SHOCK

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Objectives:

1. Understand the importance of the subject.
2. Define shock from a pathophysiologic standpoint
3. Differentiate ,recognize the four main types of shock: hypovolemic, cardiogenic, obstructive, and distributive.
4. Diagnose and treat different types of shock
5. Keep general goals of shock resuscitation in mind
6. Assess the degree of hypovolemic shock

Why to learn about SHOCK?

*Shock is a life threatening emergency that may be reversible if appropriately recognized and treated.

(Golden hours).

**early recognition and appropriate management of shock are critical to avoid adverse outcomes, due to the high mortality rate of the disease process.

***basic knowledge about shock is universally important for all doctors, not just anesthesiologist!

****In fact, one of the core professional activities endorsed by the Association of American Medical Colleges that :

MEDICAL STUDENTS should have the ability to recognize a patient requiring urgent or emergent care and to initiate evaluation and management .

Definition:

- Shock is defined as the inadequate perfusion of tissue, means that oxygen and blood volume delivery fails to meet the cellular metabolic and oxygen consumption needs.
- The pathophysiology underlying shock is related to the determinant of oxygen delivery which depends on: Cardiac output, the vascular integrity , and the oxygen content .

Epidemiology / Pathophysiology:

Classically, four types of shock are described based on the physiologic disturbance:

1. ***Hypovolemic Shock:*** when the intravascular volume is depleted from blood or fluid losses (severe gastroenteritis).
2. ***Distributive Shock:*** occurs due to inappropriate vasodilatation of the peripheral blood vessels (sepsis, anaphylaxis, drug reactions, endocrine, and neurogenic abnormalities).
3. ***Obstructive Shock*** **:** is associated with obstruction of the heart or the great vessels:

   Tension pneumo/ hemothorax and cardiac tamponade). leads to high pressure in the chest which effectively obstructs venous return and diminishes cardiac output leading to inadequate perfusion).

   Massive pulmonary embolism may impede outflow of the right heart and lead to ventilation and perfusion mismatch .

4. ***Cardiogenic Shock*** **:** is failure of the "pump" and may arise from Acute Coronary Syndrome (ACS), mechanical failure, and/or arrhythmias.

Signs , Symptoms and Physiological Responses:

In a patient presenting with hypotension and concern for shock, the clinician must evaluate for diagnostic clues to the underlying cause and type of shock.

*At the onset of shock, the process is compensated and often reversible :

1-Preferential circulation is given to the vital organs as peripheral and splanchnic vasoconstriction diverts blood flow to the essential organs.

2-The body releases stress hormones such a catecholamines, cortisol, antidiuretic hormones, renin -angiotensin system among other adaptive responses to preserve fluid volume and to activate the fight" response !!!

- without prompt and aggressive treatment, a plasma volume loss of more than 30% may progress to end organ damage and cellular death.

I. Vital Signs: are important indicators of the patient's physiologic status .

1. *Temperature:* Fever or Hypothermia
2. *Heart rate:* heart rate is typically elevated in hypotension. To compensate for the low stroke volume while maintaining cardiac output per the equation:
   Cardiac Output = Heart Rate x Stroke Volume **

   Bradycardic or normal heart rates may be observed with neurogenic and cardiogenic shock.

3. B*lood pressure(BP)*: Hypotension defined as MAP <65 mm Hg is often a prominent feature of shock.

BP = CO x SVR

4. *Respiratory rate* :Tachypnea is commonly observed in patients with shock .

5. Oxygen saturation is typically preserved by increasing oxygen extraction. Saturations fall only at very late stages of hypoperfusion !

II. Examination: Applying the ABCDEs helps to efficiently evaluate the patient while also balancing management priorities concomitantly.

A. *Airway*: The airway should be assessed for patency

B. *Breathing*: The breath sounds should be equal on both sides of the chest on auscultation.

C. *Circulation*: is assessed with an evaluation of the peripheral pulses.

D. *Disability*: routinely assessed using the Glasgow Coma Scale (GCS)

- Low GCS (<8) is an indication for intubation, and a low threshold for intubation is required for any patient who is not protecting the airway .

E. *Exposure and secondary evaluation*: An exam of the patient's entire body is important in the critically ill patient. Evaluation of sources of infection, signs of bleeding, extremity perfusion and capillary refill, and volume status to determine the etiology of hypoperfusion.

GLASGOW COMA SCALE(GCS)

Behaviour	Response
 Eye Opening Response	4. Spontaneously 3. To speech 2. To pain 1. No response
 Verbal Response	5. Oriented to time, person and place 4. Confused 3. Inappropriate words 2. Incomprehensible sounds 1. No response
 Motor Response	6. Obeys command 5. Moves to localised pain 4. Flex to withdraw from pain 3. Abnormal flexion 2. Abnormal extension 1. No response

→Clinical picture may include:

Signs of Organ Hypoperfusion

Multiorgan Dysfunction Syndrome (MODS)

Result is: end organ failure

Types of Shock:

HYPOVOLEMIC SHOCK :

Hypovolemic shock results from loss of the intravascular circulating volume from fluid loss or blood loss. It has four classes:

Normal Blood volume for an adult ?????

Class I shock :750 ml loss (15%): Small volumes of fluid loss are well tolerated due to the compensatory mechanisms of the body . Heart rate, blood pressure, and urine output are maintained.

Class II shock :1500 ml (30%):

the heart rate increases to augment cardiac output. Blood pressure and urine output are maintained. Patients may experience mild anxiety.

Class III shock: 30-40% Ongoing volume loss greater than 1500-2000 ml overcomes the ability of the heart to maintain blood pressure.

BP decreases and urine output drops.

Patient is nearing irreversible shock and immediate, aggressive intervention with volume and blood replacement is necessary .

Physical exam findings show peripheral vasoconstriction and cold, clammy extremities, dry mucous membranes, and pallor associated with extreme anemia .

Class IV shock is reached with > 2000ml or blood or >40% of the circulating volume is loss.
*Patients are lethargic, with extreme tachycardia, profound hypotension, and oliguria.

*** Tachycardia will be prominent feature of severe shock before hypotension in late class III to IV hemorrhage, just before circulatory collapse .

Classes of Hypovolemic Shock:

	Class I	Class II	Class III	Class IV
Blood Loss	< 750	750-1500	1500-2000	> 2000
% Blood Vol.	< 15%	15 – 30%	30 – 40%	> 40%
Pulse	< 100	**> 100**	> 120	> 140
Blood Pressure	Normal	Normal	**Decreased**	**Decreased**
Pulse Pressure	Normal	**Decreased**	**Decreased**	**Decreased**
Resp. Rate	14 – 20	**20 – 30**	**30 – 40**	**> 40**
UOP	> 30	**20 – 30**	**5 – 15**	**negligible**
Mental Status	sl. Anxious	mildly anx	**confused**	**lethargic**
Fluid	crystalloid	crystalloid	**blood**	**blood**

- Hypovolemic shock may arise from bleeding due to trauma or atraumatic bleeding (such as an aortic aneurysm rupture or gastrointestinal bleed).
- Fluid losses :
- the GI tract from excessive vomiting or diarrhea , malabsorption.
- hormone imbalances, such as diabetes insipidus can result in excessive volume loss that may lead to shock if left untreated.

Diagnosis:

History and physical may direct the diagnosis of hypovolemia.

A history of trauma, recent surgery, or evidence of bleeding may help diagnose acute blood loss .

On exam, the patient initially appears to have a cold shock picture, and pallor

. Hgb and Hct may be decreased in acute blood loss .

Trends in Hb , Hct are a better assessment of blood loss than a single value.

Basic metabolic Laboratory:

1.Electrolyte assessment : K , Ca

2. Acid/ Base status: Large volume loss leads to poor oxygen delivery to the tissue and a transition to anaerobic metabolism in the tissue bed. Lactate is produced and lactic acid level elevates leading to a metabolic acidosis.

Renal function: In severe hypovolemic shock the Urea level is elevated often > 20mg/dl. As shock progresses, renal failure may occur from acute tubular necrosis and cause further elevation of the renal function parameters .

Coagulation studies :(PT/ INR; PTT, fibrinogen, fibrin related markers):

In severe hemorrhagic shock coagulopathy, secondary to an overactivation of clot breakdown (termed fibrinolysis) may occur.

SVO2: Mixed venous oxygen saturation will decreased.*

Imaging: history and examination guided.

. X-Ray CXR + pelvic x-ray

. bedside ultrasound

. CT Scan in stable patients

. Angiography may localize sources of bleeding

. Direct peritoneal aspiration/lavage : free blood go for emergent laparotomy.

Treatment:

Aggressive **replacement of volume** while attending to the underlying etiology is the mainstay of treatment of hypovolemic shock .

In traumatic bleeding, patients should be triaged per the ABCDE's of Advanced Trauma Life Support (ATLS) and to identify the source of bleeding.

All forms of hemorrhage necessitate large bore IV access (14 or 16 gauge IV or short, large diameter resuscitation lines) .

Crystalloid resuscitation :

The new guide lines recommend that Crystalloids administration should be limited to 1-2 liters of IV fluid, with a transition to early blood and plasma resuscitation when it is available.

- Colloids can be given.

- In severe dehydration IV isotonic crystalloids can be given(Normal Saline (NS) or Lactated Ringers (LR)) as a bolus of 20-30ml/kg and repeated every 5-10 minutes, may quickly restore circulating volume .

End Points of Resuscitation "Goal-directed therapy"

Use objective hemodynamic and physiologic values to guide therapy:

1. Urine output > 0.5 mL/kg/hr
2. CVP 8-12 mmHg
3. MAP 65 to 90 mmHg
4. Central venous oxygen concentration > 70%

DISTRIBUTIVE SHOCK

- Distributive shock results from the inappropriate vasodilation of the peripheral vasculature(decreased SVR).
- Septic shock, anaphylactic shock, and neurogenic shock are all examples of this pathophysiology.

Septic shock:

Severe sepsis and septic shock are highly lethal conditions that occur in response to infection.

- Severe sepsis is defined as a systemic host response to infection that leads to organ dysfunction.

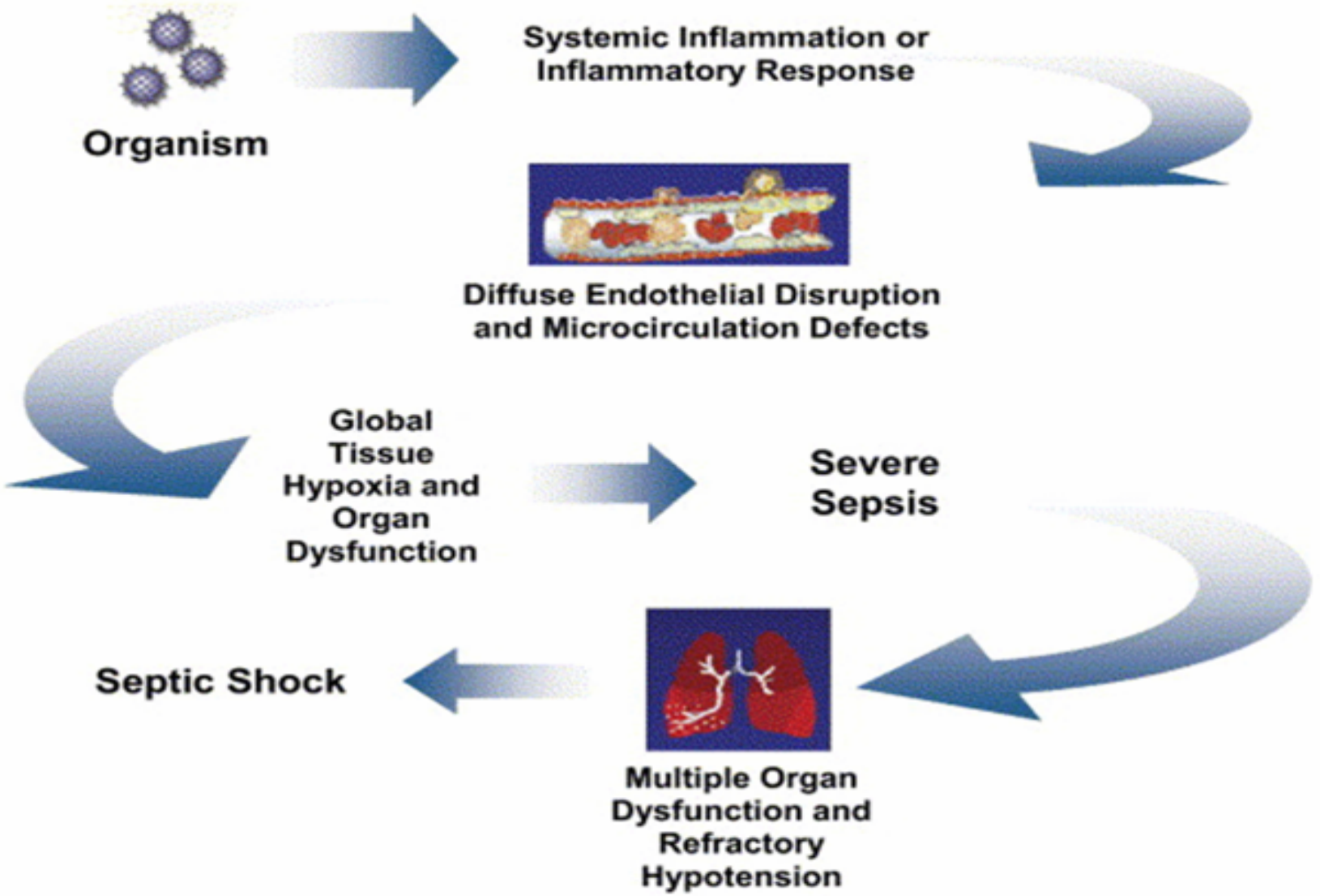
-Septic shock is termed when the response to the infection leads to hypotension requiring vasopressors to maintain a mean arterial pressure (MAP) of >65mm Hg with concomitant lactic acidosis (>2 mmol/L)

- mortality up to 18-46% .

The clinical sequela of sepsis is mediated by the host response to the pathogen.

Infection leads to the release of pro and anti-inflammatory cytokines. Tumor necrosis factor , interleukins (IL) which lead to the recruitment of macrophages, neutrophils that lead to systemic alterations in perfusion, microcirculation, cell death, and organ dysfunction.

Each system of the body is affected by the host response to infection.


Organism
Systemic Inflammation or Inflammatory Response
Diffuse Endothelial Disruption and Microcirculation Defects
Global Tissue Hypoxia and Organ Dysfunction
Severe Sepsis
Multiple Organ Dysfunction and Refractory Hypotension
Septic Shock

Diagnosis:

Two or more of SIRS criteria

- Temp > 38 or < 36 C
- HR > 90
- RR > 20
- WBC > 12,000 or < 4,000

Plus

presumed existence of infection

Treatment

- 1. Infection source control : Broad spectrum antibiotics are strongly recommended within 1 hour of sepsis recognition .

  Surgical drainage.

  Identification of the infectious source with cultures drawn before antibiotic administration.

- 2. Resuscitation and life support in critically ill patients :

- Fluid management - a 30ml /kg crystalloid infusion in the first 3 hours of the identification of sepsis is strongly recommended to support blood pressure

- urine output greater than 0.5cc/kg in is also a helpful determination of adequate volume status

- Vasopressors are often needed to support blood pressure in septic shock

Norepinephrine , Epinephrine , Vasopressin

- Mechanical ventilation is often needed to support patient with sepsis and sepsis related acute respiratory distress ARDS.

- Renal replacement therapy may be required in cases of severe acute kidney injury with hyperkalemia, severe acidosis, uremia or volume overload.

Neurogenic shock:

Neurogenic shock describes the hemodynamic changes resulting from a sudden loss of autonomic tone due to spinal cord injury. It is commonly seen when the level of the injury is above T6.

. This is estimated to occur in up to 20% of cervical spine injuries .

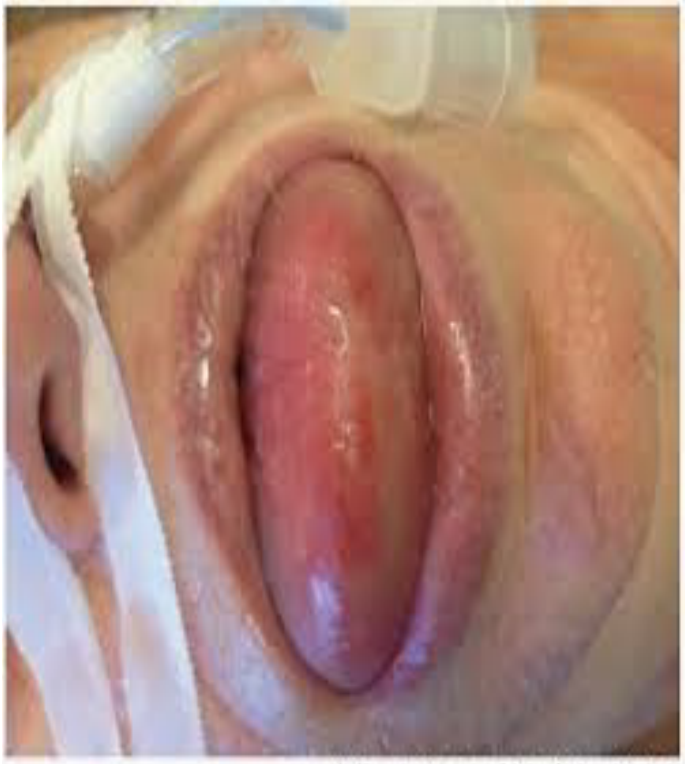
.While Spinal shock, refers to loss of all sensation below the level of injury and is not circulatory in nature.

- Diagnosis:
- In traumatic injury, the physical exam may suggest a high level spinal cord injury. Which can be confirmed by imaging CT or MRI.
- Treatment: (supportive).

After ruling out other concomitant forms of shock, fluids and pressor use may be required to support the patient.

Anaphylactic shock:

- Allergic reactions(Exaggerated immunological responses to antigenic(eg, antibiotic***, muscle relaxants*, latex**) stimulation in previously sensitized persons) liberate vasodilatory substances such as histamine through immunoglobulin E (Ig-E)(Anaphylaxis).
- Or a direct activation of the complement may trigger mast cell degranulation and basophil to release of histamine leading to "anaphylactoid reactions".
- Incidence is about 1:3000 to 1:20000(mortality is 4%).
- Despite different mechanisms BOTH are clinically indistinguishable and equally life-threatening depending on the severity of the reaction.
- Massive systemic vasodilation occurs leading to cardiovascular collapse, facial and tongue swelling leading to airway compromise, and bronchospasm of the airways.
- Diagnosis is clinical: hypotension, tachycardia, skin rash , itching, angioedema
- The antigen enters the circulation via respiratory system, eyes, skin, IV, IM, or peritoneal).



Shiber J, Tropical Med Surg 2014

Treatment: ABC's

Stop the exposure to the trigger while assessing the patient's airway and hemodynamic stability is equal to therapy .

1. Airway: Lip and tongue swelling, called angioedema, as well as pharyngeal and glottic swelling may compromise the airway.
   a. For signs of impending airway compromise, securing an endotracheal tube early is a priority.
   b. Supplemental O2 and continuous monitoring are necessary.

2- IV access should be emergently obtained and normal saline administered to raise BP.

3- Epinephrine injection(0.01-0.5 mg IV or IM) depending on the severity, with repeat dosing every 5-15 minutes as needed. A drip(IV Infusion) can be prepared for refractory response.

Epinephrine is the primary vasopressor of choice; however, others may be added to maintain MAP >65 mm Hg.

4. Albuterol is a bronchodilator for bronchospasm and can be given as a nebulizer.

5*. Both H1 and H2 antihistamines.

6*. Consider steroid for airway edema and severe reactions: hydrocortisone, dexamethazone.

OBSTRUCTIVE SHOCK

Impediment to the flow of blood in the cardiopulmonary circuit results in obstructive shock

Classic examples of obstructive shock are from

1. Tension pneumothrax :A history of trauma ,hypoxemia
2. Pericardial tamponade
3. pulmonary Emboli: Pulmonary emboli are blood clots that obstruct the pulmonary venous circulation.

4- Restrictive cardiomyopathies

Treatment:

- The mainstay of treatment is directed at the underlying etiology of the obstruction to cardiopulmonary flow.

. In tension pneumothorax, placement of a needle in the midclavicular line in the second intercostal space will change a tension pneumothorax to a simple pneumothorax.

Pressurized hemothorax and moderate to large pneumothorax should also be treated with a placement of a chest tube in the 4th intercostal space in the anterior axillary line.

Pulmonary emboli are treated with anticoagulation .

CARDIOGENIC SHOCK

- Cardiogenic shock results from failure of the cardiac "pump." Failure of forward delivery of blood and, therefore, oxygen to the tissues, leads to shock.
- When pressures elevate on the left side, pulmonary edema may occur,
- when there is right heart dysfunction, systemic congestion may result.

- **Causes:** myocardial infarct (MI); valvular insufficiency, and arrhythmias .

- **Diagnosis** :

Diagnosis of myocardial infarction per the WHO guidelines requires:

a. Detection of increase and/or decrease of cardiac biomarkers (preferably troponin) .

b. Evidence of myocardial ischemia with at least 1 of the following: symptoms, ECG changes, or supportive imaging

- ECG: ST elevation MI(STEMI)or Non-ST elevation MI (NSTEMI) and Unstable Angina (UA)
- Typically : crushing substernal chest pain, upper extremity, back, epigastric or jaw pain

Treatment:

- ABCs of resuscitation is essential to the management of cardiogenic shock. Intubation and ventilatory support (where appropriate) and obtaining excellent IV access and invasive blood pressure monitoring is crucial to effectively supporting the critical patient.

1-Medication administration

a. Aspirin 325 mg and heparin IV should be administered expediently.
b. Glycoprotein IIb/IIIa inhibitor with NSTEMI may be beneficial.

2. Pressor support to a MAP of 65 mmHg Norepinephrine and dopamine are first line agents.
3. Percutaneous Coronary Intervention (PCI) is the mainstay of therapy and should be administered to all STEMI within 12 hours of symptoms .

4. Alternatively, if PCI cannot be administered within 120 minutes of arrival, then fibrinolytic therapy should be administered in absence of contraindication.

5. Coronary Artery Bypass Grafting (CABG) is indicated in patients with STEMI with difficult PCI

6. Mechanical support
Intra-Aortic Balloon Pump (IABP) is a type of mechanical circulatory support

Goals of Shock Resuscitation

- Airway patency
- Control Work of Breathing
- Optimizing Circulation
- End Points of Resuscitation
  Goal-directed therapy: Use objective hemodynamic and physiologic values to guide therapy

1. Urine output > 0.5 mL/kg/hr
2. CVP 8-12 mmHg
3. MAP 65 to 90 mmHg
4. Central venous oxygen concentration > 70%

In general, support and treat the cause…

Thank you