




# PATHOPHYSIOLOGY OF SHOCK

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# Outline

- Case report
  - Definition
  - Classification of shock
  - Pathophysiology
  - Principles of management
- 



# Case Scenario

- 25 year old motorcyclist admitted following an accident.

Confused, pale, cold peripheries

Respiratory rate 30/minute,

Pulse 140/minute, thready

Blood pressure 70/40mmHg


Urine output 10ml/hour

Bleeding from a wound in the thigh






# Case Scenario - Questions

- a) Why is he confused?
  - b) Why are the peripheries cold?
  - c) Why is he tachycardic & hypotensive?
  - d) Why is he oliguric?
- 




# Definition

- Generalized hypoperfusion of tissue cells
  - Results in reduced delivery of O<sub>2</sub> & other nutrients to tissue cells
- 




# Factors Affecting Tissue Perfusion

- The 'pump' – Left ventricle
  - Blood volume
  - Peripheral vascular tone
- 




# The 'Pump' – Left Ventricle

- Cardiac output
  - Stroke volume & Heart rate
  - Volume load of left ventricle - Pre- load
  - Contractility - muscle
  - Afterload
- 



# Blood – the medium

- Plasma
  - Red blood cells
- 





# Peripheral Circulation

- Diversion of blood to vital tissues  
eg. Brain, heart
- Control of diversion
  - Central control
  - Transmission – spinal cord/ peripheral nerves
  - Local control

# Shock- Classification

Shock due to decreased cardiac output

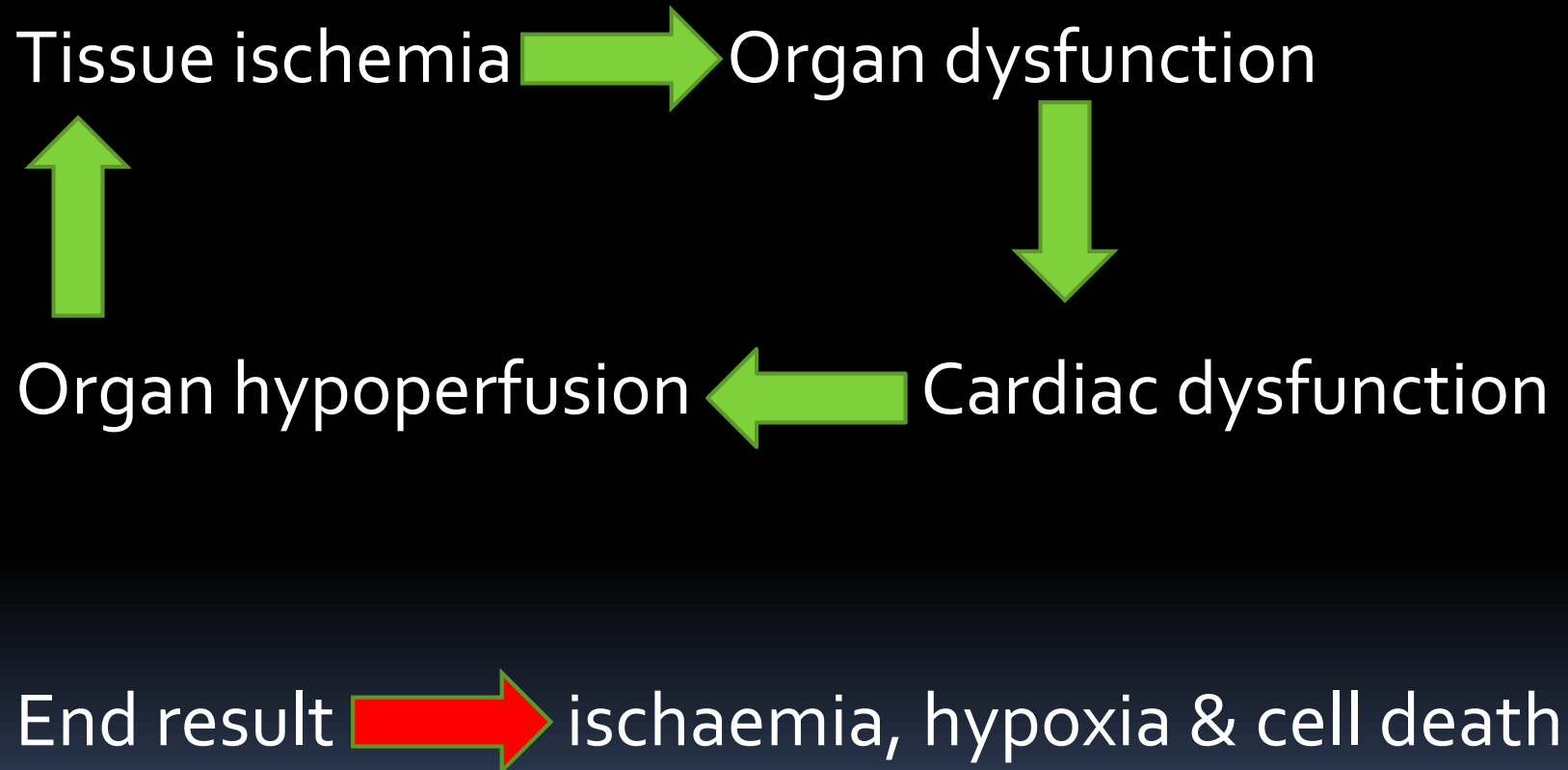
- Failure of the Pump – ‘Cardiogenic shock’
- Failure of the medium – ‘Hypovolaemic shock’

Shock with normal/ increased cardiac output

- Failure of Central control/ transmission –  
‘Neurogenic (Spinal) shock’ eg. Spinal cord injury
- Failure of peripheral control – Anaphylactic  
shock


Septic shock – combination of all of above

# Pathophysiology



# Stages of Shock-Depends on severity & delay in resuscitation

Nonprogressive/ compensated stage –  
Circulatory compensation  Recovery

Progressive  Shock worsens due to inadequate therapy to support compensation

Irreversible  Worsening of shock & death whatever therapy used




# Hypovolaemic Shock

Eg. Haemorrhagic shock

Reduced venous return due to reduced cardiac  
filling pressures

Reduced Cardiac output





# Hypovolemic Shock – Compensation - i

- Goal – maintain perfusion to brain & heart
- Baroreceptor reflex
- Sympathetic system stimulation
- Arteriolar constriction – except in brain & heart
- Venous constriction – Increased venous return
- Tachycardia & increased contractility

# Hypovolemic Shock – Compensation - ii

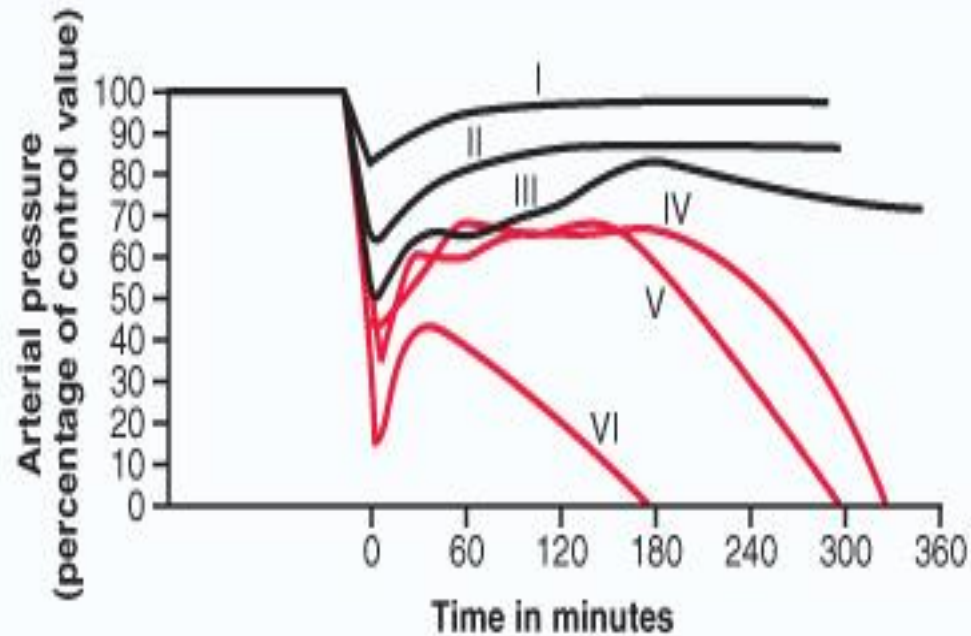
- Fig 24.1

Loss of upto 10% blood volume – no effect on cardiac output & Blood pressure

Loss of > 10% - progressively worsening cardiac output & Blood pressure

Loss >40% blood volume – cardiac output & blood pressure almost zero

# Hemodynamic Changes in Progressive Hypovolaemia



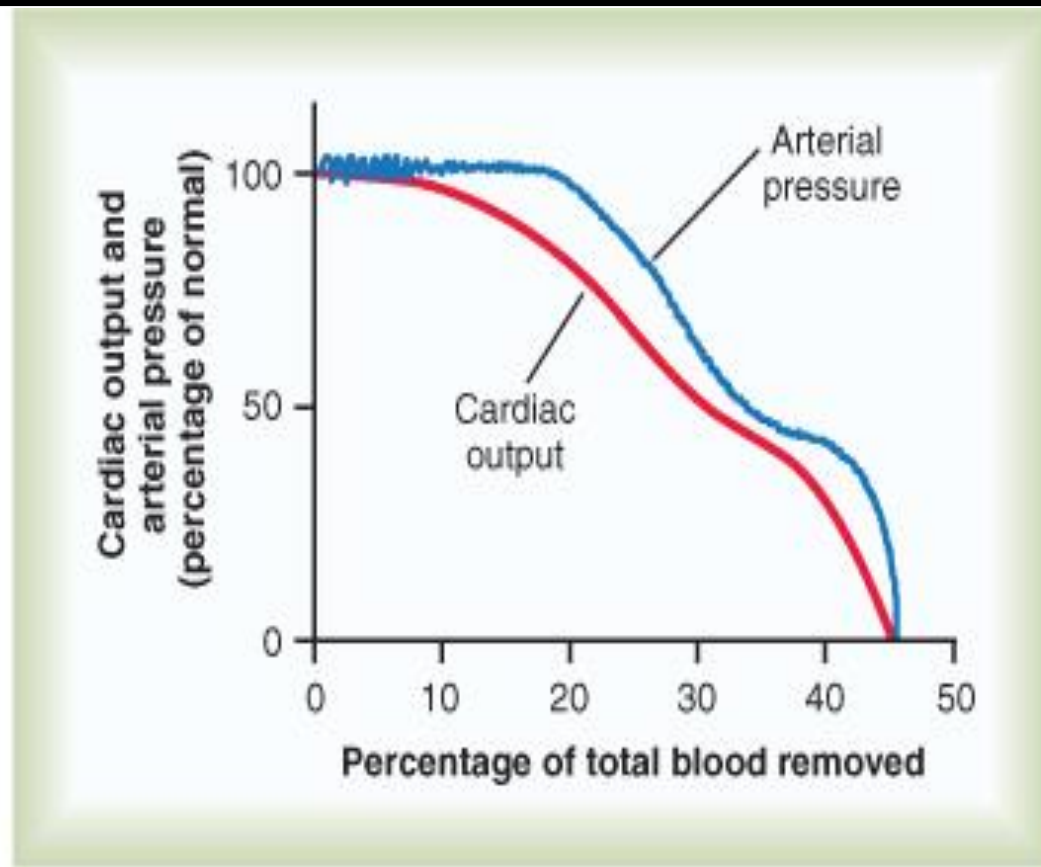


# Hypovolemic Shock – Compensation - iii

Role of the Sympathetic NS – ‘Negative feedback’

- Only 15-20% loss tolerated without sympathetic NS
- More effective in maintaining blood pressure than cardiac output

# Effect of Progressive Hypovolaemia on Cardiac Output & Blood Pressure





# Shock – Changes in the Tissues

- Initial insult – Hypoperfusion
- Compensation – vasoconstriction in 'non vital tissues'

Constriction of pre-capillary sphincters

Reduced flow & hydrostatic pressure in capillaries



Reversal of 'Starling forces'

Absorption of fluid from Interstitial space

# Nonprogressive Shock – Compensation-iv

Compensatory Mechanism	Time to maximum activation
Baroreceptor/ sympathetic MS reflex	30- 60s
Reverse stress relaxation of circulatory system Angiotensin Vasopressin	10min – 1 hour
Reabsorption of interstitial fluid	1 -48 hours
Central nervous system ischaemic response	When SMP less than 50mmHg

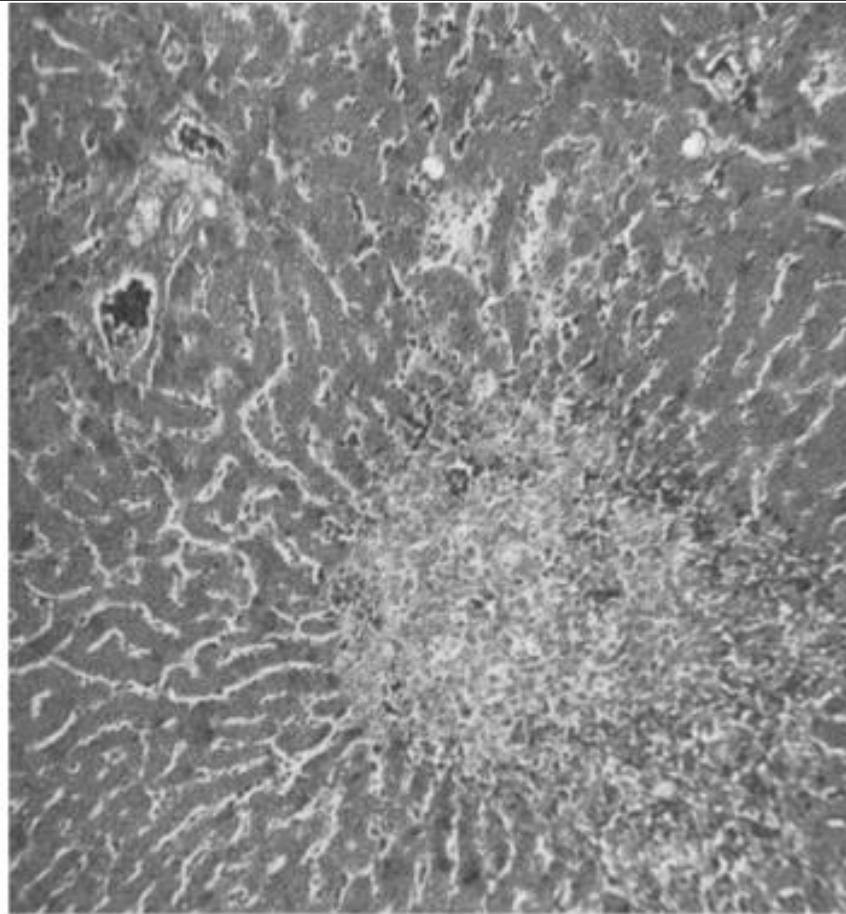
# Hypovolaemic – Progressive Shock - i

- 'Positive feedback'
- Progressive reduction in cardiac output
- Reduction in coronary perfusion & ischemia  
Reduced cardiac output
- Reduced perfusion of brain  
Vasomotor failure – failure to maintain  
sympathetic tone & arteriolar dilatation
- Reduced peripheral perfusion  
'Slow' circulation & micro-clots

# Hypovolaemic – Progressive Shock - ii

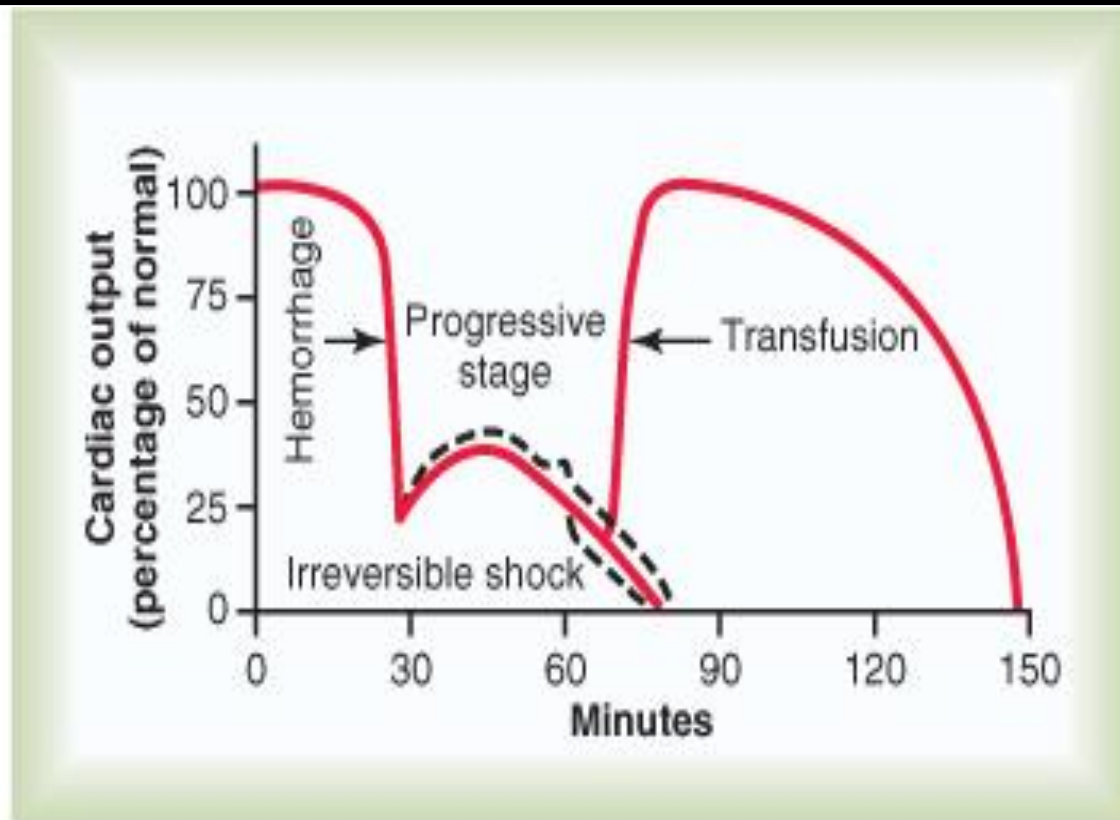
- Increased capillary permeability
- Release of toxins from ischemic tissues eg. Acids,  $K^+$ , histamine, serotonin
- Cellular damage
  - Loss of cell membrane  $Na^+/K^+$  ATPase
  - Reduced mitochondrial activity
  - Lysosomes release lytic enzymes
- Affects all organs eg lungs, heart, liver, kidney

# Liver Injury in Progressive Shock



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# Progression of Reversible Shock to Irreversible Shock








# Hypovolaemic Irreversible Shock

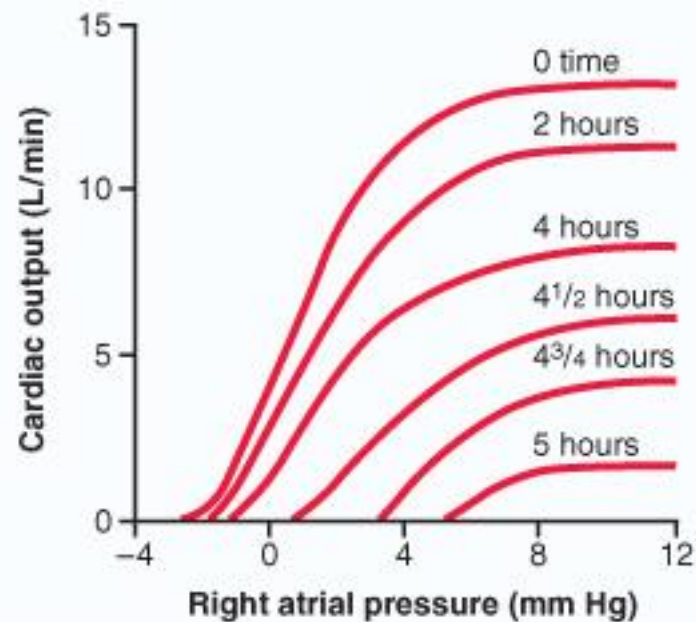
- Positive feedback >>>> negative feedback
  - Cardiac output below a 'Critical level'
  - Worsening tissue hypoxia
- 



# Hypovolaemic Irreversible Shock

- Accumulation of metabolites – vasodilators/toxins
  - Failure of sympathetic mediated vasoconstriction
  - Flow restored to ischaemic tissue
- 

# Irreversible Shock

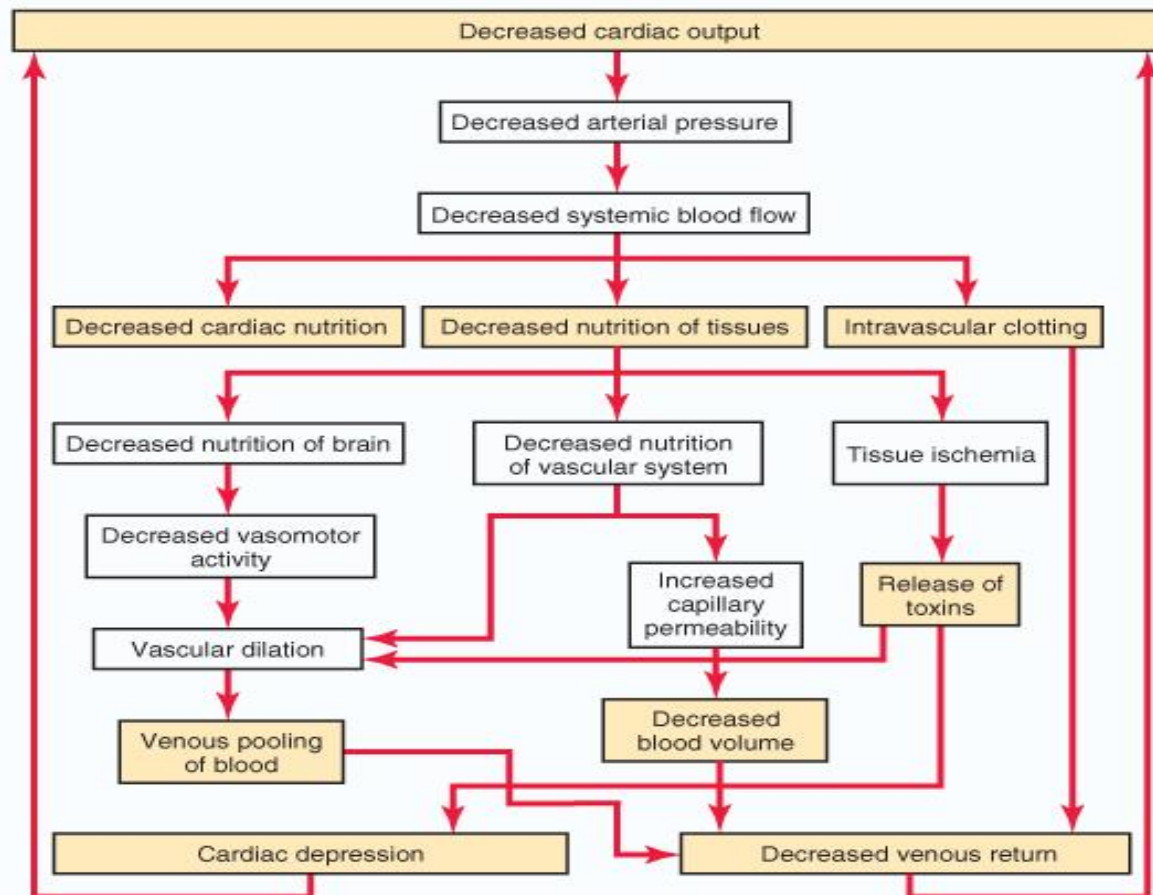


# Hypovolaemic Irreversible Shock

- Metabolites, lytic enzymes, 'carried' away by circulation
- Toxic effects on vital tissues eg. Myocardium, CNS
- Depletion of 'High energy phosphates'


Death!

# Positive Feedback in Progressive & Irreversible Shock






# Anaphylactic Shock

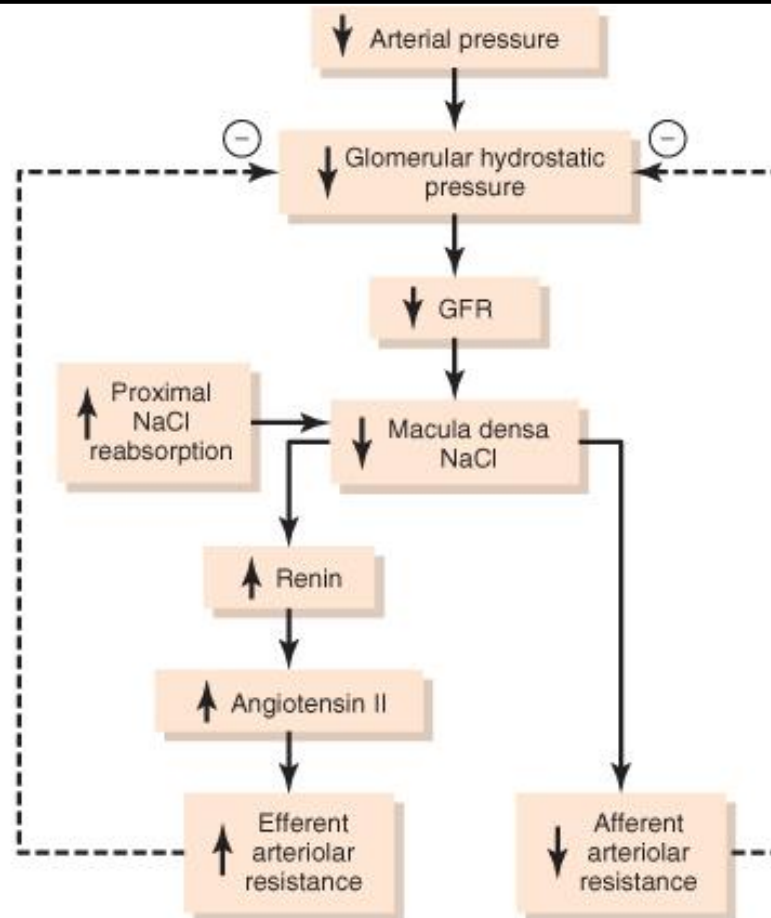
- IgE mediated hypersensitivity – True/false?
  - Mediators include – Histamine – True/False?
  - Veno-dilatation - True/False?
  - Arteriolar dilatation – True/False?
  - Increased Capillary permeability – True/False?
- 



# Case scenario -Answers

- a) Why is he confused?
  - b) Why are the peripheries cold?
  - c) Why is he tachycardic & hypotensive?
  - d) Why is he oliguric?
- 

# Why is he oliguric?







# Summary

- Shock
  - Definition
  - Classification – Normal/ Abnormal cardiac output
  - Stages of shock –
    - Non Progressive/ compensated
    - Progressive
    - Irreversible
  - Pathophysiology
- 