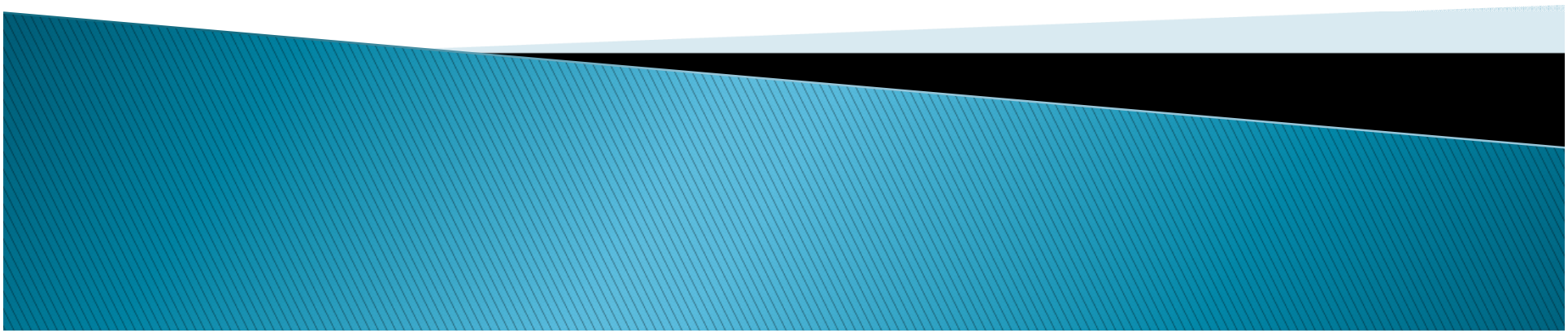
Circulatory shock

Jean-Louis Vincent,M.D.,Ph.D.,and Daniel De Backer,M.D.,Ph.D

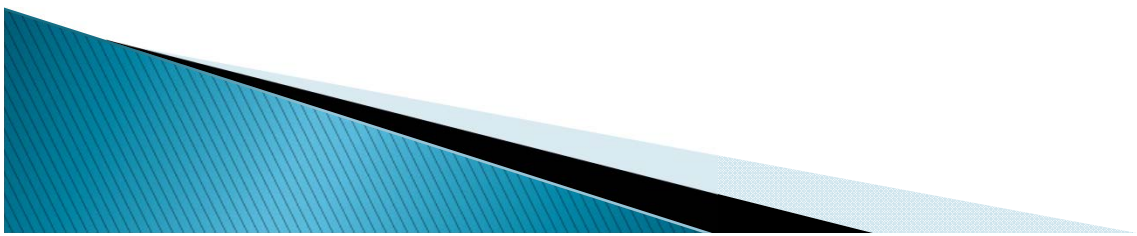
NEJM, Nov 2013



- Shock is a common condition in critical care
- about one third of patients in the intensive care unit (ICU)

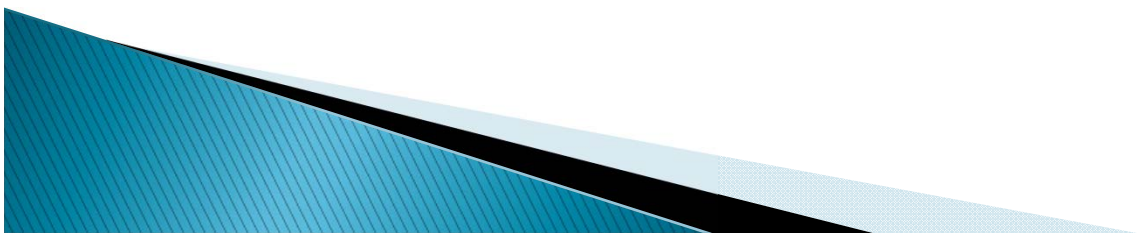
- A diagnosis of shock is based on clinical, hemodynamic, and biochemical signs.

- Summarized into three components

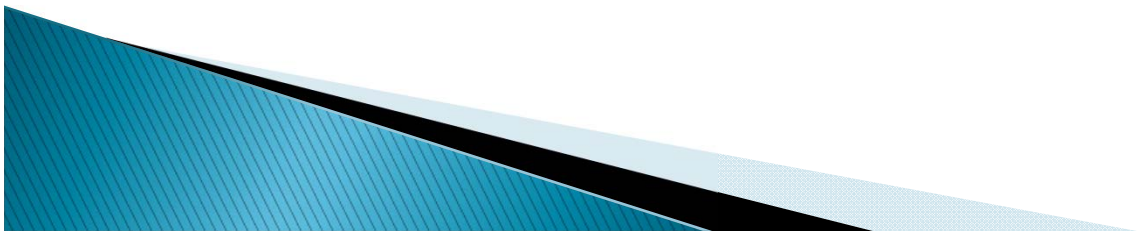


Summarized into three components

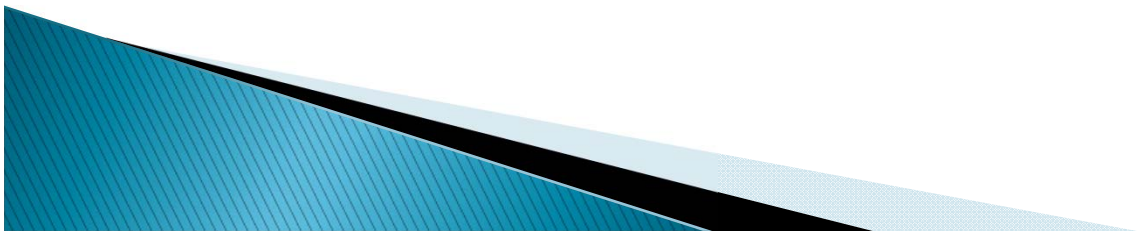
First, systemic arterial hypotension is usually present.



- Second, there are clinical signs of tissue hypoperfusion, apparent through the three "windows" of the body
  - **cutaneous** (cold and clammy, with vasoconstriction and cyanosis)
  - **renal** (urine output of <0.5 ml/kg/hr)
  - **neurologic** (altered mental state)



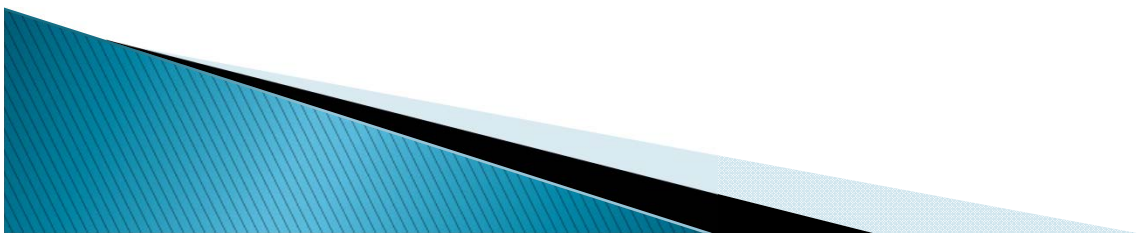
- Third, hyperlactatemia indicating abnormal cellular oxygen metabolism
- Blood lactate>1.5 mmol per liter in acute circulatory failure (normal 1 mmol per liter).



Pathophysiological Mechanisms

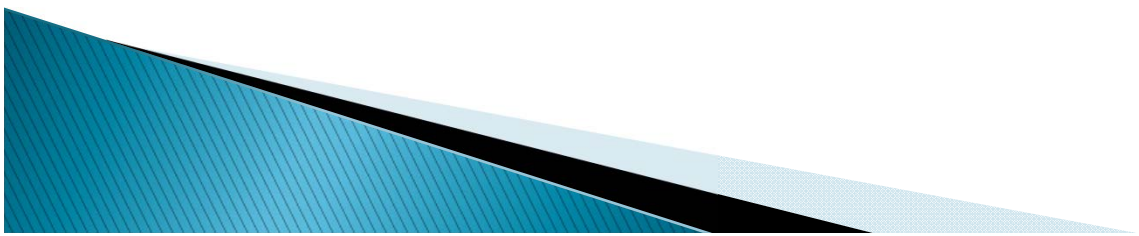
- four potential pathophysiological mechanisms

- 1)Hypovolemia (from internal or external fluid loss),
- 2)cardiogenic factors (e.g., acute myocardial infarction, end-stage cardiomyopathy, advanced valvular heart disease, myocarditis, or cardiac arrhythmias),



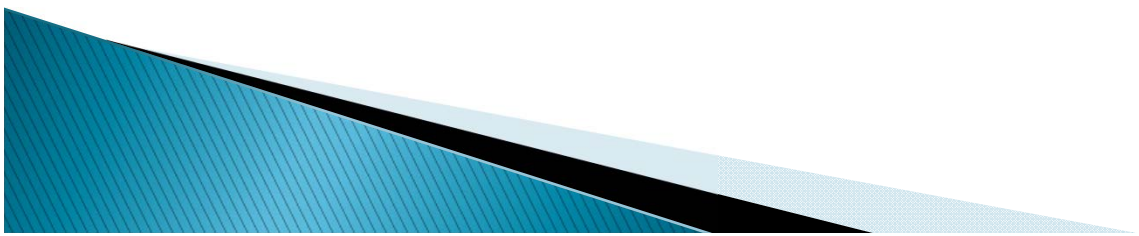
- 3)Obstruction (e.g., pulmonary embolism, cardiac tamponade, or tension pneumothorax), or

- 4)Distributive factors (e.g., severe sepsis or anaphylaxis)



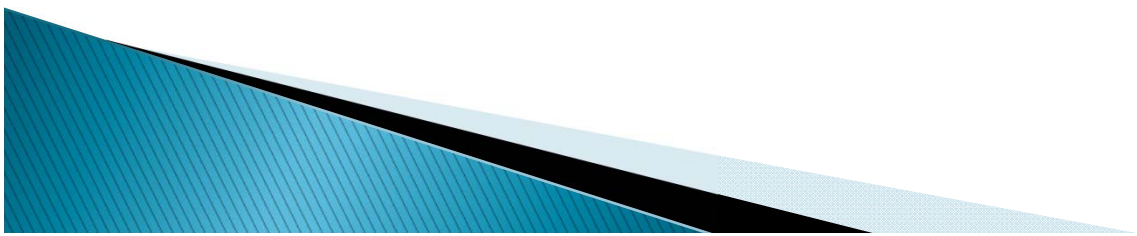
- The first three mechanisms are characterized by low cardiac output and inadequate oxygen transport.

- In distributive shock, the main deficit lies in the periphery, with decreased systemic vascular resistance and altered oxygen extraction.

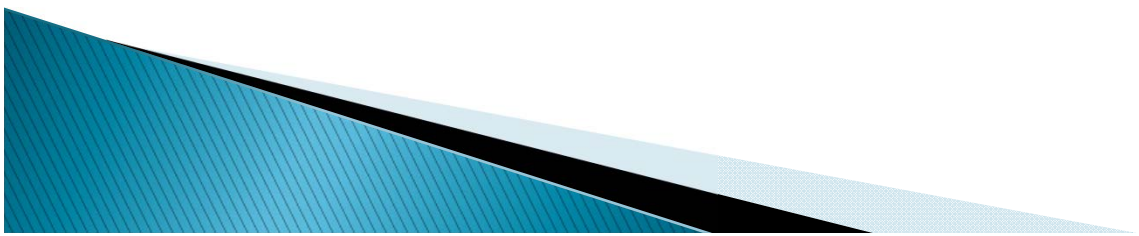


Differential Diagnosis

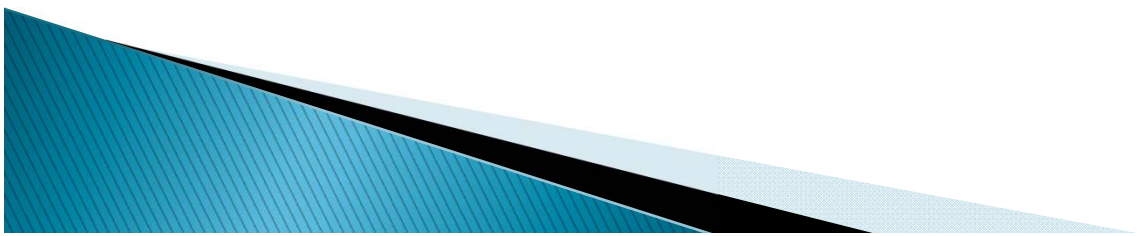
- Septic shock is the most common form of shock followed by cardiogenic and hypovolemic shock.
- Obstructive shock is relatively rare.



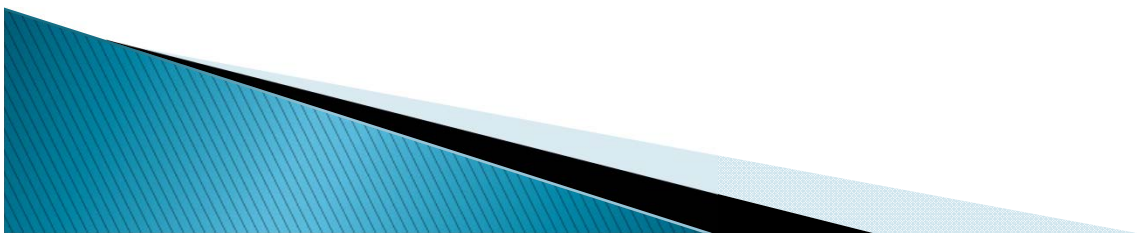
- The type and cause of shock may be obvious from the medical history, physical examination, or clinical investigations
- For example, shock after traumatic injury is likely to be hypovolemic (due to blood loss),
- Cardiogenic shock or distributive shock may also occur, alone or in combination



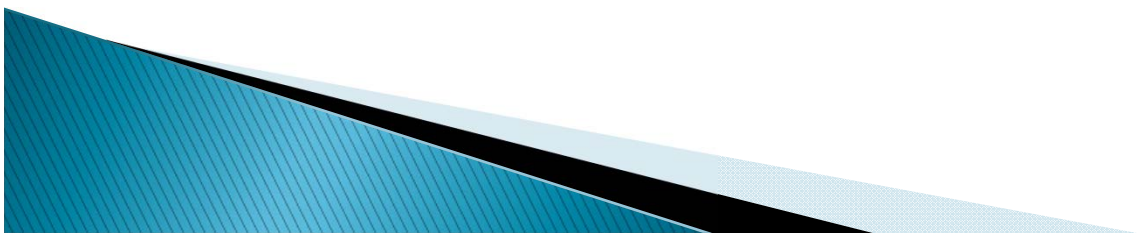
- A full clinical examination should include assessment of skin color and temperature, jugular venous distention, and peripheral edema



- The diagnosis can be refined with point-of-care echocardiographic evaluation, which includes assessment for
- 1) Pericardial effusion,
- 2) Measurement of left and right ventricular size and function
- 3) Assessment for respiratory variations in vena cava dimensions,and
- 4) Calculation of the aortic velocity-time integral, measure of stroke volume

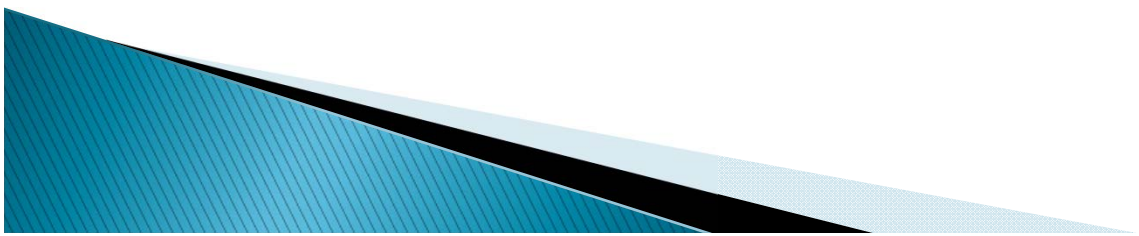


- Whenever possible, focused echocardiography should be performed as soon as possible in any patient presenting with shock.

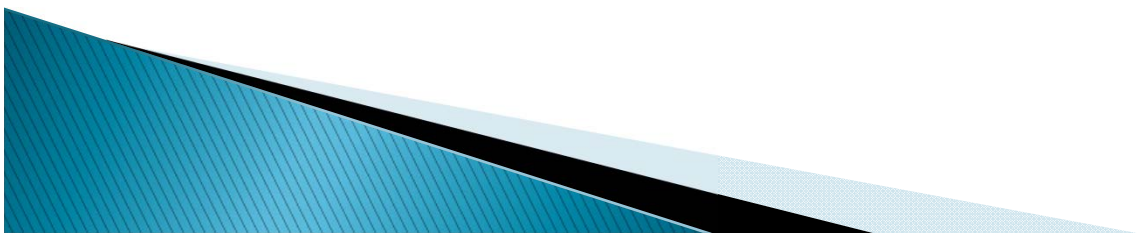


Initial Approach to the Patient in Shock

- Early, adequate hemodynamic support of patients in shock is crucial to prevent worsening organ dysfunction and failure.
- Resuscitation should be started even while investigation of the cause is ongoing.

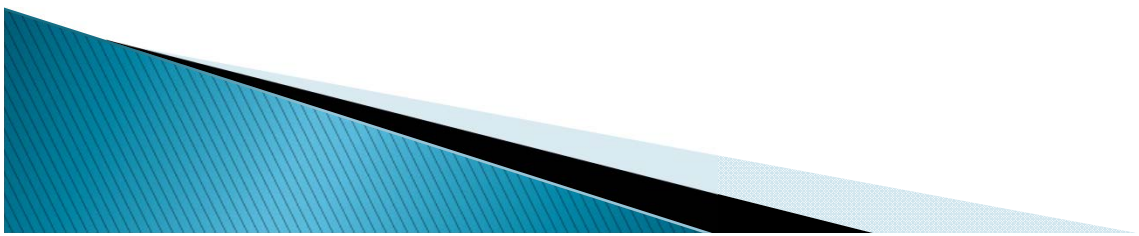


- Once identified, the cause must be corrected rapidly
- Unless the condition is rapidly reversed, an arterial catheter should be inserted for monitoring of arterial blood pressure and blood sampling.
- Plus a central venous catheter for the infusion of fluids and vasoactive agents and to guide fluid therapy.



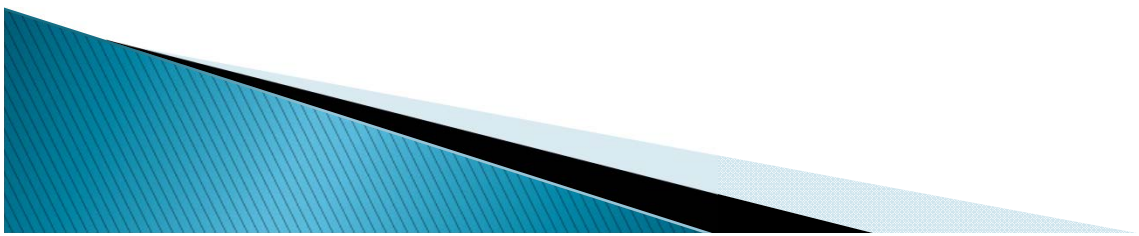
VIP rule

- ventilate (oxygen administration)
- infuse (fluid resuscitation)
- pump (administration of vasoactive agents).



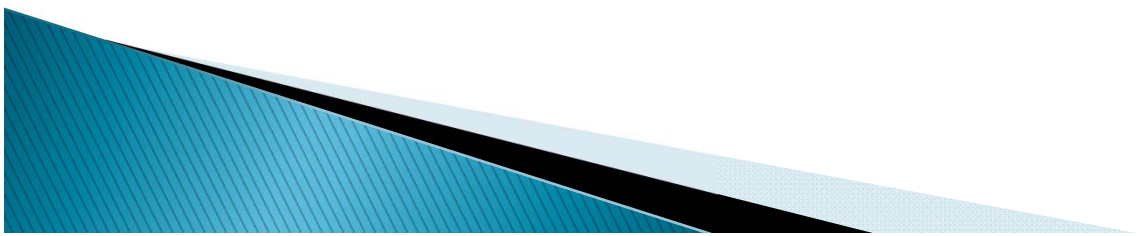
Ventilatory Support

- Oxygen should be started immediately.
- To increase oxygen delivery and prevent pulmonary hypertension
- Pulse oximetry is often unreliable – vasoconstriction
- Precise determination of oxygen requirements will often require blood gas monitoring



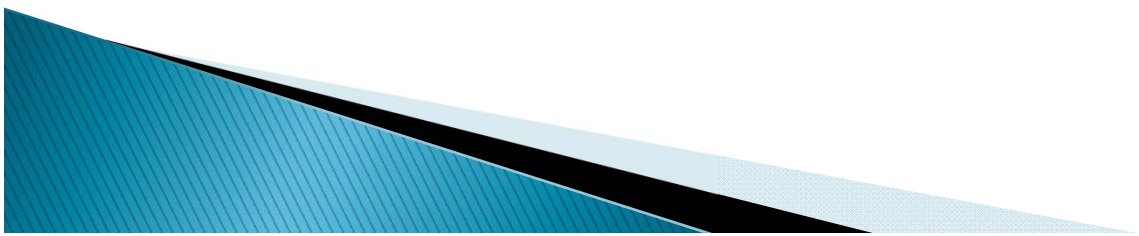
- invasive mechanical ventilation in nearly all patients with severe dyspnea, hypoxemia, or persistent or worsening acidemia (pH, <7.30)

- the additional benefits of reducing the oxygen demand of respiratory muscles and decreasing left ventricular afterload by increasing intrathoracic pressure



- An abrupt decrease in arterial pressure after the initiation of invasive mechanical ventilation strongly suggests hypovolemia and a decrease in venous return

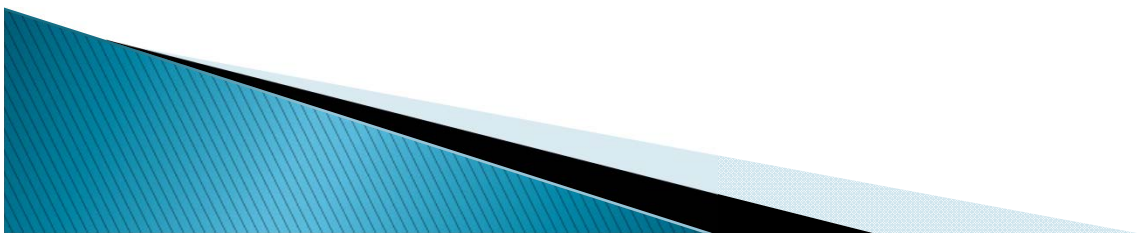
- The use of sedative agents should be kept to a minimum to avoid further decreases in arterial pressure and cardiac output



Fluid Resuscitation

- to improve microvascular blood flow and increase cardiac output is an **essential part of the treatment of any form of shock**

- Even patients with cardiogenic shock may benefit from fluids



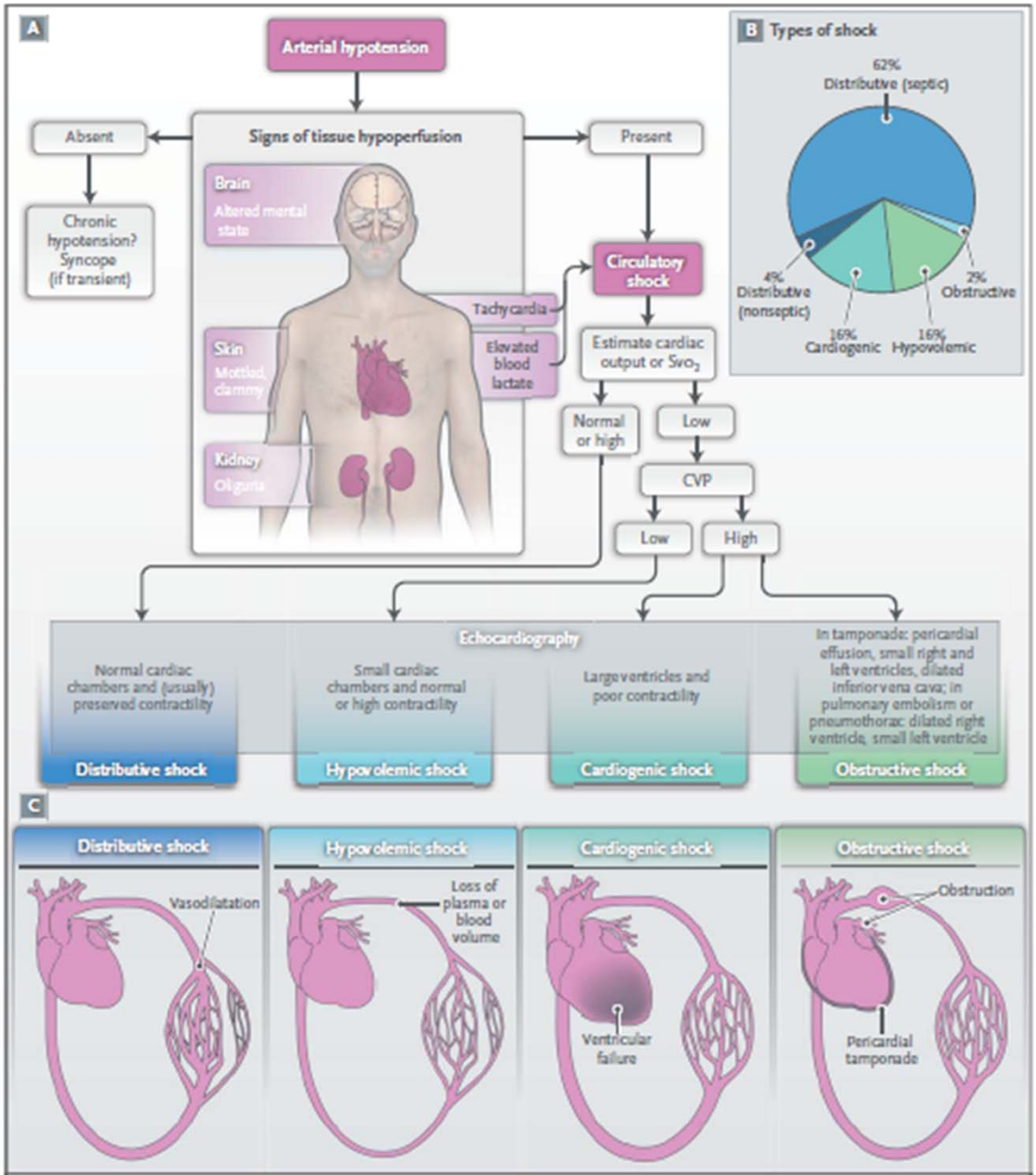


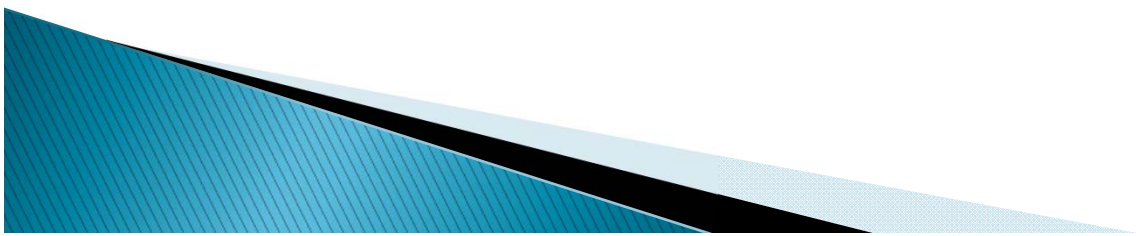
Figure 1. Initial Assessment of Shock States.

Shown is an algorithm for the initial assessment of a patient in shock (Panel A), relative frequencies of the main types of shock (Panel B), and schematic representations of the four main types of shock (Panel C). The algorithm starts with the most common presentation (i.e., arterial hypotension), but hypotension is sometimes minimal or absent. CVP denotes central venous pressure, and Svo_2 mixed venous oxygen saturation.

A fluid challenge incorporates four elements

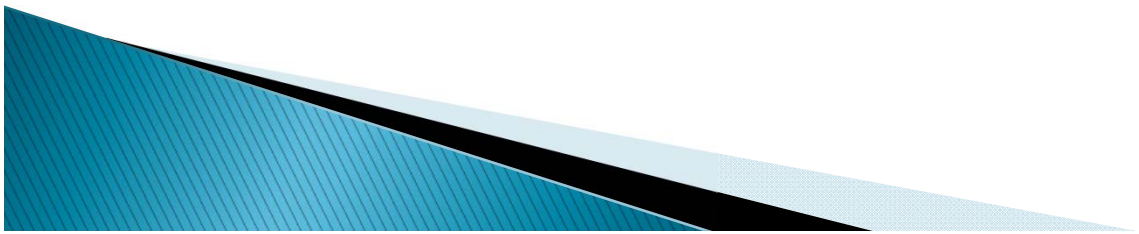
- First, the **type** of fluid must be selected. ***Crystalloid solutions*** are the first choice.
- Second, the **rate** of fluid administration must be defined. Fluids should be infused ***rapidly to induce a quick response*****.**
- An infusion of ***300 to 500 ml*** of fluid is administered during a period ***of 20 to 30 minutes.***

- Third, the ***objective of the fluid challenge*** must be defined. In shock, the objective is usually an ***increase in systemic arterial pressure.***
- Finally, the ***safety limits*** must be defined.
- Pulmonary edema is the most serious complication of fluid infusion.



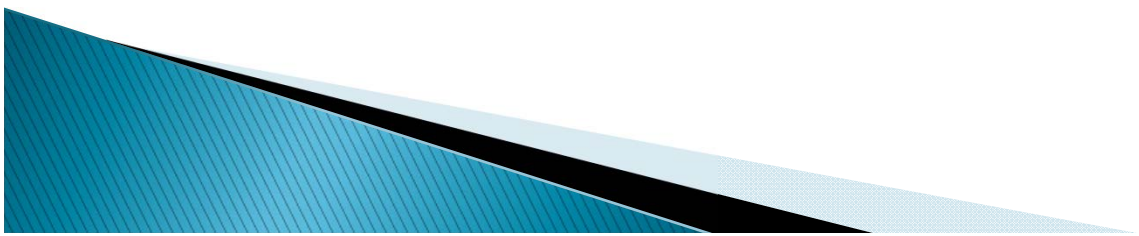
- Pulmonary edema is the most serious complication of fluid infusion.

- Fluid challenges can be repeated as required but must be stopped rapidly in case of nonresponse in order to avoid fluid overload



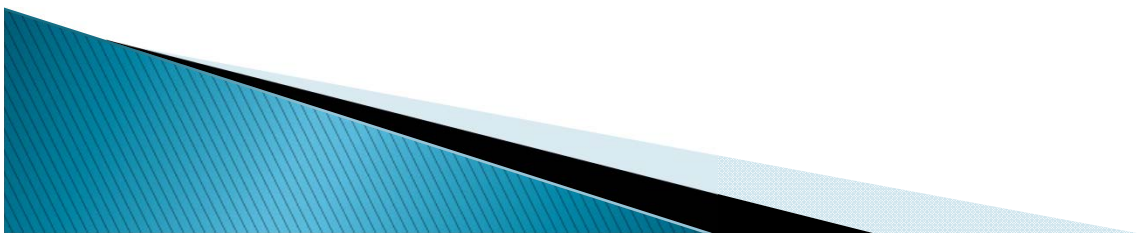
Vasoactive Agents

- ***Vasopressors***
- If hypotension is severe or if it persists despite fluid administration, the use of vasopressors is indicated.
- Adrenergic agonists are the first-line vasopressors (rapid onset of action, high potency, and short half-life), which allows easy dose adjustment.



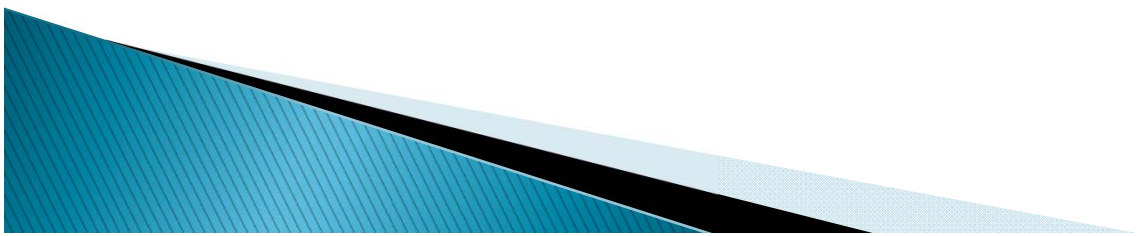
- β-adrenergic stimulation can increase blood flow but also increases the risk of myocardial ischemia as a result of increased heart rate and contractility.

- α-adrenergic stimulation will increase vascular tone and blood pressure but can also decrease cardiac output and impair tissue blood flow.



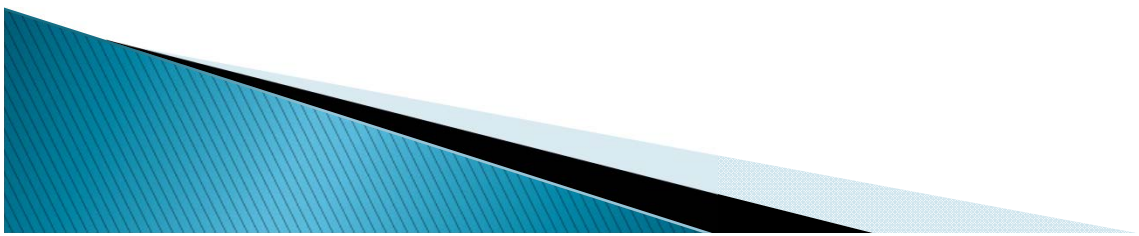
Norepinephrine

- the vasopressor of first choice
- Predominantly α-adrenergic properties,modest β-adrenergic effects help to maintain cardiac output
- Increase in mean arterial pressure, with little change in heart rate or cardiac output
- 0.1 to 2.0 µg per kilogram of body weight per minute.

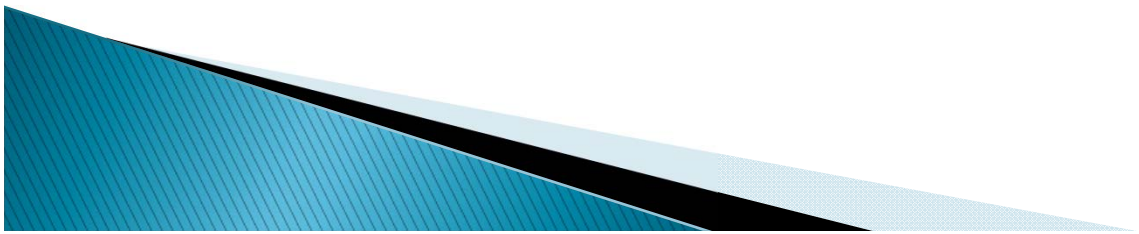


Dopamine

- predominantly β-adrenergic effects at lower doses and α-adrenergic effects at higher doses,
- its effects are relatively weak.
- no advantage over norepinephrine as the first-line vasopressor agent;

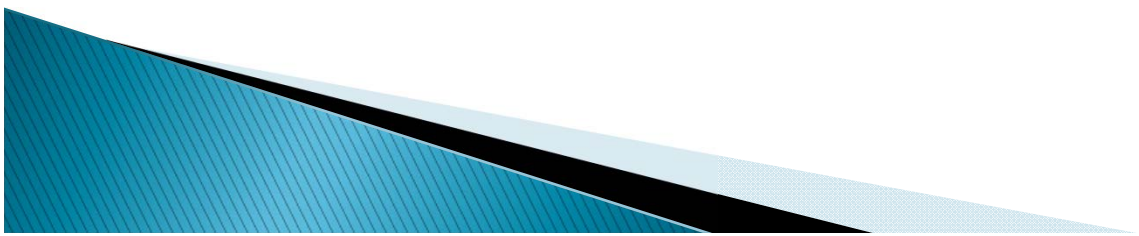


- it induced more arrhythmias
- increased 28-day rate of death among patients with cardiogenic shock
- Higher rates of death among patients with septic shock



Epinephrine

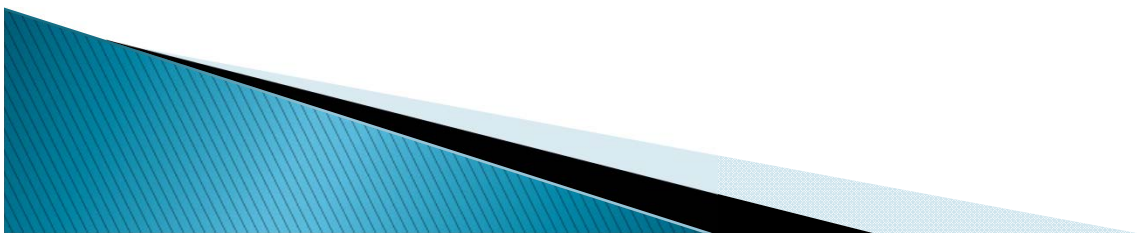
- predominantly β-adrenergic effects at low doses, with α-adrenergic effects becoming more clinically significant at higher doses.
- Increased rate of arrhythmia and
- a decrease in splanchnic blood flow and
- can increase blood lactate levels, probably by increasing cellular metabolism.



Vasopressin

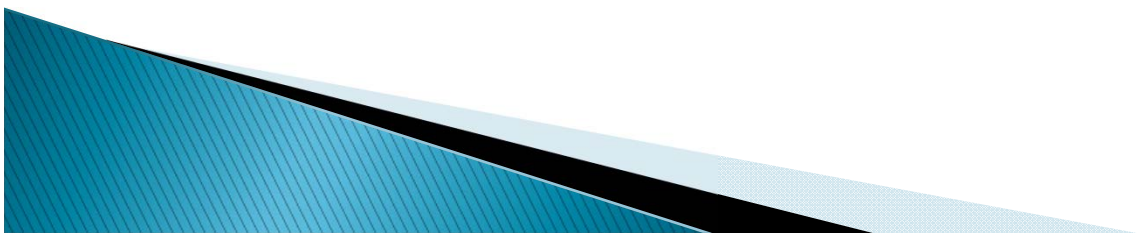
- Vasopressin deficiency can develop in patients with very hyperkinetic forms of distributive shock,
- the administration of low-dose vasopressin may result in substantial increases in arterial pressure.
- doses not higher than 0.04 U per minute

- The addition of low-dose vasopressin to norepinephrine in the treatment of patients with septic shock was safe and
- may have been associated with a survival benefit for patients with forms of shock that were not severe and for those who also received glucocorticoids



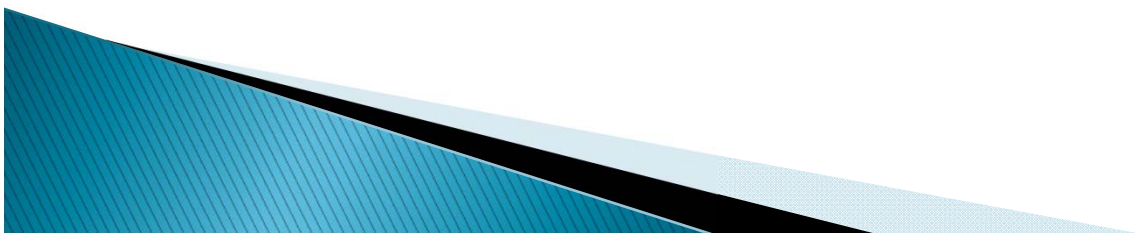
Inotropic Agents–Dobutamine

- the inotropic agent of choice
- for increasing cardiac output, regardless of whether norepinephrine is also being given.
- With predominantly β-adrenergic properties dobutamine is less likely to induce tachycardia.
- Intravenous doses in excess of **20 µg per kilogram per minute** usually provide little additional benefit



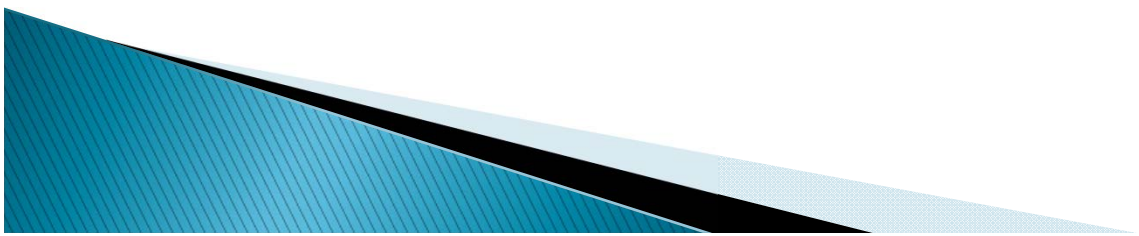
Phosphodiesterase type III inhibitors

- such as milrinone and enoximone, combine inotropic and vasodilating properties
- By decreasing the metabolism of cyclic AMP, may reinforce the effects of dobutamine
- Also be useful when β-adrenergic receptors are downregulated or in patients recently treated with beta-blockers

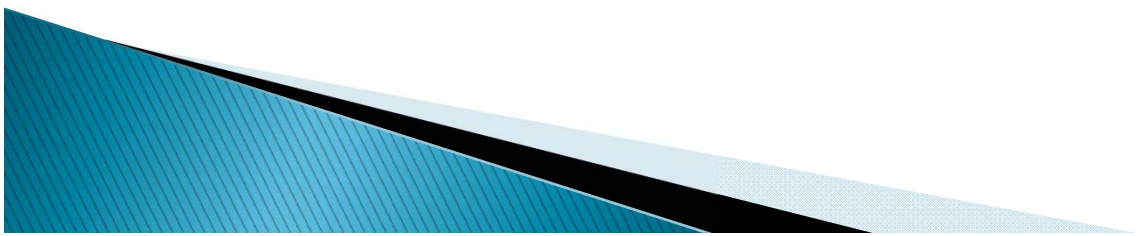


- The long half-lives of these agents (4 to 6 hours) prevent minute-to-minute adjustment.

- Intermittent, short-term infusions of small doses of phosphodiesterase III inhibitors may be preferable to a continuous infusion.



- **Levosimendan** acts primarily by binding to cardiac troponin C and increasing the calcium sensitivity of myocytes, it also acts as a vasodilator by opening ATPsensitive potassium channels in vascular smooth muscle. This agent has a half-life of several days

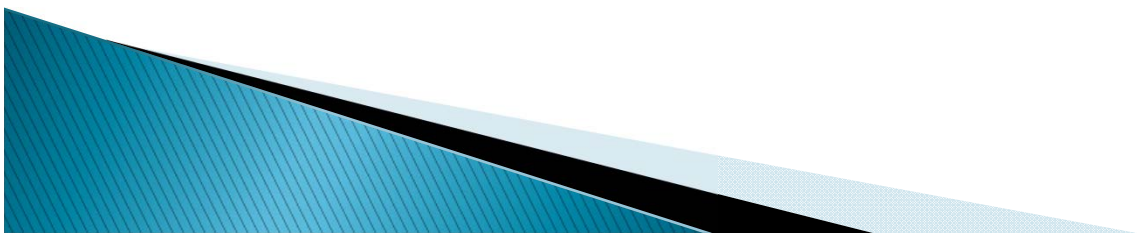


Vasodilators

- By reducing ventricular afterload
- increase cardiac output without increasing myocardial demand for oxygen
- The risk of decreasing arterial pressure to a level that compromises tissue perfusion.
- use of nitrates and possibly other vasodilators may improve microvascular perfusion and cellular function.

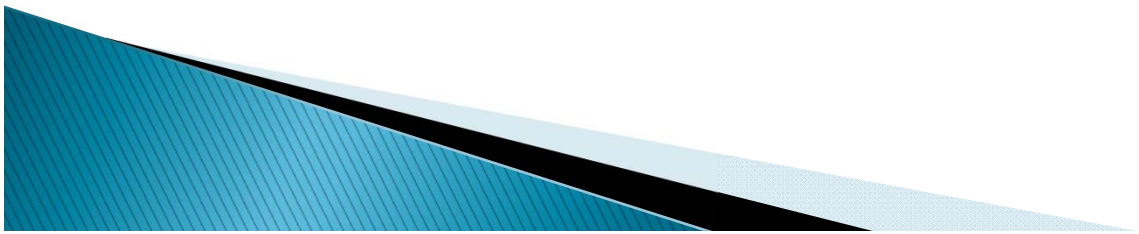
Mechanical Support

- IABC can reduce left ventricular afterload and increase coronary blood
- No beneficial effect of IABC in patients with cardiogenic shock
- Is not currently recommended to use routinely.



ECMO

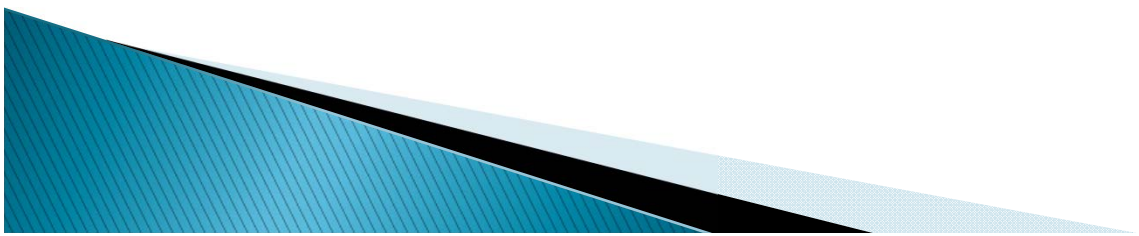
- used as a temporary lifesaving measure in patients with reversible cardiogenic shock or as a bridge to heart transplantation.



Goals of Hemodynamic Support Arterial Pressure

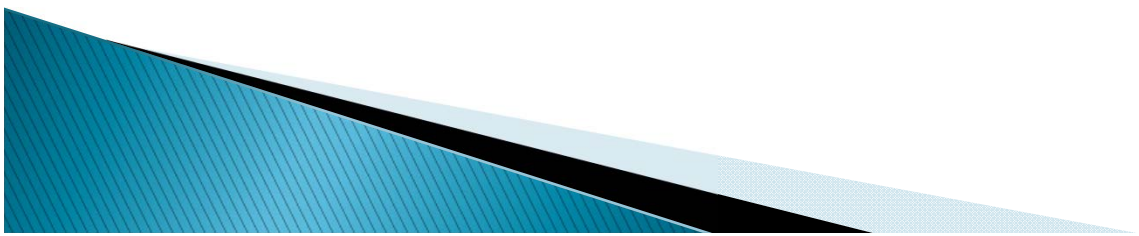
- The primary goal of resuscitation should be not only to restore blood pressure but also to provide adequate cellular metabolism.

- Mean systemic arterial pressure of 65 to 70 mm Hg is a good initial goal.



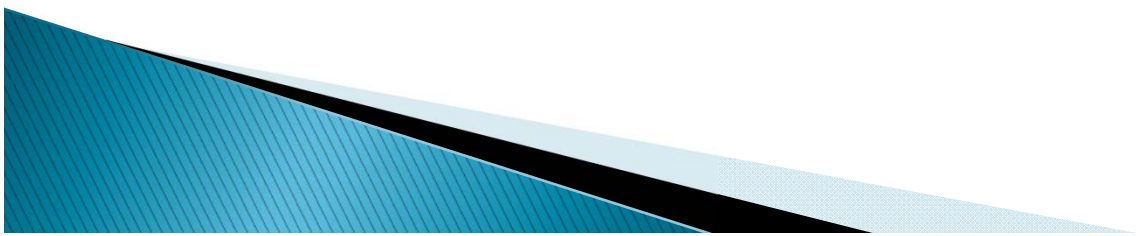
- should be adjusted to restore tissue perfusion, assessed on the basis of mental status, skin appearance, and urine output.

- Oliguric patients the effects of a further increase in arterial pressure on urine output should be assessed regularly, unless acute renal failure is already established.



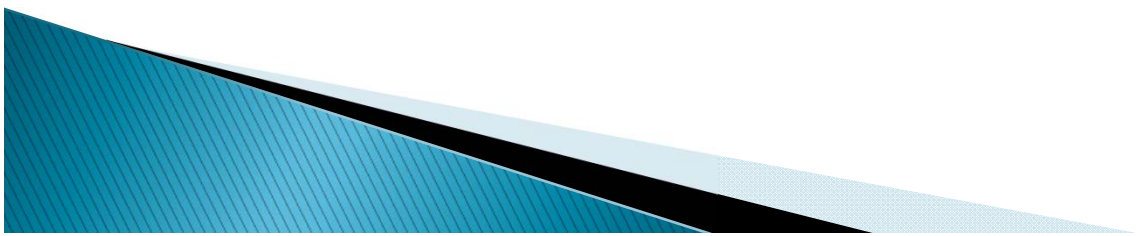
- A mean arterial pressure lower than 65 to 70 mm Hg may be acceptable in a patient with acute bleeding who has no major neurologic problems

- Until the bleeding is controlled



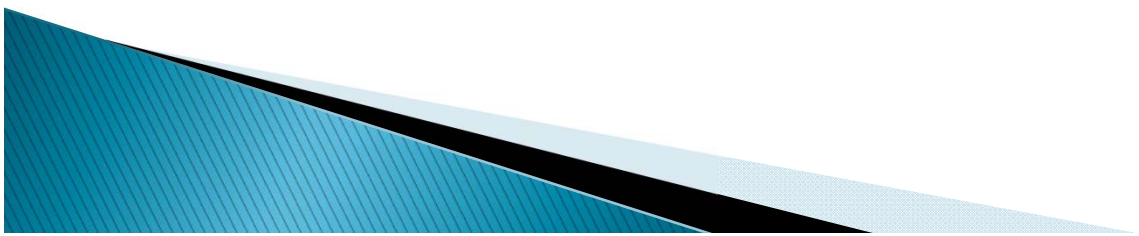
Cardiac Output and Oxygen Delivery

- After correction of hypoxemia and severe anemia, cardiac output is the principal determinant of oxygen delivery.
- The optimal cardiac output is difficult to define.
- Can be measured by means of various techniques.

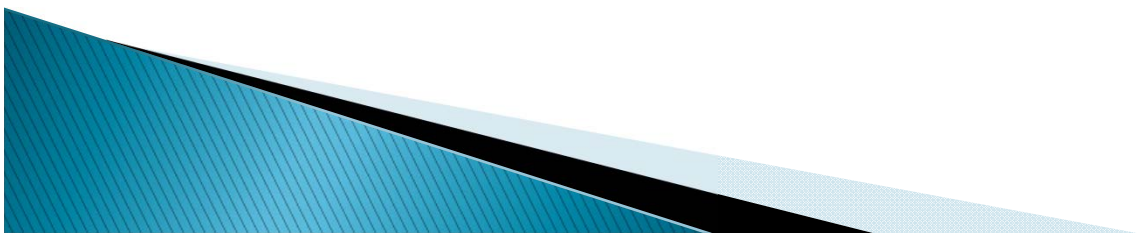


- Absolute measures of cardiac output are less important than monitoring trends in response to interventions such as a fluid challenge

- SvO2 may be helpful in assessing the adequacy of the balance between oxygen demand and supply



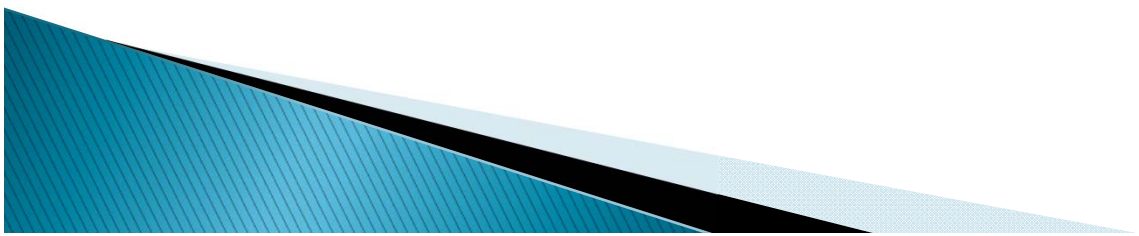
- SvO2 measurements are also very useful in the interpretation of cardiac output.
- Decreased in patients with low-flow states or anemia
- Normal or high in those with distributive shock



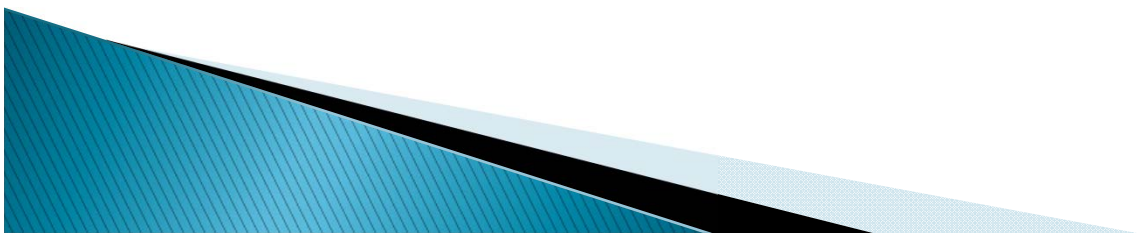
Blood Lactate Level

- An increase in the blood lactate level reflects abnormal cellular function.

- In low-flow states, the primary mechanism of hyperlactatemia is tissue hypoxia with development of anaerobic metabolism

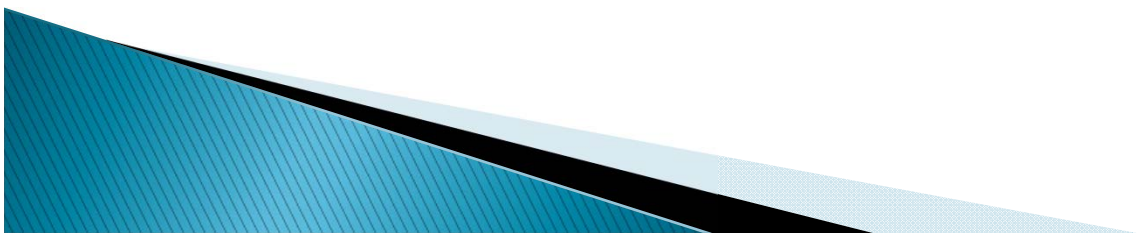


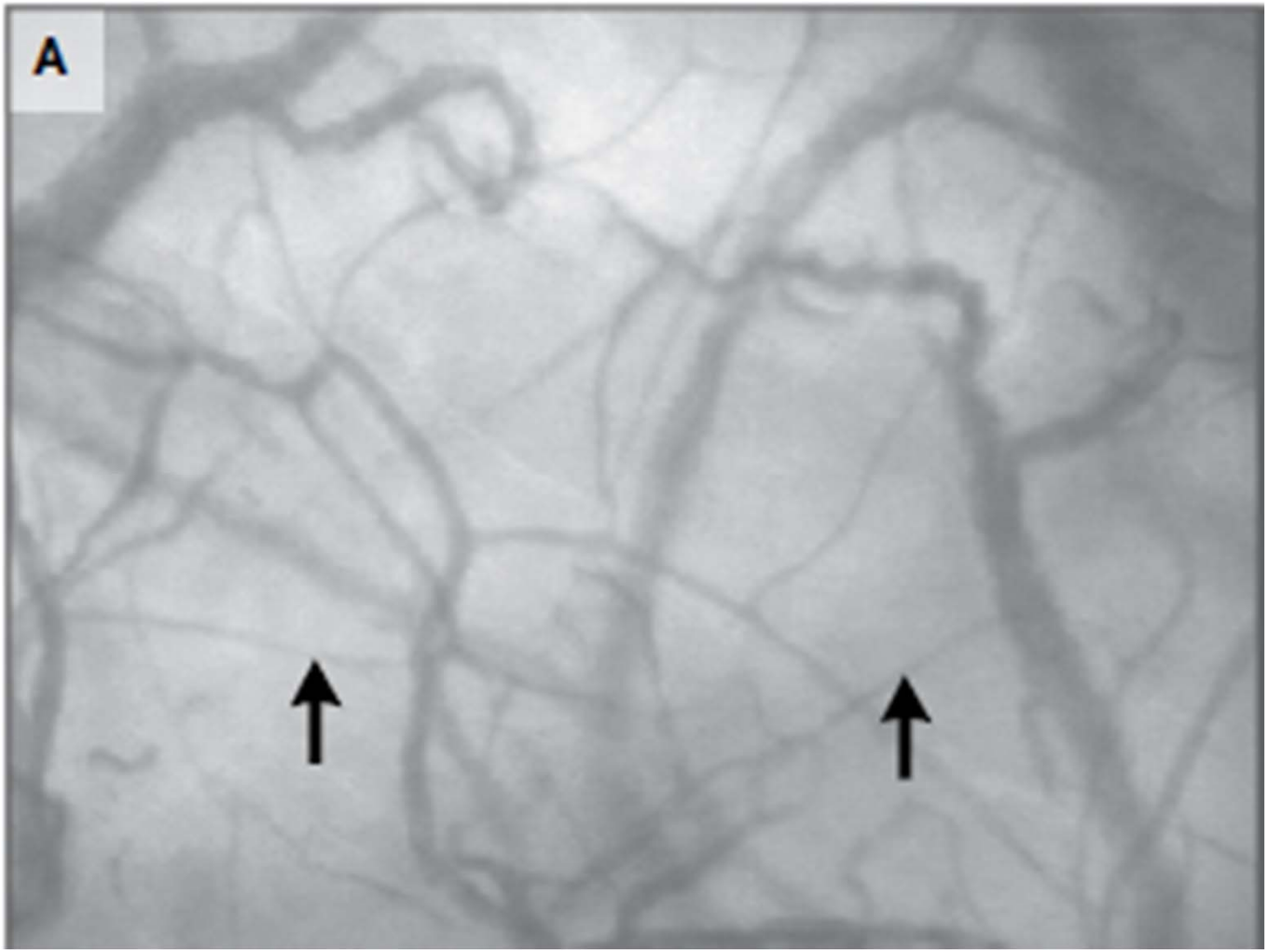
- But in distributive shock, the pathophysiology is more complex.
- In all cases, alterations in clearance can be due to impaired liver function
- the blood lactate level should decrease over a period of hours with effective therapy.

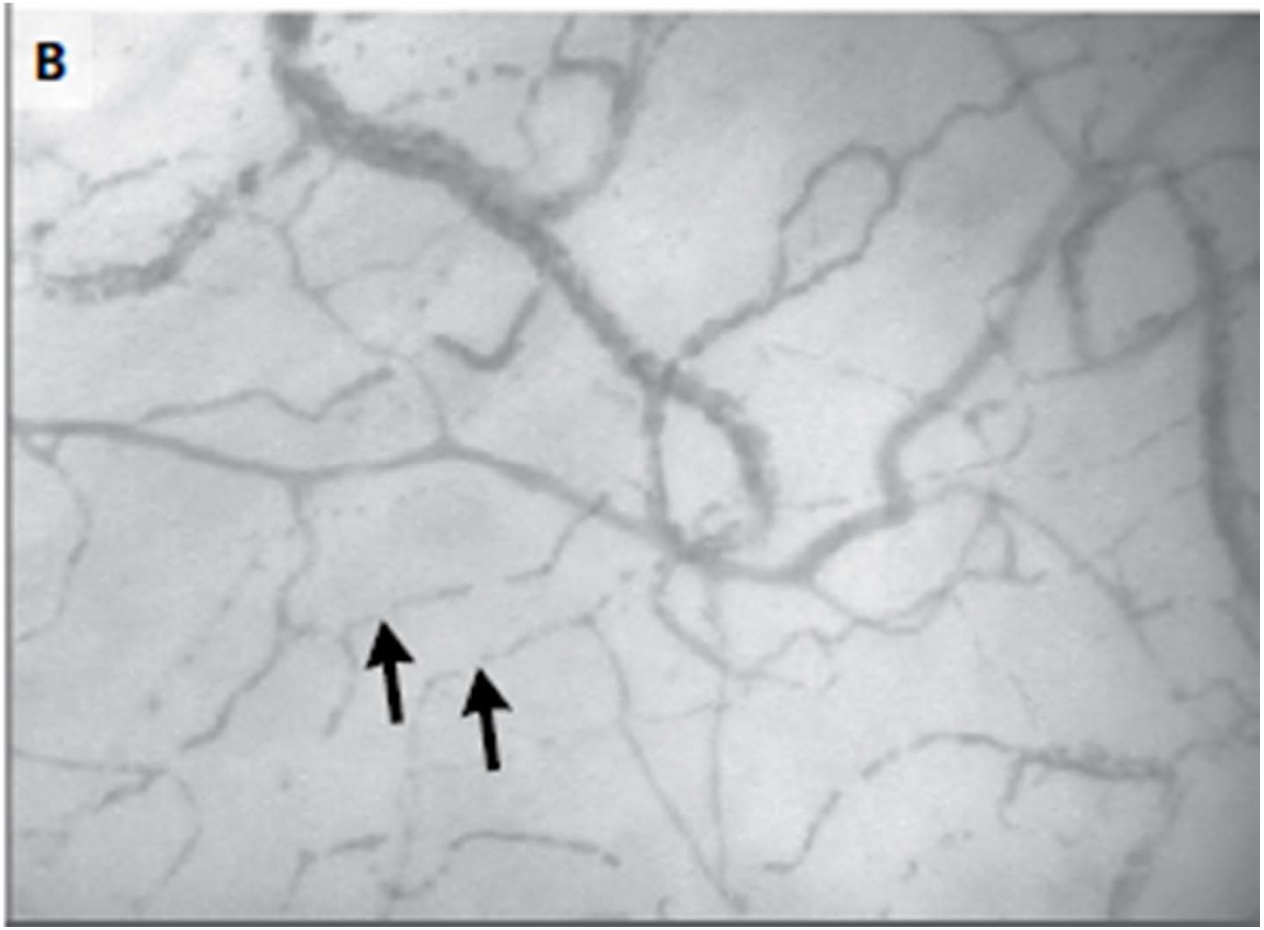


Microcirculatory Variables

- The development of handheld devices for orthogonal polarization spectral (OPS) imaging and its successor sidestream dark-field (SDF) imaging, is providing new means of directly visualizing the microcirculation and evaluating the effects of interventions on microcirculatory flow in easily accessible surfaces, such as the sublingual area

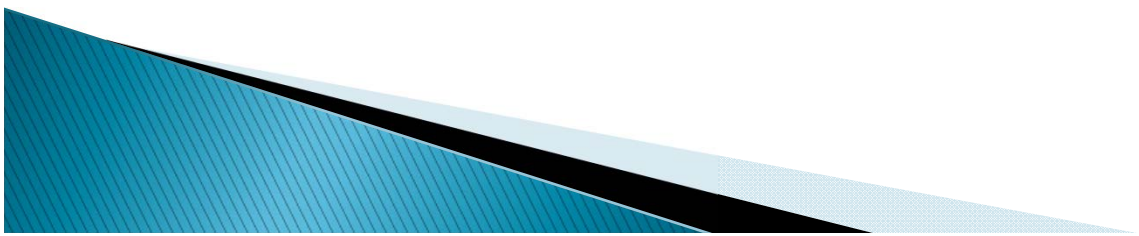



A


B

- Microcirculatory changes,
  - Decreased capillary density,
  - Reduced proportion of perfused capillaries, and
  - Increased heterogeneity of bloodflow

- The persistence of these alterations is associated with worse outcomes.



Therapeutic Priorities and Goals

- Essentially four phases in the treatment of shock

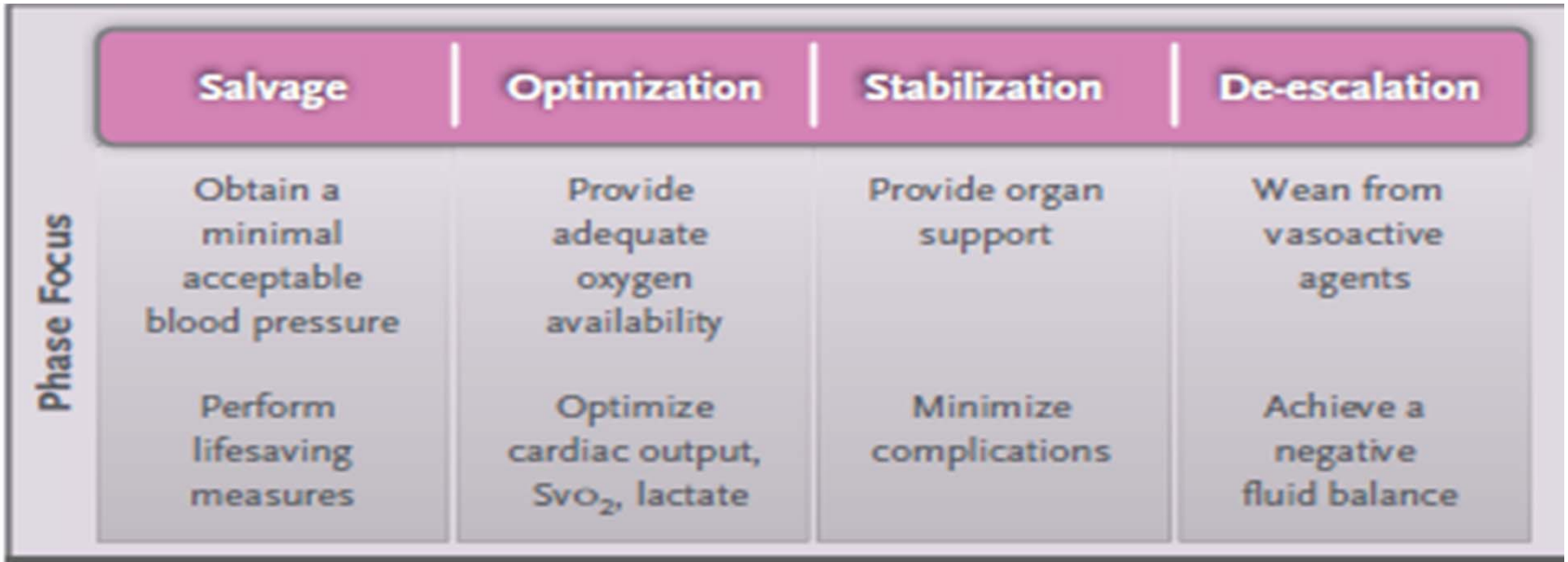
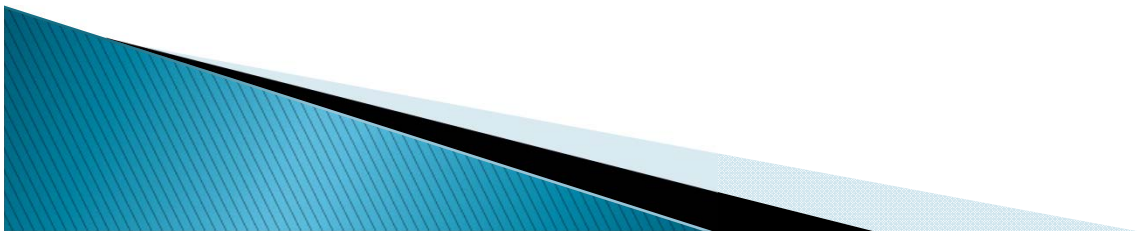


Figure 3. Four Phases in the Treatment of Shock.

The salvage phase focuses on achieving a blood pressure and cardiac output compatible with immediate survival and performing lifesaving procedures to treat the underlying cause of shock. The optimization phase focuses on promoting cellular oxygen availability and monitoring cardiac output, mixed venous oxygen saturation (Svo_2), and lactate levels. The stabilization phase focuses on preventing organ dysfunction, even after hemodynamic stability has been achieved. The de-escalation phase focuses on weaning the patient from vasoactive agents and providing treatments to help achieve a negative fluid balance.

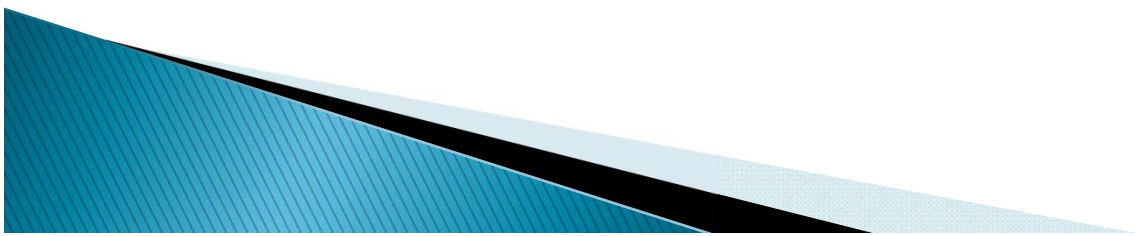
Conclusions

- Circulatory shock is associated with high morbidity and mortality
- Prompt identification is essential so that aggressive management can be started.



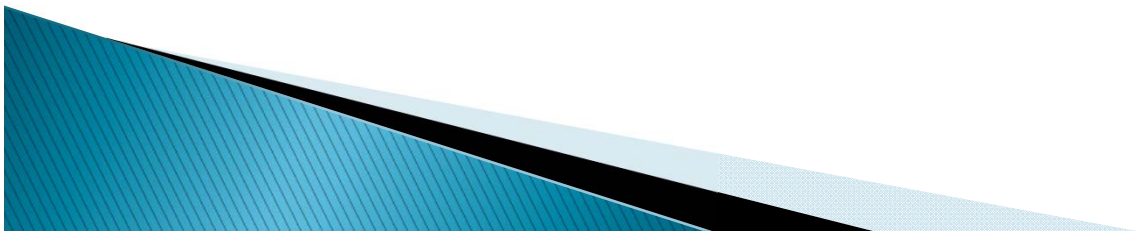
- Appropriate treatment is based on a good ***understanding of the underlying pathophysiological mechanisms***

- Correction of the cause of shock and hemodynamic stabilization, primarily through ***fluid infusion*** and ***administration of vasoactive agents.***



- Monitored by means of careful clinical evaluation and blood lactate measurements.

- Microvascular evaluation may be feasible in the future



THANK YOU