

Physiological Compensation and Emergency Management of Hemorrhagic Shock

Posted on October 29, 2019

The motorcyclist suffered from hemorrhage shock following the occurrence of the accident. Generally, hemorrhage shock refers to a life-threatening condition that results from excessive loss of blood and body fluids. The severe loss of fluid affects the normal functioning of the body in that the heart cannot transfer the necessary amount of blood around the body. This, in turn, poses a life-threatening condition for the patient and, if not quickly corrected, could lead to death. Severe blood loss from the body can result from bleeding wounds and cuts, bleeding of the digestive tract, significant vaginal bleeding as well as internal bleeding- just to mention a few. This type of shock is more common to young children, old people and people involved in severe accidents. It also causes many body organs, such as kidneys, to stop functioning effectively. The major Symptoms of hemorrhage shock include but are not limited to -low blood pressure, unconsciousness, rapid pulse rate, body weakness, minimal urine output and a decrease in the overall body temperature.

In addition to the actual blood loss, severe body fluids transpire to the reduction in the volume of blood, which in turn causes nausea, dizziness, increased sweating, excessive vomiting and diarrhea to the victim. These symptoms may not appear immediately but begin to show when the shock progresses. When severe bleeding occurs, the available blood is not enough for the heart to pump. In this essence, various body parts do not access the essential nutrients and substances hence causing them to begin shutting down.

When blood is lost from the body, the mean pulse and arterial pressures fall. The rate of heartbeat increases in relevance to the amount of blood lost by the victim. When the hemorrhage shock is stopped, the pressure of the arteries slowly recovers as the rate of heartbeat declines because the long-term compensatory mechanisms are triggered to fix normal arterial pressure. Administration of body fluids can fasten the quick recovery from the shock.

Therefore, the motorists' low blood pressure was due to heart failure caused by the low blood volume and perfusion of vital body organs. Additionally, the low blood pressure led to low blood flow within the body organs and, in turn, caused kidney failure, which is directly linked to the minimal urine released from the kidneys.

Compensatory Mechanisms

The motorist's blood vessels had been injured in the event of the accident, leading to increased blood loss. Generally, the decline in blood volume during acute blood loss leads to a reduction in cardiac filling and central venous pressure. This causes a decline in arterial pressure and cardiac output.

However, the body incorporates several compensatory mechanisms that are activated to bring back the blood volume and arterial pressure to normal. These include mechanisms such as the baroreceptor reflexes, which immediately detect any slight changes in blood pressure. The baroreceptors then trigger the sympathetic adrenal system to fasten the heart rate and contractility. The activation of the sympathetic adrenergic system also influences the coronary blood vessels and the heart, leading to an increase in arterial pressure and systemic vascular resistance. In other words, cardiac output is redistributed from other body organs and into the brain and myocardium which help to promote healing.

The combined effects of sympathetic activation and arterial hypotension stimulate the humeral compensatory mechanism. Sympathetic activation of the adrenal glands triggers the release of catecholamine which strengthens the sympathetic activation effects on vasculature and the heart. The kidneys release more renin, which causes an increase in the circulation of aldosterone. The sympathetic activation also stimulates the release of vasopressin and renal water and sodium reabsorption to increase the volume of blood. This renal mechanism is vital in ensuring long-term recovery from the loss of blood.

Bibliography

Blatt, S. J., & Bers, S. J. (2010). The sense of self in depression: A psychodynamic perspective.

Castonguay, L. G., & Hill, C. E. (2012). *Transformation in psychotherapy: Corrective experiences across cognitive-behavioural, humanistic, and psychodynamic approaches*. American Psychological Association.

Decety, J., & Lamm, C. (2009). Empathy versus personal distress: Recent evidence from social neuroscience. *The social neuroscience of empathy*, 199-213.

Eisenberg, N., & Eggum, N. D. (2009). Empathic responding: Sympathy and personal distress. *The social neuroscience of empathy*, 6, 71-83.

Lewis, M. A., & Rook, K. S. (1999). Social control in personal relationships: Impact on health behaviours and psychological distress. *Health Psychology*, 18(1), 63.

Longres, J. F. (2000). *Human behaviour in the social environment*. Brooks/Cole Publishing Company.

Nolen-Hoeksema, S., & Rector, N. A. (2015). *Abnormal psychology*. Boston: McGraw-Hill.

Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (2012). *A developmental perspective on antisocial behaviour* (Vol. 44, No. 2, p. 329). US: American Psychological Association.

Sarason, I. G., & Sarason, B. R. (2009). *Abnormal psychology*. New York: Appleton-Century-Crofts.

Sue, D., Sue, D. W., Sue, S., & Sue, D. M. (2015). *Understanding abnormal behaviour*. Cengage Learning.

Ullmann, L. P., & Krasner, L. (2015). *A psychological approach to abnormal behaviour*. Prentice-Hall.

Kohlberg, L., 1969. *Stage and sequence: The cognitive-developmental approach to socialization*. Rand McNally.

Maccoby, E.E. and Martin, J.A., 1983. Socialization in the context of the family: Parent-child interaction. *Handbook of Child Psychology: formerly Carmichael's Manual of Child Psychology*/Paul H. Mussen, editor.