrenergic blockade prevents the inotropic response, the decrease in end-systolic dimensions, and approximately 50% of the tachycardia of exercise. The enhanced cardiac output is distributed preferentially to the exercising muscles including the heart. Blood flow to the heart increases fourfold to fivefold as well, mainly reflecting the augmented metabolic

requirements of the myocardium due to near maximal increases in cardiac rate and contractility. Blood flow to the inactive viscera (e.g., kidney and gastrointestinal tract) is maintained during severe exercise in the normal dog. It is suggested that local auto regulatory mechanisms are responsible for maintained visceral flow in the face of neural and hormonal autonomic drive, which acts to constrict renal and mesenteric vessels and to reduce blood flow. However, in the presence of circulatory impairment, where oxygen delivery to the exercising muscles is impaired as occurs to complete heart block where normal heart rate increases during exercise are prevented, or in congestive right heart failure, where normal stroke volume increases during exercise are impaired, or in the presence of severe anemia, where oxygen-carrying capacity of the blood is limited, visceral blood flows are reduced drastically and blood is diverted to the exercising musculature. Thus, visceral flow is normally maintained during severe exercise as long as all other compensatory mechanisms remain intact. However, when any other compensatory mechanism is disrupted (even the elimination of splenic reserve in the dog), reduction and diversion of visceral flow occur.