1. **Renal Volume Regulation**

The kidneys respond to changes in systemic arterial pressure by modifying their urinary excretion of sodium and water. When arterial pressures are elevated, renal urinary excretion of sodium and water increases; conversely, when arterial pressures are deficient, renal urinary excretion of sodium and water decreases. The mechanisms which connect changes in arterial pressures to renal urinary excretion of salt and water are described more fully in ECF volume regulation and principally rely on mechanisms pressure natriuresis and the RAAS System. Nevertheless, this relationship between systemic arterial pressures and renal urinary excretion is largely independent of the SVR; consequently, whether or not the SVR is high or low, the kidneys will respond as described above by matching their urinary excretion to the effective systemic arterial pressure.

The capacity of the kidneys to regulate urinary salt and water excretion allows these organs to regulate the total ECF volume which, as discussed below, is a major determinant of systemic arterial pressure in healthy individuals. Taken together, the relationship between arterial pressure, renal salt and water excretion, and ECF volume resembles a negative feedback control circuit in which changes to arterial pressure modulate renal sodium and water excretion which in turn affect ECF volume and thus modulate arterial pressure. Once again, this negative feedback appears to act completely independently of the SVR and explains why changes to the SVR can only affect arterial pressures transiently.

While a rapid increase in SVR will immediately boost the blood pressure, the kidneys will respond by progressively excreting salt and water, thus reducing the ECF volume and thus causing a slow decline in arterial pressure. Conversely, while a rapid decrease in SVR will immediately reduce the blood pressure, the kidneys will respond by retaining more salt and water than that ingested, thus increasing the ECF volume and thus causing a slow increase in the arterial pressure.

ECF Volume and Arterial Pressure

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.

◦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. A. **Pulmonary Circulation**

Pulmonary circulation is the system of transportation that shunts de-oxygenated blood from the heart to the lungs to be re-saturated with oxygen before being dispersed into systemic circulation. Deoxygenated blood from the lower half of the body enters the heart from the inferior vena cava while deoxygenated blood from the upper body is delivered to the heart via the superior vena cava. Both the superior vena cava and inferior vena cava empty blood into the right atrium. Blood flows through the tricuspid valve into the right ventricle. It then flows through the pulmonic valve into the pulmonary artery before being delivered to the lungs. While in the lungs, blood diverges into the numerous pulmonary capillaries where it releases carbon dioxide and is replenished with oxygen. Once fully saturated with oxygen, the blood is transported via the pulmonary vein into the left atrium which pumps blood through the mitral valve and into the left ventricle. With a powerful contraction, the left ventricle expels oxygen-rich blood through the aortic valve and into the aorta: This is the beginning of systemic circulation.

B. **Circle of Willis**

The blood supply to the brain divides into an anterior and posterior circulation. The anterior circulation derives blood from the bilateral internal carotid arteries (ICA) and supplies blood to the majority of the cerebral hemispheres, including the frontal lobes, parietal lobes, lateral temporal lobes and anterior part of deep cerebral hemispheres. The posterior circulation derives blood from the bilateral vertebral arteries (VA). It supplies the brainstem, cerebellum, occipital lobes, medial temporal lobes and posterior part of the deep hemisphere, mainly the thalamus. The circle of Willis (CoW) is an anatomical structure that provides an anastomotic connection between the anterior and posterior circulations, providing collateral flow to affected brain regions in the event of arterial incompetency.

C**. Splanchnic Circulation**

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**

The heart is highly metabolically active and boasts the highest oxygen consumption by mass of any organ. This demand for oxygen is met by the coronary circulation, which is responsible for delivering blood to the myocardium and represents approximately 5% of cardiac output.[1] Adequate blood flow

through the coronary vessels is critical to avoid ischemia and maintain the integrity of the myocardial tissue.

E. **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. 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. Major changes are made during exercise which are;

I. Increase in heart rate, stroke volume, cardiac output because of withdrawal of parasympathetic stimuli and increased input from sympathetic nerves.
II. Temperature increases, feedback from proprioceptors and accumulation of metabolites

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 autoregulatory 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.