Types of Shock

Part Two in Our Series on Shock

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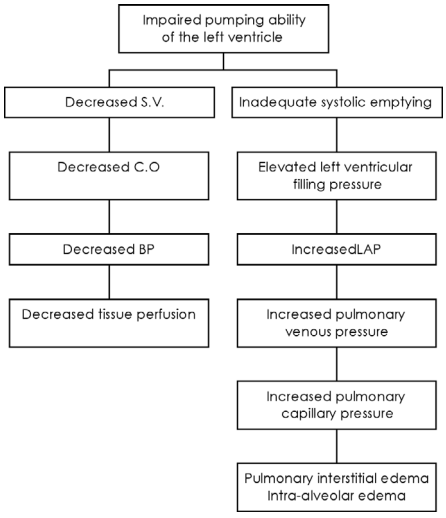
Introduction

Failure of the normal regulatory mechanisms in the body can lead to rapid and profound shock. This article reviews the pathophysiology of cardiogenic, hypovolemic, anaphylactic, and neurogenic shock. A brief review of sepsis and septic shock is also included.

Cardiogenic Shock

Cardiogenic shock is caused by inadequate myocardial contractility from acute myocardial infarction, coronary artery disease, or mechanical factors (valvular regurgitation, low output syndrome, arrhythmias).

Pathophysiology of Cardiogenic Shock



In cardiogenic shock, the left ventricle has been injured in some way, leading to impaired pumping.

Because the pumping is ineffective, less blood is pushed out with each heartbeat, leading to a decreased stroke volume*. The heart rate increases to compensate for a low cardiac output and blood pressure, but will eventually be insufficient to compensate for the decreased stroke volume. The tissues begin to be inadequately perfused.
The impaired pumping also leads to less blood being pushed from the ventricle during systole. The left ventricle gradually fills with more and more blood, causing an elevated pressure within the LV and left atrium. This pressure "backs up" into the pulmonary vasculature, causing an increased pulmonary capillary pressure.

* Stroke volume = the amount of blood pumped out of the left ventricle with each contraction.

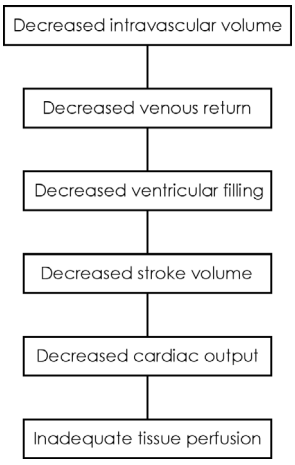
Hypovolemic Shock

(most common), plasma loss due to burns, dehydration, traumatic shock due to blood loss and major tissue damage.

Pathophysiology of Hypovolemic Shock

The pathophysiologic process of hypovolemic shock is straight-forward. Blood and/or fluids have left the body, causing a decreased amount of volume in the blood vessels.

Venous return is decreased because of the lack of fluid in the vascular space, causing decreased ventricular filling. The ventricles do not have as much blood as normal to pump out, so the stroke volume is decreased.



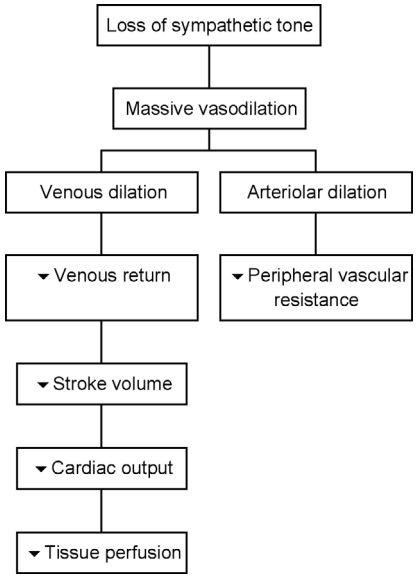
The heart rate will increase to compensate for the diminished stroke volume and resulting poor cardiac output and blood pressure. Eventually, if the fluid or blood loss continues, the heart rate will not be able to compensate for the decreased stroke volume.

The end result of hypovolemic shock is inadequate tissue perfusion.

Neurogenic Shock

Neurogenic shock is caused by the loss of sympathetic control (tone) of resistance vessels, resulting in the massive dilatation of arterioles and venules. Neurogenic shock can be caused by general or spinal anesthesia, spinal cord injury, pain, and anxiety.

Pathophysiology of Neurogenic Shock



In neurogenic shock, there has been an insult to the nervous system so that impulses from the sympathetic nervous system (the fight or flight response) cannot maintain normal vascular tone or stimulate vasoconstriction.

The lack of SNS stimulation causes a massive venous and arterial vasodilation. On the venous side, blood pools in the distensible veins and does not return to the larger veins. Because of this pooling, there is a diminished amount of blood that returns to the heart. Stroke volume, cardiac output, and blood pressure all fall.
On the arterial side, there is decreased peripheral vascular resistance, which actually helps the heart to pump with less energy. The drawback is that with decreased peripheral pressure, there is not the driving force to get blood, oxygen, and nutrients to the cells. This also causes a small degree of arterial blood pooling, which decreases the amount of blood returning to the heart.

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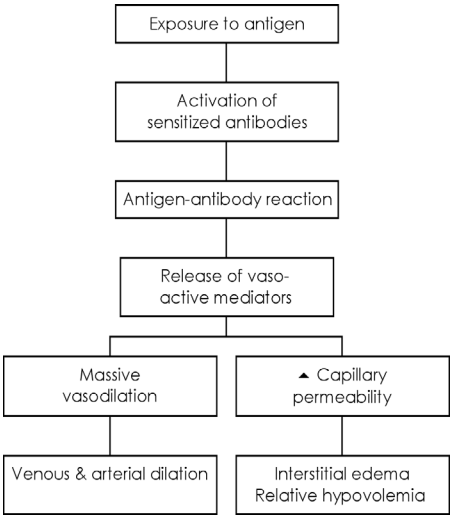
Anaphylactic Shock

Shock due to the severe allergic antigen-antibody reaction to substances such as drugs, contrast media, blood products, or insect or animal venom is called anaphylactic shock. Food products such as seafood, nuts, peanuts, peanut butter, and MSG can also cause anaphylactic shock.

Pathophysiology of Anaphylactic Shock



The immune system goes "haywire" in anaphylactic shock in an extreme allergic reaction. At some point, the individual is exposed to the substance and develops antibodies against it. On subsequent exposure to the substance (the antigen), these antibodies will aggressively bind to the antigen, forming an antigen-antibody complex. This complex causes the release of chemicals that cause vasodilation (in particular, histamine).

Both veins and arteries vasodilate, leading to decreased blood returning to the heart. The capillaries become permeable to nearly everything, allowing fluids, proteins, and sometimes blood to pass through into the interstitial space. This causes massive interstitial edema. The vasodilation and fluid sequestration in the interstitium causes a relative hypovolemia.

Septic Shock

Sepsis is a condition that occurs in many critically ill patients. Sepsis is the systemic response to infection. Many types of organisms can cause sepsis, including gram-negative bacteria, gram-positive bacteria, and fungi. The infections can occur anywhere in the body; urinary tract infections are probably the most common cause of sepsis. Septic shock is said to occur when the sepsis has progressed to the point where it is affecting many organ systems.

Pathophysiology of Septic Shock

The immune and inflammatory response begins to try to combat the organism that is causing an infection. The body releases multiple chemicals into the blood stream, including cytokines, vasodilators, complement factors, and free radicals. In septic shock, this response is not adequate to eliminate the infection and actually causes increased damage. The organism itself also releases substances called endotoxins or exotoxins, which further harm the organs and tissues.

The combination of these chemicals and toxins cause: (1) peripheral vasodilation – interstitial edema and decreased blood return to the heart, and (2) decreased ability of the cells and tissues to take up oxygen and nutrients.

The heart tries harder and harder to get oxygen and nutrients to the cells by increasing the heart rate and contractility initially, sometimes driving the cardiac output twice to three times its normal amount.

Eventually, however, the immune response and compensatory mechanisms may not enough to combat the infection and resulting cellular destruction. The patient may develop multi-organ dysfunction syndrome (MODS); AKA multi-system organ failure (MSOF).

Conclusion

Patients with a wide variety of problems can develop shock. Understanding the pathophysiology may help you anticipate problems, and assess and manage the care of the patient with shock. Most significantly, early recognition and intervention in shock may help to improve the patient's outcome.

Some of the material in this article was excerpted from the Shock and Infection in Critical Care Primer offered by TCHP. The TCHP Education Consortium offers homestudy education on a variety of health care topics on their website at www.tchpeducation.com. Homestudies are available to read free of charge on their website. If contact hours are desired, the processing cost is $5 per contact hour.

References

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