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Shock: Pathophysiology, Stage, Classification, and Treatment

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Abstract: Shock is a state of resulting from an indented state of multiple important body works owing to decreased tissue perfusion. It is a circumstance that could menace life. Progressive stage of shock means failing compensatory mechanisms and the body's compensatory mechanisms weaken to vindicate tissue perfusion. Hypovolemic shock is a circumstance of insufficient organ perfusion caused by destruction of intravascular volume, ordinarily acute. Distributive shock is a state of comparative hypovolemia sequencing from affiliated to pathology spread to other areas of the perfect intravascular volume and is more common form of shock. Dextran has considerable merits over distinctive types of colloids for the initial shock treatment owing to its antithrombotic properties whereby cell satisfactory is obviated and the occurrence of systematic circulations is decisively de-escalated. Drugs that de-escalate the hearts workload and pain such as morphine or fentanyl relieve anxiety or regulate heart rhythm. Vasopressor medications such as dopamine, norepinephrine, which are medications that constrict blood vessels and assists, escalate blood pressure. High amount of IV fluids very quickly and bestowing medications that will escalate the blood pressure to escalate blood flow to the rest of the body and organs.

Keywords: Classifications, Pathophysiology, Shock, Stages, Treatment

1. Introduction

Shock is a state of resulting from a recessed state of multiplex crucial body works owing to decreased tissue perfusion. It directs to de-escalate the delivering of supply with oxygen to the blood to the body's organs and tissues or functionally defective oxygen (O_2) usage by peripheral tissues, sequencing in end-organ malfunction [1]. Shock is usually, but not always, associated with systemic arterial hypotension; i.e., systolic blood pressure less than 90 mm Hg. Pressure is the product of flow and resistance [mean arterial pressure (MAP)=CO * systemic vascular resistance (SVR)]. Blood pressure may not fall if there is increase in peripheral vascular resistance in the presence of decreased cardiac output, resulting in inadequate flow to the tissue or global tissue hypoperfusion. The insensitivity of blood pressure to detect global tissue hypoperfusion has been repeatedly confirmed [1]. Thus, shock may occur with a normal blood pressure, and hypotension may occur without shock. Shock represents progression of a cascade of events that begins when cells or tissues are deprived of an adequate energy source because of oxygen deprivation. Shock occurs as a result of inadequate tissue perfusion; the lack of an adequate energy supply leads to the buildup of waste products and failure of energy-dependent functions, release of cellular enzymes, and accumulation of calcium and reactive oxygen species (ROS) resulting in cellular injury and ultimately cellular death. Activation of the inflammatory, coagulation, and complement cascades results in further cellular injury and microvascular thrombosis [2]. The amplification of these processes coupled with increased absorption of endotoxin and bacteria (as a result of liver and gastrointestinal dysfunction) leads to the systemic inflammatory response syndrome (SIRS) (see Chapter 2), multiple organ dysfunction (MOD), and if uncontrolled, ultimately death. Changes in preload, stroke volume, system vascular resistance, and cardiac output can result in impaired tissue and organ perfusion. The impaired delivery of oxygen to peripheral cells that occurs in shock results in a transition from aerobic to anaerobic cellular metabolism [1]. Anaerobic metabolism generates lactate via metabolism of glucose to pyruvate, and lactate can be used as a surrogate marker for tissue hypoxemia and the severity of shock. Cells can engage

in anaerobic metabolism for a limited time, but persistent cellular hypoxia results in cell death and tissue necrosis, leading to multiorgan system dysfunction and failure. The saturation of venous oxygen measured from central vessels (such as the superior vena cava), is another biochemical marker of peripheral oxygen uptake and can be used diagnostically to help with prognosis in the comprehensive assessment of patients presenting in shock [1]. This study is intended to recapitulate the background, pathophysiology, stage, classifications and treatment of shock.

2. Literature Review

Shock is defined as circulatory insufficiency that creates an imbalance between tissue oxygen supply and oxygen demand. The result of shock is global tissue hypoperfusion and is associated with a decreased venous oxygen content and metabolic acidosis (lactic acidosis).

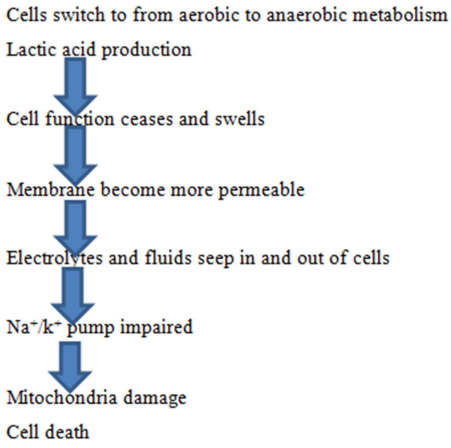

Figure 1. *Pathogenesis of shock.*

2.1. Pathogenesis of Shock

Inducing vasoconstriction reaction will be different among organ systems, with the highest reaction happening in the internal organ of the body, enveloping layer of the body, and renal. When O_2 furnish is inadequate to encounter requisition, the initial compensatory mechanism is escalating in cardiac output. If the escalate in cardiac output is scarceness, the quanta of O_2 removed from Hg by the tissues escalates, which de-escalates mixed venous oxygen (O_2) saturation. In failure of compensatory response there is de-escalated blood circulation to the tissues antecedents cellular in which scarceness of oxygen reaching the tissues of the body, metabolism which induced by anaerobes commences, cell bumping, mitochondrial impairment, and contingent cell death, and finally if less diffusion states persist irredeemable death perhaps happening very soon (figure 1).

As shock consecution, lysosomal enzymes are delivered into the cells with subsequent chemical breakdown of a compound owing to reaction with water of membranes, DNA, RNA, and phosphate esters. As the cataract of shock persists, the ruin of cellular adherence and the disintegration of cellular homeostasis sequence in cellular death. These relating to pathology circumstances bestow upgrade based on metabolism attributions of increased concentration of cells and solids in the blood, abnormal escalate of potassium level in the blood, abnormal de-escalate of sodium level in the blood, abnormal high level of nitrogen waste products in the blood, hyperglycemia or abnormal de-escalate of sugar in the blood, and lactic acid build up in the bloodstream. Lactic acidosis happens because of destruction in tissue O_2 usage, as in septic shock and post resuscitation from cardiac arrest; ordinary mixed venous oxygen saturation with an accelerated lactate delineates such destruction. Escalated lactate is a label damaged by O_2 release and/or usage and correlate with interim forecast of hypercritically sick long sufferings in the emergency department. Mixed venous oxygen saturation can also be utilized as a scale of the equilibrium among tissue O_2 furnish and requisition. Decrease renal flow; delivers renin angiotensin I; and angiotensin II potent vasoconstriction & delivers aldosterone adrenal cortex, and initially antecedent the state of being retained sodium & water, and then toxic substances enter the circulation; capability of endothelium perhaps damaged, and finally destruction, malfunction, and also there is unexpected cell death [2] (figure 2).

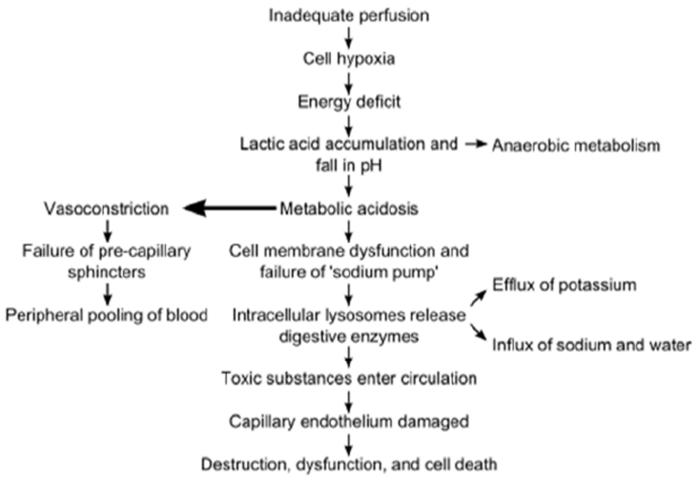

Figure 2. *Effects of insufficient perfusion on cell function.*

2.2. Stages of Shock

There 4 stage of shock [3, 4]: Primary stage: Tissue are beneath perfused, de-escalated carbon dioxide, escalated anaerobic metabolism, lactic acid is building. Compensated: means reversible. The body's compensatory mechanisms are capable to vindicate certain level of tissue perfusion. Progressive: means dereliction compensatory mechanisms and the body's compensatory mechanisms failing to maintain tissue perfusion. Irreversible: means refractory. Tissue and cellular affliction is so ponderous that the organism dies even if perfusion is renewed.

2.3. Classification of Shock

Currently shock is classified into 4 main classes, enclosing; hypovolemic shock; distributive shock; cardiogenic shock; obstructive shock [5];

Table 1. *Categories and causes of shock.*

	Hypovolemic shock	Obstructive shock	Distributive shock Septic shock Neurogenic shock Anaphylactic shock	Cardiogenic shock
Hemodynamics	decr preload incr SVR decr CO	decr preload incr SVR decr CO	decr preload decr SVR incr/decr CO	incr preload incr SVR decr CO
Causes	Hemorrhage, GI losses, third spacing, burns	Pulmonary embolism, tension pneumothorax, pericardial tamponade	Sepsis, anaphylaxis, neurogenic shock, pancreatitis	Myocardial infarction, symptomatic bradycardia, valvular disease, heart blocks, end-stage heart failure

2.3.1. Hypovolemic Shock

Hypovolemic shock is a circumstance of insufficient organ circulation caused by destruction of intravascular volume, ordinarily very serious. The sequence is a drip in cardiac preload to a hypercritical degree and decreased macro-and microcirculation, with interrogative outcomes for tissue metabolism and the triggering of an inflammatory reaction. In hypovolemic shock there is decrement of blood pressure; decrement of venous return; decrement of cardiac output; decrement of stroke volume; decrement of tissue perfusion, and finally cause hypovolemic shock [5, 6] (figure 3).

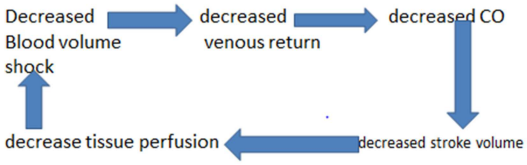

Figure 3. *Pathogenesis of hypovolemic shock.*

Hypovolemic shock is classified into 4 subclasses; (1) hemorrhagic shock, sequencing from dangerous hemorrhage without considerable soft tissue affliction. (2) Traumatic hemorrhagic shock, sequencing from very serious hemorrhages with soft tissue detriment and, furthermore, delivery of immune system initiators. (4) Hypovolemic shock in the needlelike sensation, sequencing from a hypercritical decrement in perfusing plasma volume without dangerous hemorrhage. (3) Traumatic hypovolemic shock, sequencing from a hypercritical suppression in perfusing plasma volume without serious hemorrhage, owing to soft tissue affliction and the delivery of immune system conciliators [5, 7]. Treatment: The preclinical and clinical management of hypovolemic shock contains of instantaneous intravascular volume substitution with equilibrium crystalloids using broad-bore peripheral venous access and, in a long suffering that is hemorrhaging, hast bleeding restrain [5]; supplemental O_2; supine position. Colloids: Frequently administered colloids enclose derivatives of human plasma (albumin), and semisynthetic colloids starches, gelatins, and dextrans). Dextran has considerable merits over distinctive types of colloids for the initial shock treatment owing to its antithromboitic properties whereby cell satisfactory is obviated and the occurrence of systematic circulations is decisively de-escalated. Colloids are solutions consisting high molecules that, because of their size and charge, are consequentially continued interior the vascular space. Because colloid accumulations are greater in the intravascular space, they use an oncotic pressure that resists the hydrostatic pressure and assists keep H_2O in or make it into the intravascular space. Both synthetic and natural colloids are applicable. Natural colloids involve plasma, whole blood, and bovine albumin. The merit of natural colloids is that they furnish protein such as albumin, antibodies, hypercritical clotting factors, antithrombin-3, and distinctive plasma components. Because FFP must be melted prior to be infusing, it is frequently not applicable in an emergency circumstance where important now fluid treatment perhaps displayed. Treatment determines near constraining destruction of fluid and blood displacing what is been missing, and stabilizing affliction that both antecedent and sequenced from hypovemic shock such as blood plasma transfusing, platelet transfusing, red blood cell transfusing, IV crystalloids. Whole blood is the optimal resuscitation fluid for patients who are severely bleeding and also used to treat patients who necessitate entire the constituents of blood, such as those who have sustained substantial blood loss owing to trauma or surgery. Packed RBCs to obviate tissue

hypoxia and also hypovolemic shock necessitates enough RBCs and H_2O in the blood for the heart to propel the fluids near interior the blood vessels. Isotonic crystalloids: Marketable applicable isotonic crystalloids (equated electrolyte solutions for high animal medicines are proposed to be displacement fluids, not maintenance fluids, expressing that the electrolyte component is proposed to exclude comparatively the electrolyte component of the extracellular fluid and not the quotidian displacement seeks. The ubiquitous balanced electrolyte supplementation is applicable for horses involve RL solution, Plasma-Lyte, and Normosol-R and are substantially possessed of sodium and chloride with diversifying quanta of calcium potassium, and magnesium. Physiologic NS (0.9% NaCl) distinguishes in that it consist solely sodium and chloride but no distinctive electrolytes. Vasopressin is an encouraging optional to epinephrine for resuscitation in cardiac arrest casualty, and make hemodynamic stable in septic shock long sufferings. Furthermore, vasopressin seems to be a separately effective vasopressor in the irredeemable phase of hemorrhagic shock unresponsive to volume displacement and catecholamine vasopressors. Vasopressin bestowed amid cardiac arrest and hemorrhagic shock perhaps ameliorates essential organ blood stream amid CPR, and stabilizes cardiocirculatory work after successful resuscitation. Medication that escalate the heart's pumping power to ameliorate circulation and get blood where it's necessitated such as dopamine, dobutamine, epinephrine, norepinephrine [8].

2.3.2. Distributive Shock

Distributive shock is a state of comparative hypovolemia sequencing from affiliated to pathology spread to other areas of the perfect intravascular volume and is more common form of shock. The cause is either a loss of regulation of vascular tone, with volume being shifted within the vascular system, and/or disordered permeability of the vascular system with shifting of intravascular volume into the interstitium. The three subclasses os distributive shock are septic, anaphylactic/ anaphylactoid, and neurogenic shock [5, 9]. (1a) Septic shock: Sepsis is delineated comprehending to the recent Sepsis-3 standard as a dysregulated reaction by the body to infection sequencing in lethal organ malfunctions. These are expressed and calculated by escalated in sequential organ failure assessment score by $\geq$ 2 points [5, 10]. Treatment: Airway and ventilator management; supplemental O_2. Vasopressor medications such as dopamine, norepinephrine, which are medications that constrict blood vessels and assists escalate BP; Insulin for blood sugar stability; Corticosteroid's: is category of medications down regulates the profuse and malfunction pro-inflammatory reaction, confines the anti-inflammatory reaction while at the identical time conserving innate immunity; IV fluids (crystalloids) will likely be administered to treat dehydration and assist escalate BP and blood flow to the organs; Empiric antibiotics: are consummately bestowed empirically during the time until the determination of the active microorganism; Sodium bicarbonate is used to treat lactic acidosis and it increase the strong ion gap caused owing to shock; Anti-endotoxin antibodies to treat septic shock caused initially by endotoxins released during solemn gram-negative bacterial infections and carries a staggering 30-50% mortality rate [11]. (2b) Anaphylactic and anaphylactoid shock: is described by high histamine-mediated vasodilation and faulty distribution with a transpose of fluid from the intravascular to the extravascular space [5, 12]. Treatment: Airway (has low threshold for inopportunely introduction of a tube into a hollow organ); supplemental O_2 and ventilation; beta agonists (Epinephrine, dopamine, dobutamine) (IV, IM): Inotropic Agents to downgrade the severity of allergic reaction. Dobutamine is to be the inotropic agent of discretion for escalating cardiac output, irrespective of whichever norepinephrine is also being bestowed. With substantially β-adrenergic properties, dobutamine is least probably to activate tachycardia than isoproterenol; IV Fluids (crystalloids) particularly isotonic crystalloid solutions such as NS and RL are selected owing to the implicit necessity for high volume IV fluid resuscitation; antihistamines are regulates the allergic response and minimize the clinical impact of histamine release; steroids are effective inhibitors of inflammatory procedures and effective anti-allergic constituents de-escalating the number, maturation and initiation of mast cell, which play a dominant function in anaphylaxis; aminophylline (Methylxanthines) [13]. (3c) Neurogenic shock: is a state of unbalance among sympathetic and parasympathetic regulations of cardiac action and vascular smooth muscle. The foremost signs are strong vasodilation with comparative hypovolemia while blood volume stays unchanged, at least primarily [5, 14]. Treatment: PA catheter useful in obviating over hydration; anticipate distinctive antecedent of hypotension; vasopressor assist with dopamine or dobutamine; transfer patient to regional spine center. Fluid resuscitation with crystalloid is the initially treatment for low BP. They replace fluid levels in the veins to assist stabilize BP. Vasopressors ubiquitously used are norepinephrine, dopamine and epinephrine can assist to thicken blood vessels and escalate BP. Atropine assists to normalize heart rate by suppressing parasympathetic activity [15].

2.3.3. Cardiogenic Shock

Cardiogenic shock is initially a disarrangement of cardiac work in the figure of a hypercritical suppression of the heart's pumping ability, antecedent by systolic or diastolic malfunction influencing to a de-escalated ejection fraction or impaired ventricular filling. It is delineated by SAP < 90 mmHg or mean ABP of 30 mmHg beneath the baseline value and cardiac index (CI) < 1.8 L/min/m^2 without pharmacologic or mechanical support or < 2.0 L/min/m2 with assist. In cardiogenic shock there is decrement of cardiac contractility; decrement of SV and CO; decrement of systemic tissue perfusion; and decrement of coronary artery perfusion, ultimately cause cardiogenic shock [16] (figure 4).

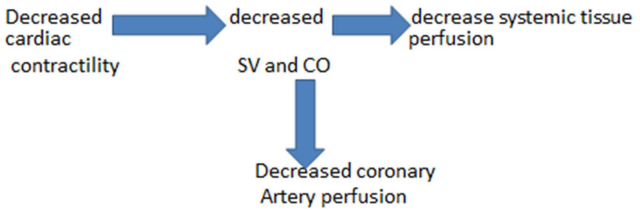

Figure 4. *Pathogenesis of cardiogenic shock.*

Treatment: Supplemental O_2. By downgrading ventricular afterload, vasodilating agents perhaps escalate CO without escalating myocardial ultimatum for O_2. The considerable termination of these medications is the peril of de-escalating arterial pressure to a degree that intermediates tissue perfusion. However, in certain patients, judicious use of nitrates and implicitly distinctive vasodilators may be improves microvascular perfusion and cellular work. Vasopressor support (dopamine or dobutamine): If hypotension is solemn or if it continues despite fluid administration, the usage of vasopressors is displayed. It is tolerable rehearse to administer a vasopressor temporarily while fluid resuscitation is proceeding, with the objective of withdrawing it, if feasible, after hypovolemia has been rectified. Adrenergic agonists are the 1st-line vasopressors because of hast onset of action, great potency, and short $t1/_2$, which permits easy dose adjustment. Definitive therapy (fibrinolytic therapy, PCI, CABG, ventricular support device, cardiac transplant) or clot-busting medications such as tPA to dissolve coronary artery clots. Anti-clotting (blood thinners) medications such as aspirin, clopidogrel and heparin to obviate fresh clots; medications that de-escalate the hearts workload and pain such as Morphine or fentanyl relieve anxiety or regulate heart rhythm. Beta agonists such as dobutamine, dopamine and norepinephrine to escalate the heart's pumping ability; Vasodilators such as nitrates (nitroglycerin) to relax and widen blood vessels [17].

2.3.4. Obstructive Shock

Obstructive shock is a circumstance caused by the obstruction of the large vessels of the heart itself. Although the symptoms look like those of cardiogenic shock, obstructive shock necessitates to be vividly differentiated from the ultimate because it is managed resign distinctively. In obstructive shock there is lack of adequate supply of blood; deprivation of oxygen and nutrients; energy produced anaerobically intercellular environment; low energy produced and acidotic [5, 18] (figure 5).

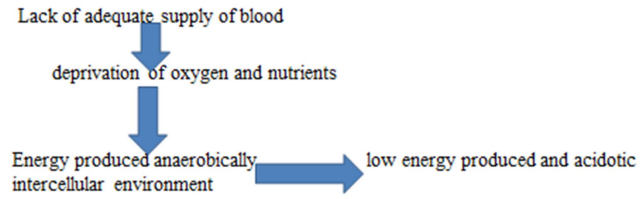

Figure 5. *Pathogenesis of obstructive shock.*

Treatment: Resuscitation addresses the ABCs-airway, breathing and circulation; supplemental O2 is bestowed, and introduction of a tube in a hollow organ is committed if displayed. High amount of IV fluids very quickly and bestowing medications that will escalate the blood pressure to escalate blood flow to the rest of the body and organs. Massive pulmonary embolism requires thrombolysis via IV alteplase (tPA) works to break up the clot. Obstructive shock antecedent by tension pneumothorax is needle decompression and chest tube placement to the affected area [19].

3. Conclusion

Shock is a state of sequencing from a recessed state of multiplex crucial body works owing to de-escalated tissue perfusion. It directs to de-escalate the delivering of oxygenated blood to the body's organs and tissues or functionally defective oxygen (O2) usage by peripheral tissues, sequencing in end-organ malfunction. Irreversible stage of shock is refractory stage and there perhaps adequate tissue affliction and cell death have happened. Tissue and cellular detriment are so ponderous that the organism dies even if perfusion is renewed. Whole blood is the optimal resuscitation fluid for patients who are severely bleeding and also used to treat patients who necessitate entire the constituents of blood, such as those who have sustained substantial blood loss owing to trauma or surgery. Packed RBCs to obviate tissue hypoxia and also hypovolemic shock necessitates adequate RBCs and water in the blood for the heart to propel the fluids adjacent to the blood vessels. Sodium bicarbonate is used to treat lactic acidosis and it increase the strong ion gap caused owing to shock; and anti-endotoxin antibodies to treat septic shock caused initially by endotoxins released during solemn gram-negative bacterial infections and carries a staggering 30-50% mortality rate.

Abbreviations

BP: Blood pressure; CPR: Cardiopulmonary resuscitation; DNA: Deoxyribonucleic acid; H2O: Water; NaCl: Sodium chloride; O2: Oxygen; RNA: Ribonucleic acid; SOFA: Sequential Organ Failure Assessment.

Disclosure

The author report no conflicts of interest in this work.

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