HAEMODYNAMICS OF SHOCK

Dr.G.Rajalakshmi
2nd Year P.G
Dept of Physiology

OBJECTIVES

- Definition
- Types and causes of shock
- Stages of shock
- Compensatory mechanisms to overcome shock
- Summary

- Shock is defined as a syndrome characterized by serious reduction of tissue perfusion with a relatively or absolutely inadequate cardiac output.
- Shock is a condition characterized by inadequate delivery of oxygen and nutrients to critical organs such as heart, brain, liver and kidneys.

TYPES AND CAUSES OF SHOCK

Depending on the cause of inadequacy of cardiac output it may be of following types;

I. Hypovolaemic shock
II. Low-resistance or distributive or vasogenic shock
III. Cardiogenic shock
IV. Obstructive shock

HYPOVOLAEMIC SHOCK

- Is also known as cold shock & is caused by low blood volume resulting in decreased cardiac output.
- Depending on the causes it may of following types;

1. Haemorrhagic shock
2. Dehydration shock
3. Traumatic shock

LOW-RESISTANCE SHOCK

- Occurs when neural reflexes or toxic substances cause excessive vasodilation within the vascular system.
- Due to vasodilation the size of capacitance vessels is increased & cardiac output is decreased inspite of normal blood volume.
- Also known as warm shock.
- Depending on the causes it may of following types;

1. Neurogenic shock
2. Anaphylactic shock
3. Septicaemic shock
4. Endotoxic shock

CARDIOGENIC SHOCK

- Occurs due to decreased pumping ability of the heart because of some cardiac abnormality.
- Heart is not able to pump out all the venous return so there occurs congestion of lungs and viscera, so also known as congested shock.
- Causes of cardiogenic shock are;

1. Myocardial infarction
2. Cardiac arrhythmias
3. Congestive heart failure
4. Severe valvular dysfunction

OBSTRUCTIVE SHOCK

- Occurs due to impairment of ventricular filling during diastole due to some external pressure on the heart.
- Due to decreased ventricular filling the stroke volume and the cardiac output is decreased causing shock.
- Causes of obstructive shock are;

1. Pericardial cardiac tamponade
2. Tension pneumothorax
3. Constrictive pericarditis
4. Pulmonary embolism

STAGES OF SHOCK

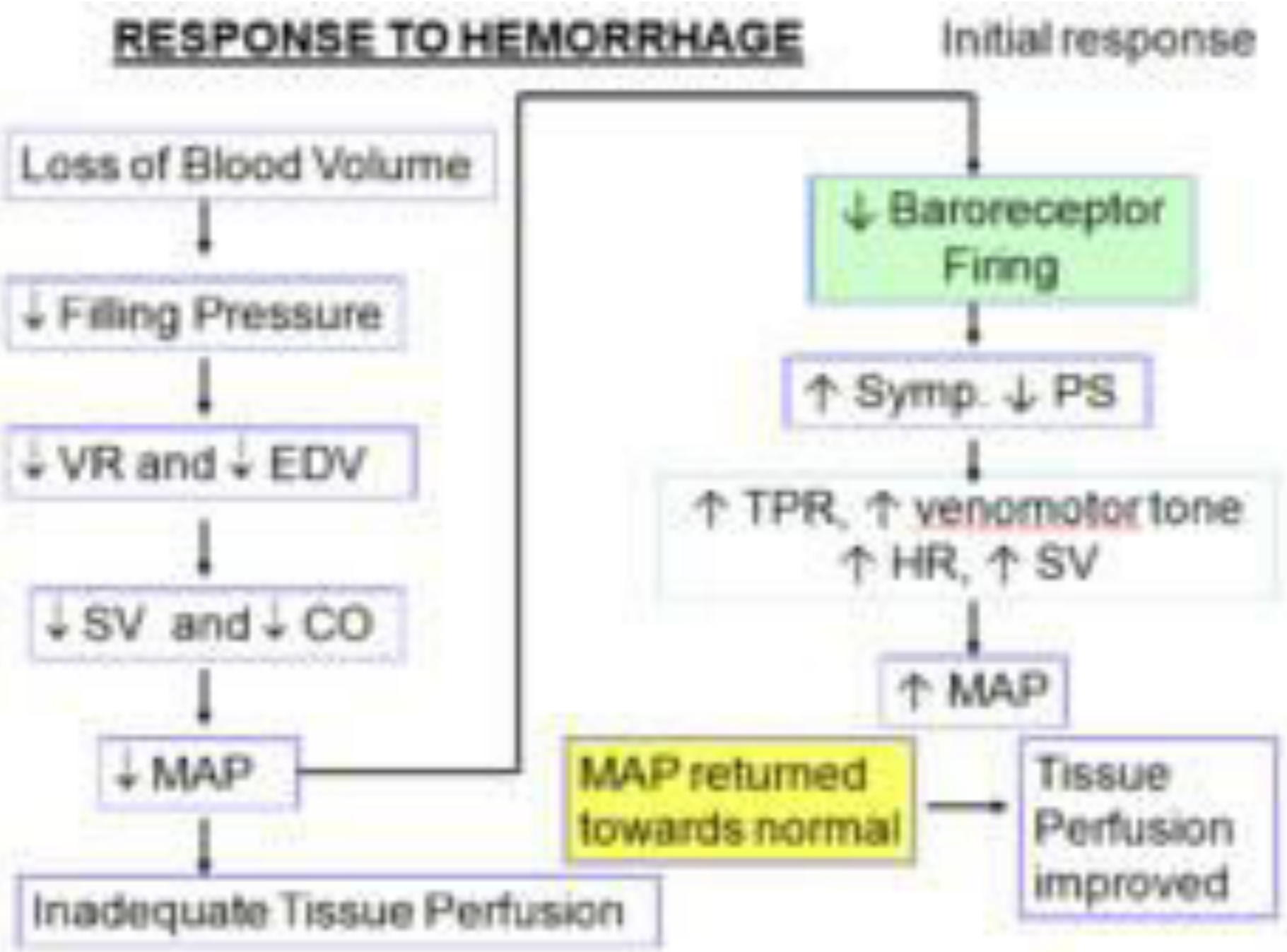
Depending on the severity shock can be divided into 3 stages;

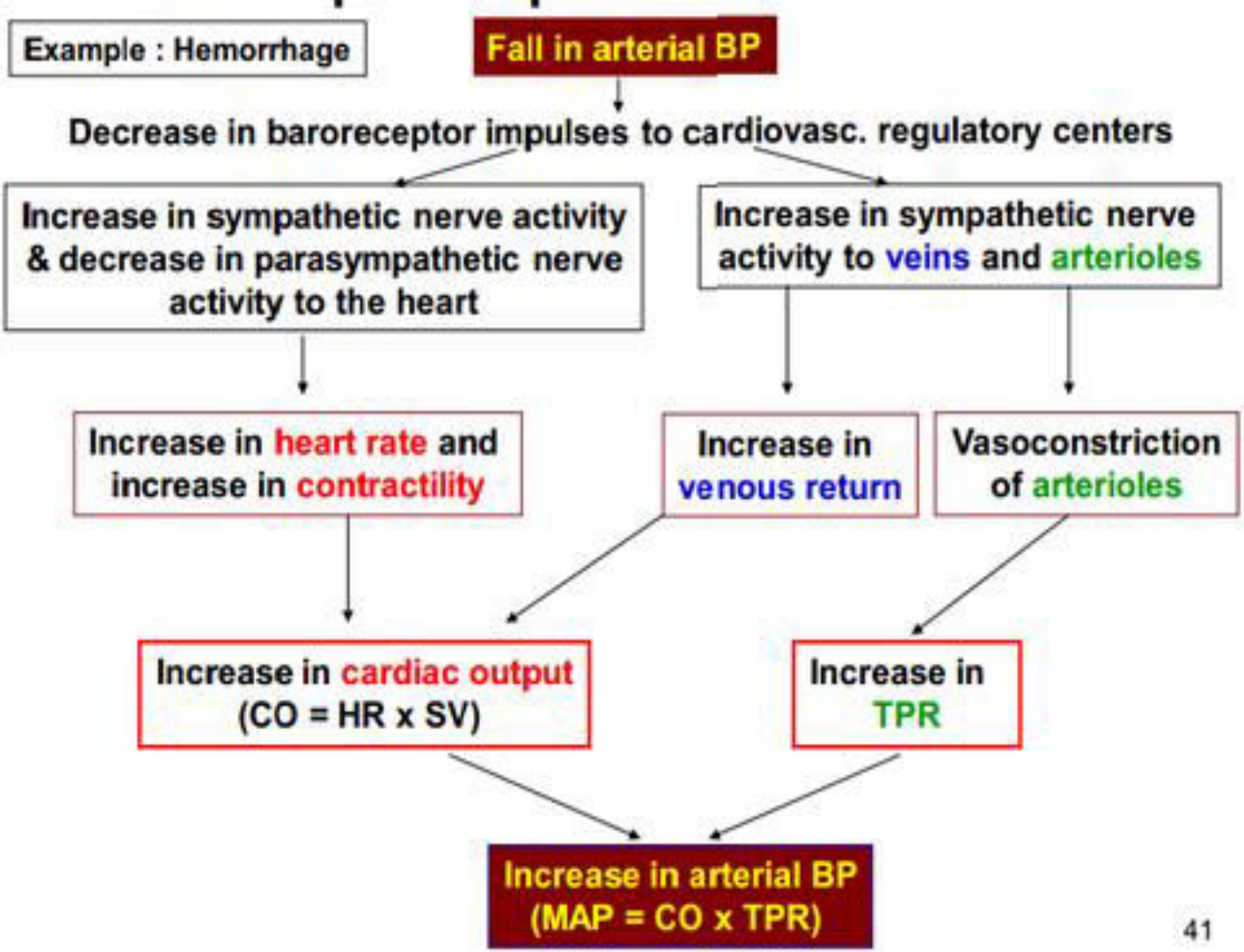
1. Non – progressive shock: compensated shock in which normal circulatory compensatory mechanisms cause full recovery.
2. Progressive shock: in which, without therapy the shock becomes worse until death.
3. Refractory shock: in which the shock has progressed to such an extent that all forms of known therapy are inadequate to save the person's life, even though, for the moment, the person is still alive.

Compensatory reactions activated by hemorrhage

- Vasoconstriction
- Tachycardia
- Increased movement of interstitial fluid into capillaries
- Increased secretion of norepinephrine & epinephrine
- Increased secretion of vasopressin
- Increased secretion of glucocorticoids
- Increased secretion of renin & aldosterone
- Increased secretion of erythropoietin
- Increased plasma protein synthesis

RESPONSE TO HEMORRHAGE

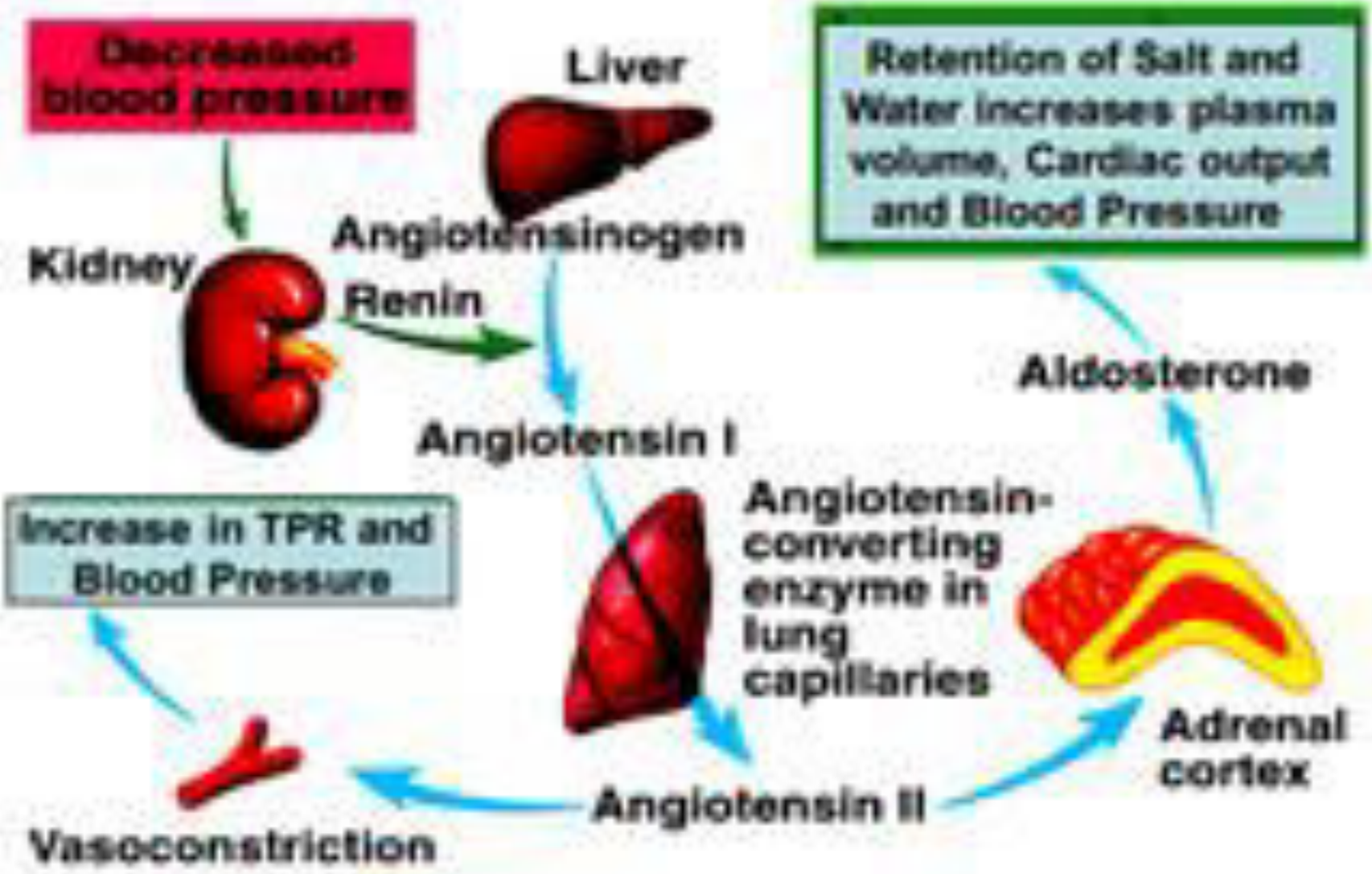



Example : Hemorrhage
Fall in arterial BP
Decrease in baroreceptor impulses to cardiovasc. regulatory centers
Increase in sympathetic nerve activity & decrease in parasympathetic nerve activity to the heart
Increase in sympathetic nerve activity to veins and arterioles
Increase in heart rate and increase in contractility
Increase in venous return
Vasoconstriction of arterioles
Increase in cardiac output (CO = HR x SV)
Increase in TPR
Increase in arterial BP (MAP = CO x TPR)

- Central nervous system ischemic response: when BP falls below 50mm Hg, blood flow to vasomotor area in the brainstem decreases to cause CNS ischaemia.
- As a result CO2/ lactic acid are accumulated locally near VMC and excite the neurons of VMC strongly.
- Excitation of VMC causes strong sympathetic stimulation leading to vasoconstriction.
- There occurs immediate increase in BP.
- This acts as an emergency arterial pressure control system.

- Increased secretion by the adrenal medullae of epinephrine and norepinephrine, which constricts the peripheral arteries and veins and increases the heart rate.
- The increase in circulating catecholamines may lead to stimulation of reticular formation causing restlessness and apprehension in some patients.

Regulation of arterial BP by Renin-angiotensin-aldosterone mechanism



- Reverse stress-relaxation of the circulatory system: when BP falls due to prolonged slow bleeding,there occurs tightening of blood vessel walls by vascular tone adjustment secondary to less stress on the vessel wall.
- This mechanism tries to restore the BP back to normal.
- Capillary fluid shift mechanism: when BP is low, the mean capillary pressure is also low, resulting in absorption of fluid from interstitial compartment to circulation.
- Thus the blood volume is increased which helps to return the BP back to normal.

Long-term compensatory mechanisms

- Restoration of plasma volume and proteins: after a moderate haemorrhage the plasma volume is restored to normal in 12 to 72 hrs.
- Plasma proteins are restored by hepatic synthesis over a period of 3-4 days.
- Restoration of red cell mass: there is excess release of erythropoietin which increases the rate of cell production in bone marrow within 10 days.
- Normal cell mass is restored in 4 to 8 wks.

Progressive shock

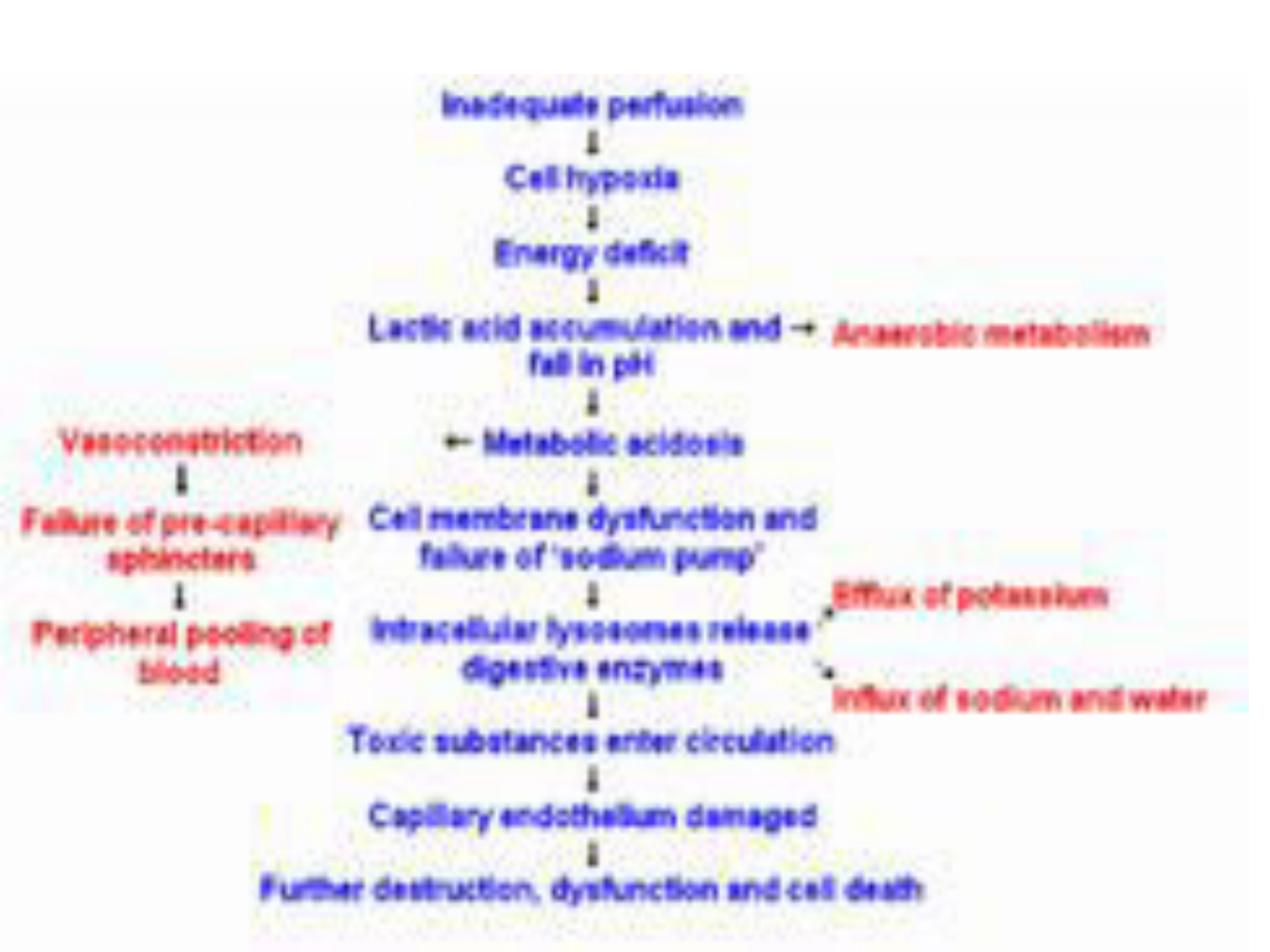
- This occurs when there is 15 to 25% loss of total blood volume.
- In this stage the compensatory mechanisms are not able to stop the progression of shock.
- The structures of circulatory system begin to deteriorate and various types of positive feedback mechanisms develop.
- Timely therapeutic interventions are essential in this stage, otherwise the vicious cycle of positive feedback mechanisms cause progressive decrease in the cardiac output and pt will go into stage of refractory shock.

- Cardiac depression: When the arterial pressure falls low enough, coronary blood flow decreases below that required for adequate nutrition of the myocardium.
- This weakens the heart muscle and decreases the cardiac output more.
- Thus, a positive feedback cycle develops, whereby the shock becomes more and more severe.
- Endotoxin is released from the bodies of dead gram-negative bacteria in the intestines.

- Vasomotor failure: diminished blood flow to the brain's vasomotor center depresses the center so much that it, becomes progressively less active and finally totally inactive.
- For example, when there is complete circulatory arrest to the brain during the first 4 to 8 minutes, there occurs the most intense of all sympathetic discharges, but by the end of 10 to 15 minutes, the vasomotor center becomes so depressed that no further evidence of sympathetic discharge can be demonstrated.

- Blockage of small vessels: initiating cause of blockage is sluggish blood flow in the microvessels.
- Because tissue metabolism continues despite the low flow, large amounts of acid, both carbonic acid and lactic acid, continue to empty into the local blood vessels and greatly increase the local acidity of the blood.
- This acid, plus other deterioration products from the ischemic tissues, causes local blood agglutination, resulting in minute blood clots, leading to very small plugs in the small vessels.
- Even if the vessels do not become plugged, an increased tendency for the blood cells to stick to one another makes it more difficult for blood to flow through the microvasculature.

- Increased capillary permeability: because of capillary hypoxia and lack of other nutrients, the permeability of the capillaries gradually increases, and large quantities of fluid begin to transude into the tissues.
- This decreases the blood volume even more, with a resultant further decrease in cardiac output, making the shock still more severe.
- Release of toxins by ischemic tissue: shock causes tissues to release toxic substances, such as histamine, serotonin, and tissue enzymes, that cause further deterioration of the circulatory system.


Inadequate perfusion
Cell hypoxia
Energy deficit
Lactic acid accumulation and fall in pH
Anaerobic metabolism
Metabolic acidosis
Vasoconstriction
Failure of pre-capillary sphincters
Peripheral pooling of blood
Cell membrane dysfunction and failure of 'sodium pump'
Efflux of potassium
Intracellular lysosomes release digestive enzymes
Influx of sodium and water
Toxic substances enter circulation
Capillary endothelium damaged
Further destruction, dysfunction and cell death

- Tissue necrosis: Not all cells of the body are equally damaged by shock because some tissues have better blood supply than others.
- The cells adjacent to the arterial ends of capillaries receive better nutrition than cells adjacent to the venous ends of the same capillaries.
- Therefore, more nutritive deficiency occurs around the venous ends of capillaries than elsewhere.
- Punctate lesions occur in heart muscle.

- The cardiac lesions play an important role in leading to the final irreversible stage of shock.
- Deteriorative lesions also occur in the kidneys, especially in the epithelium of the kidney tubules, leading to kidney failure and occasionally uremic death several days later.
- Deterioration of the lungs also often leads to respiratory distress and death several days later—called the shock lung syndrome.

Refractory shock

- When shock is in progressive stage and is not treated adequately, a vicious cycle of various positive feedback mechanisms set in and pt passes into third stage of shock, the refractory shock.
- In this stage all therapeutic interventions are usually ineffective and eventually pt dies.

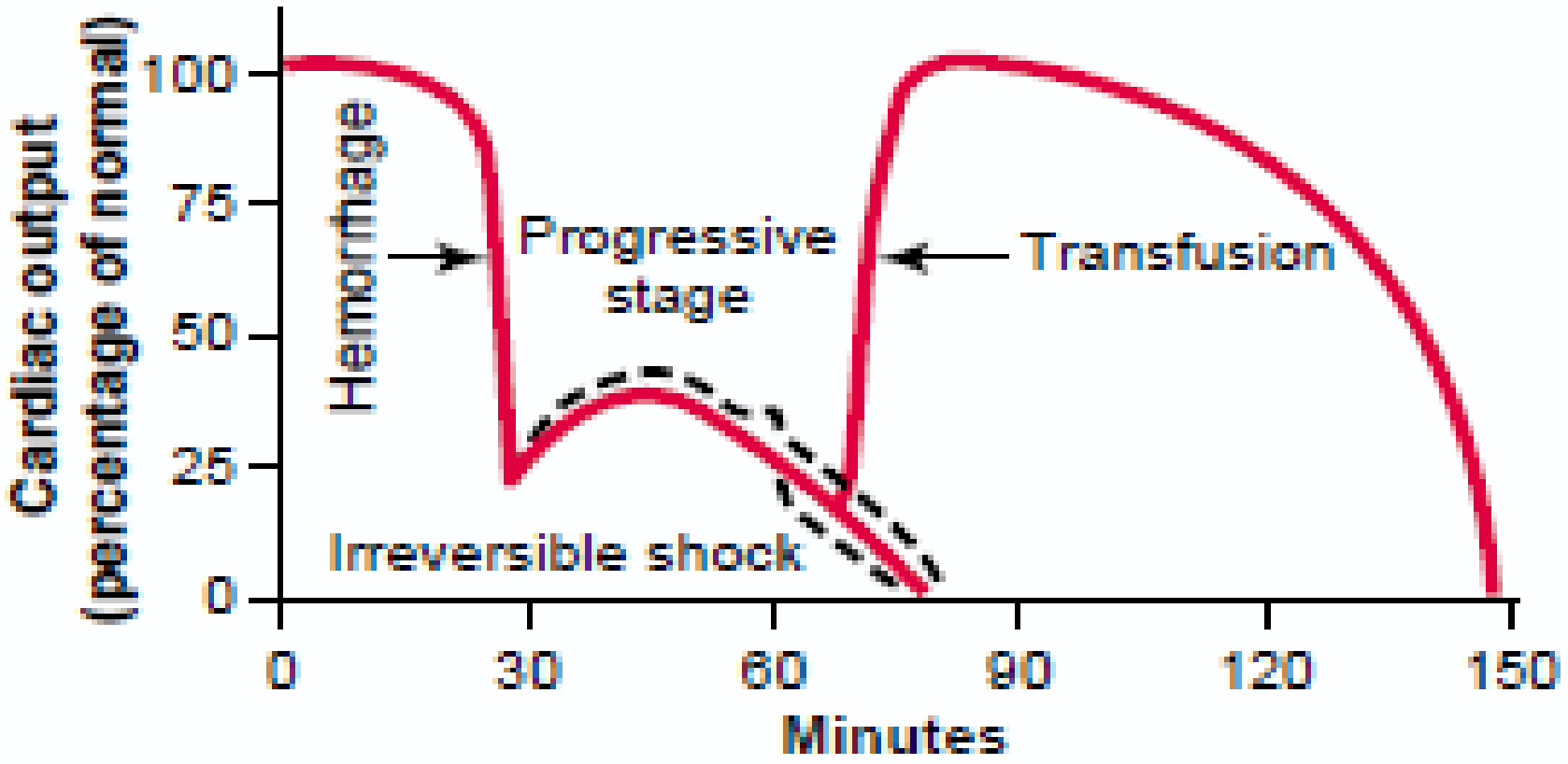
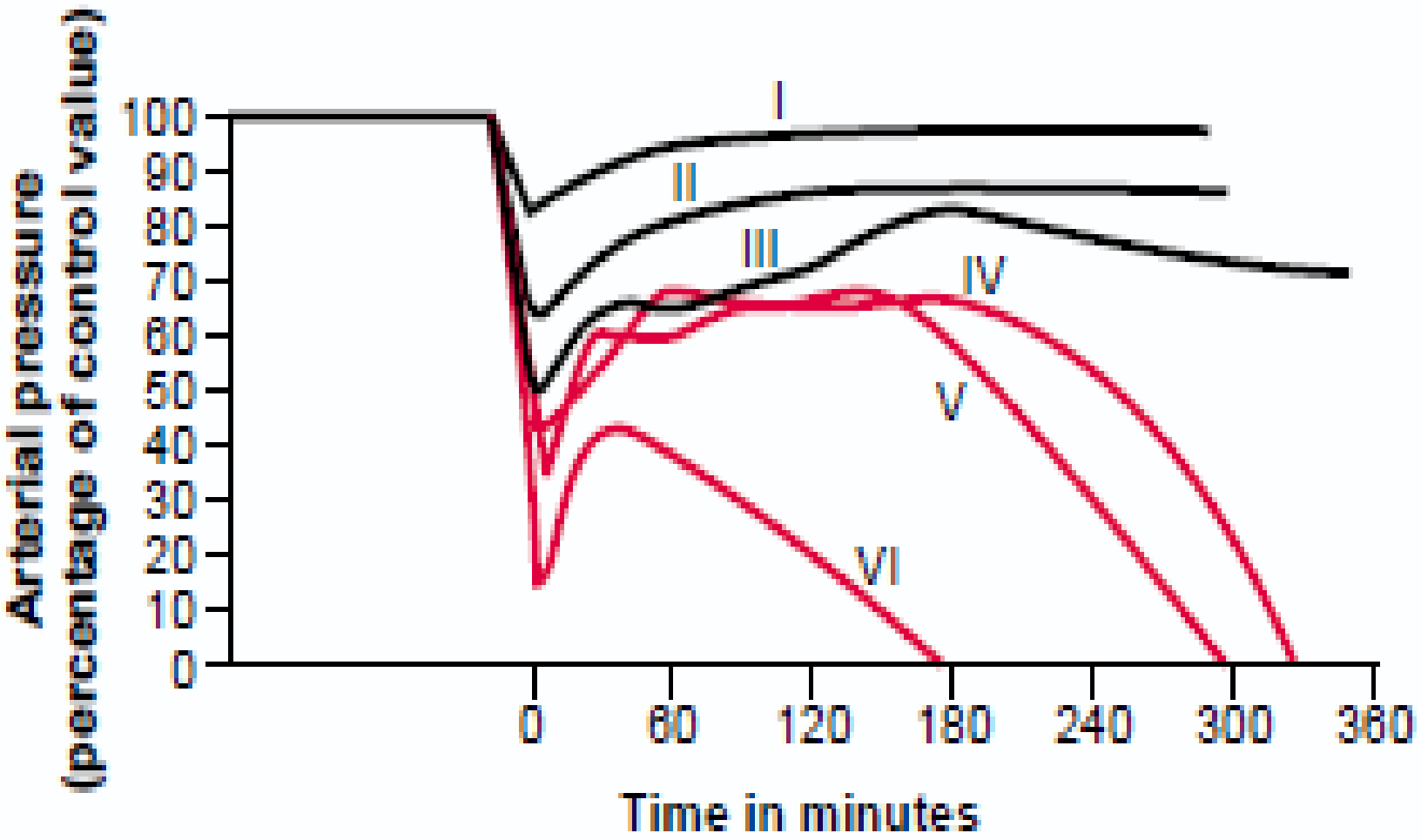


Figure 24–6

Failure of transfusion to prevent death in irreversible shock.

- The high-energy phosphate reserves in the tissues of the body are greatly diminished in severe degrees of shock.
- All the creatine phosphate is degraded, and almost all the adenosine triphosphate is downgraded to adenosine diphosphate, adenosine monophosphate, and, eventually, adenosine.
- This adenosine diffuses out of the cells into the circulating blood and is converted into uric acid, a substance that cannot re-enter the cells to reconstitute the adenosine phosphate system.

- New adenosine can be synthesized at a rate of only about 2 percent of the normal cellular amount an hour, meaning that once the high-energy phosphate stores of the cells are depleted, they are difficult to replenish.
- Thus, one of the most devastating end results of deterioration in shock, and the one that is perhaps most significant for development of the final state of irreversibility, is this cellular depletion of these high-energy compounds.

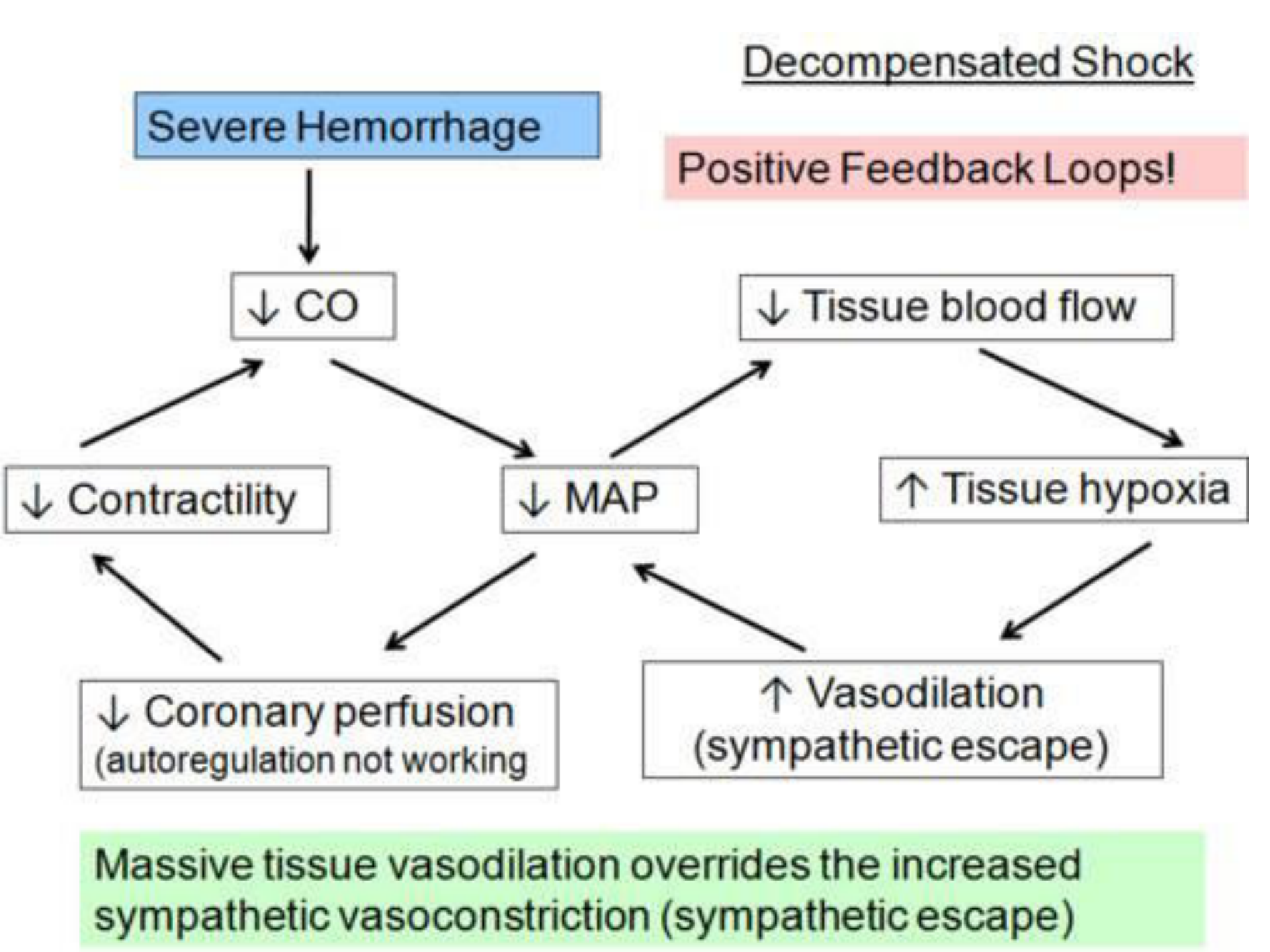

Arterial pressure
(percentage of control value)
100
90
80
70
60
50
40
30
20
10
0
I
II
III
IV
V
VI
0
60
120
180
240
300
360
Time in minutes

Summary

- Shock is defined as a syndrome characterized by relative or absolute inadequate cardiac output with inadequate delivery of oxygen & nutrients to critical organs.
- Hypovolaemic shock due to haemorrhage is most common type of shock.
- The haemodynamic changes that occur are low blood volume, decrease in arterial pressure and cardiac output.
- There are rapid, intermittent and long term physiologic compensatory mechanisms to overcome shock in early stages.
- Timely therapeutic interventions in progressive stage of shock is essential to prevent progression of shock into refractory stage.

References

- Guyton and Hall Textbook of Medical Physiology 12th Edition
- Ganong's Review of Medical Physiology 24th Edition
- Best and Taylor's Physiological Basis of Medical Practice 13th Edition

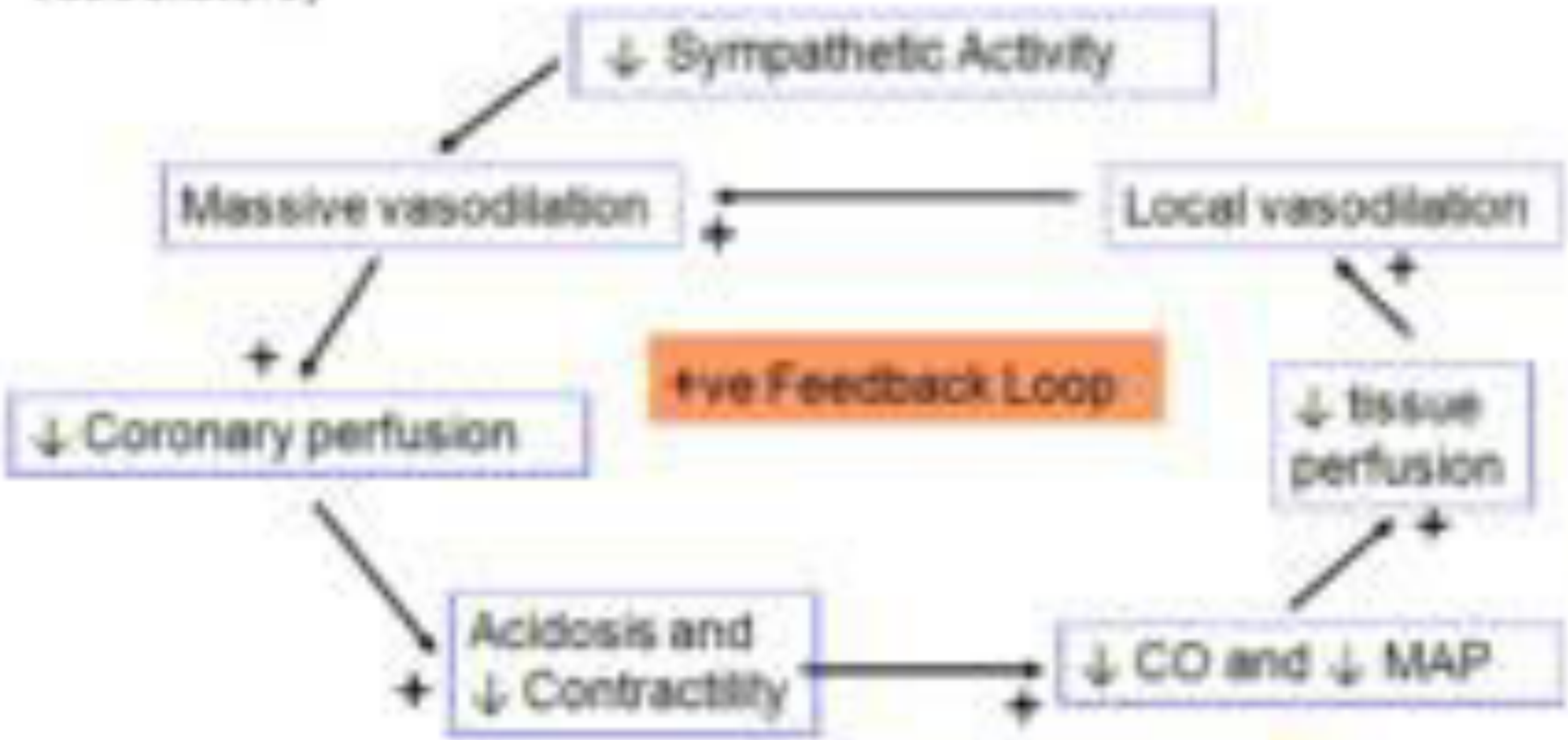

Decompensated Shock
Severe Hemorrhage
Positive Feedback Loops!
↓ CO
↓ Tissue blood flow
↓ Contractility
↓ MAP
↑ Tissue hypoxia
↓ Coronary perfusion
(autoregulation not working
↑ Vasodilation
(sympathetic escape)
Massive tissue vasodilation overrides the increased sympathetic vasoconstriction (sympathetic escape)

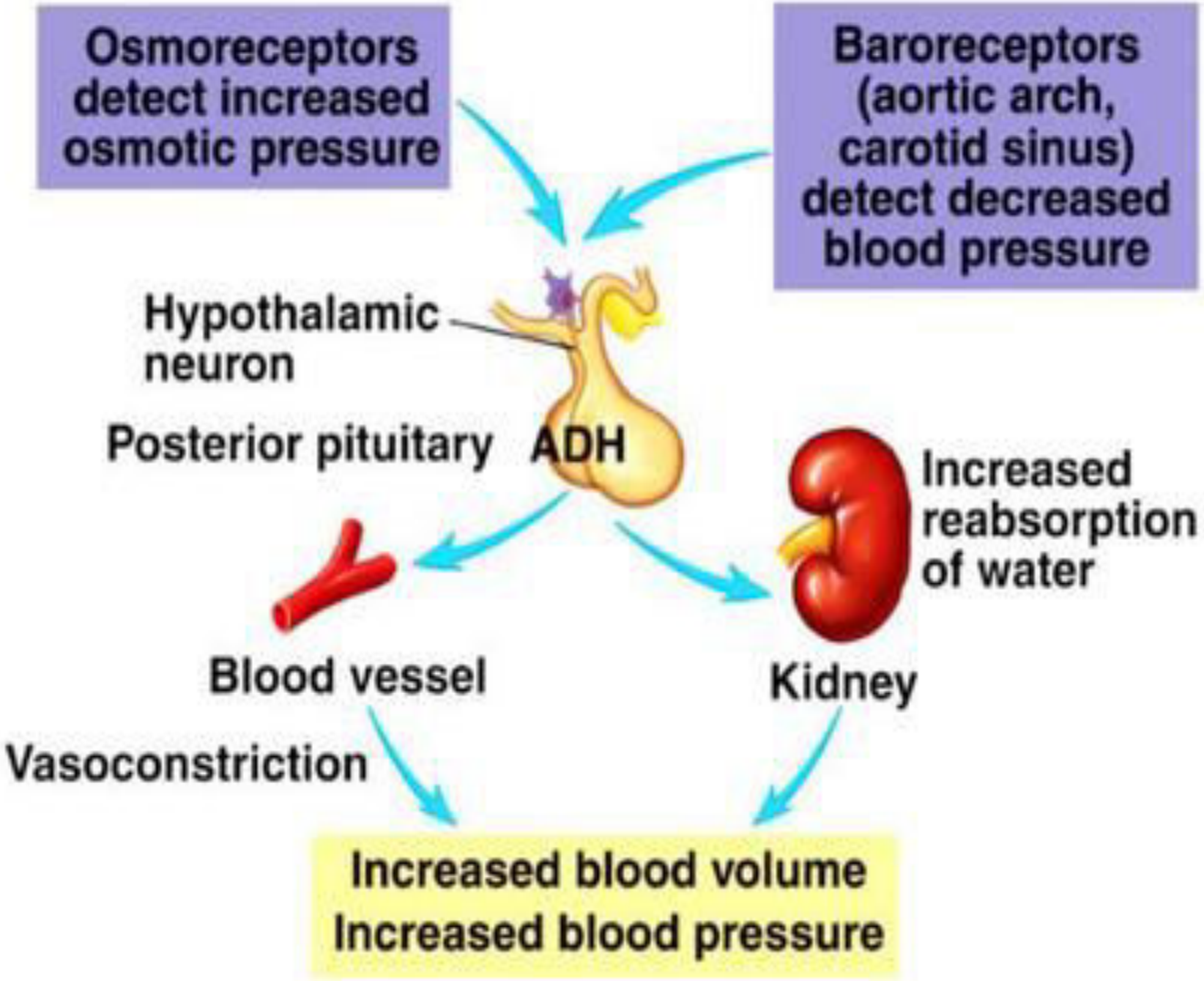
Severe, Prolonged Shock: Decompensated Shock

Occurs: Loss of > 30% blood volume, no fluid replacement for 3 – 4 hr

Initially: ↑ sympathetic activity (BP may be maintained)

Later: ↓ Fall in effective sympathetic activity (overridden by metabolic vasodilators)




Osmoreceptors detect increased osmotic pressure
Baroreceptors (aortic arch, carotid sinus) detect decreased blood pressure
Hypothalamic neuron
Posterior pituitary
ADH
Increased reabsorption of water
Blood vessel
Kidney
Vasoconstriction
Increased blood volume
Increased blood pressure

Intermediate compensatory mechanisms

- Increased secretion of renin by the kidneys and formation of angiotensin II, which constricts the peripheral arteries and also causes decreased output of water and salt by the kidneys, both of which help prevent progression of shock.
- Increased secretion by the posterior pituitary gland of vasopressin (antidiuretic hormone), which constricts the peripheral arteries and veins and greatly increases water retention by the kidneys.

- Generalized cellular deterioration: One organ especially affected is the liver.
- This occurs because of lack of enough nutrients to support the normally high rate of metabolism in liver cells, but also partly because of the exposure of the liver cells to any vascular toxin or other abnormal metabolic factor occurring in shock.
- Some of the damaging cellular effects are the following:
- Active transport of sodium and potassium through the cell membrane is greatly diminished.
- As a result, sodium and chloride accumulate in the cells and potassium is lost from the cells and the cells begin to swell.

- Mitochondrial activity in the liver cells, as well as in other tissues of the body, becomes severely depressed.
- Lysosomes in the cells in widespread tissue areas begin to break open, with intracellular release of hydrolases that cause further intracellular deterioration.
- Cellular metabolism of nutrients, such as glucose, becomes greatly depressed in the last stages of shock.

- The actions of some hormones are depressed including almost 100 percent depression of the action of insulin.
- All these effects contribute to further deterioration of many organs of the body, including especially

(1) liver, with depression of its many metabolic and detoxification functions;

(2) lungs, with eventual development of pulmonary edema and poor ability to oxygenate the blood; and

(3) heart, further depressing its contractility.

- Acidosis : Most metabolic derangements that occur in shock can lead to acidosis all through the body.
- This results from poor delivery of oxygen to the tissues, which greatly diminishes oxidative metabolism of the foodstuffs.
- The cells obtain most of their energy by the anaerobic process of glycolysis, which leads to tremendous quantities of excess lactic acid in the blood.

- Poor blood flow through tissues prevents normal removal of carbon dioxide.
- The carbon dioxide reacts locally in the cells with water to form high concentrations of intracellular carbonic acid; this reacts with various tissue chemicals to form still other intracellular acidic substances.
- There is both generalized and local tissue acidosis, leading to further progression of the shock itself

- Diminished blood flow to the intestines often causes enhanced formation and absorption of this toxic substance.
- The circulating toxin causes increased cellular metabolism despite inadequate nutrition of the cells; this has a specific effect on the heart muscle, causing cardiac depression.