SHOCK

Piťha J.

Contents:

DEFINITION:

- ***Shock = syndrome***
- **Failure of hear pump and/or (harmonisation/coordination) of peripheral vasculature leading to critically limited blood flow – oxygenation/nutrition of peripheral tissues and to their dysfunction on the cellular level (**pyruvate x lactate, mitochondria x cytoplasm).

Classification:

- **Distributive**
- **Cardiogenic**
- **Hypovolemic**
- **Obstructive**

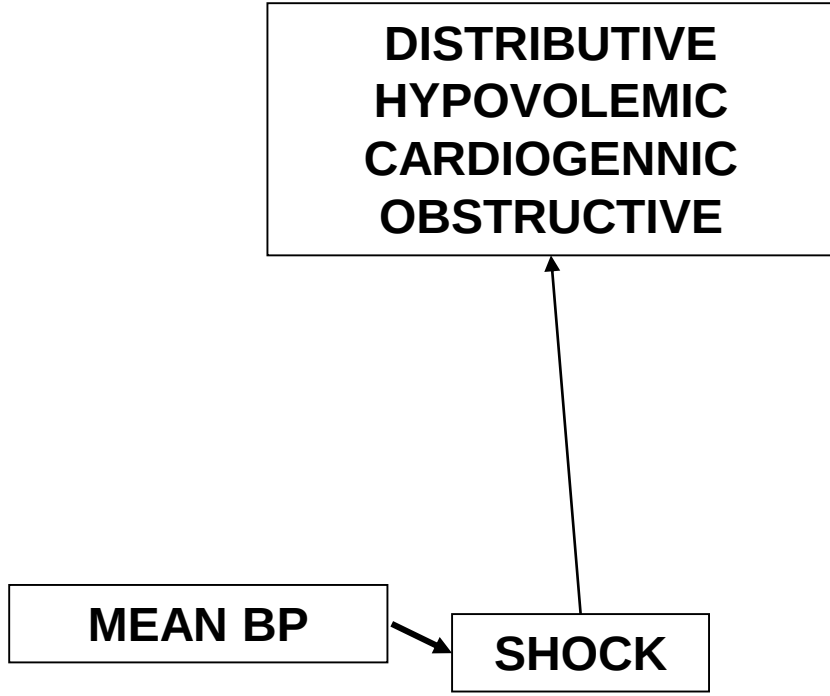
+ irreversible (refractory)

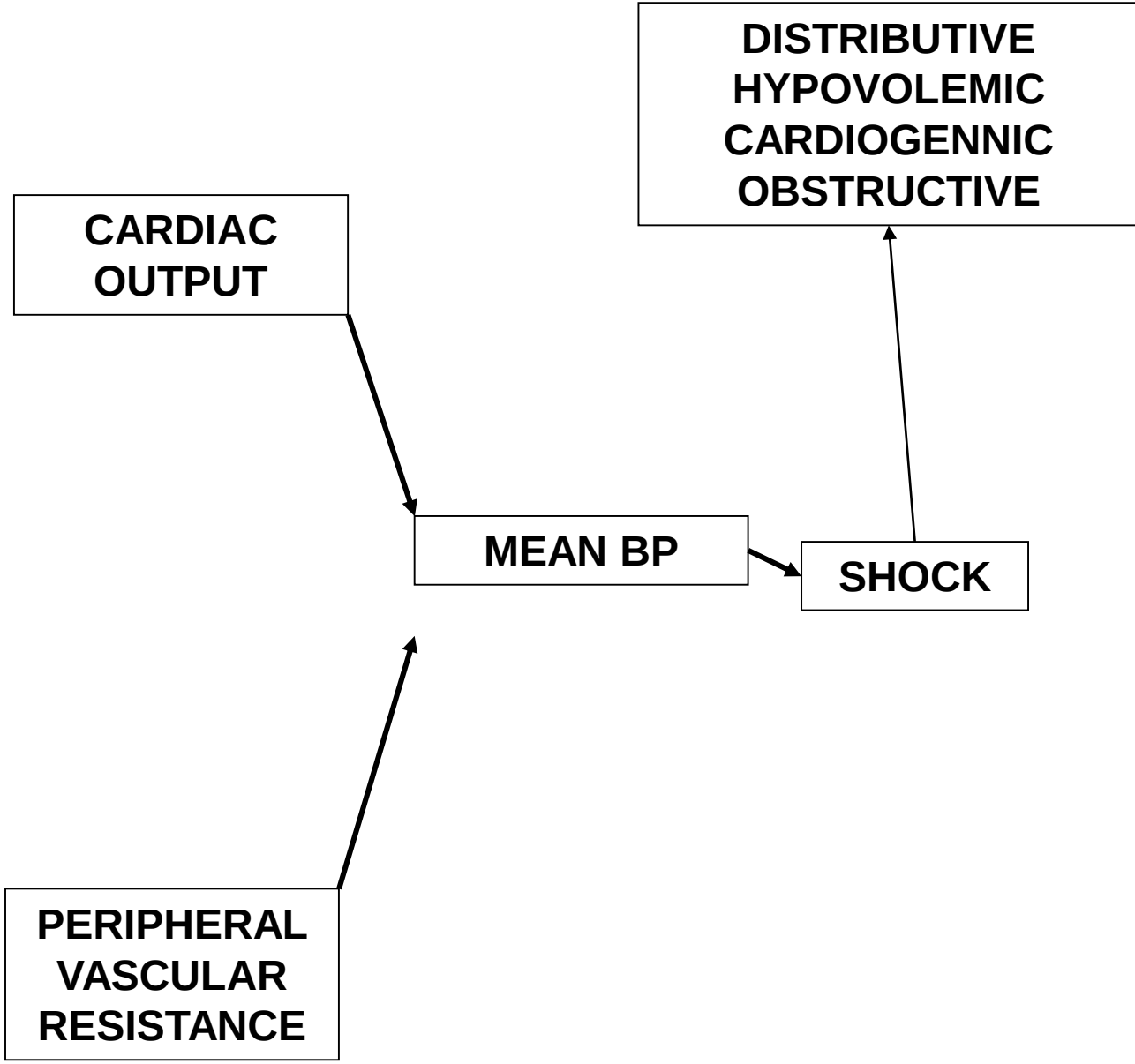
Representation of types of shock (depends on the department):

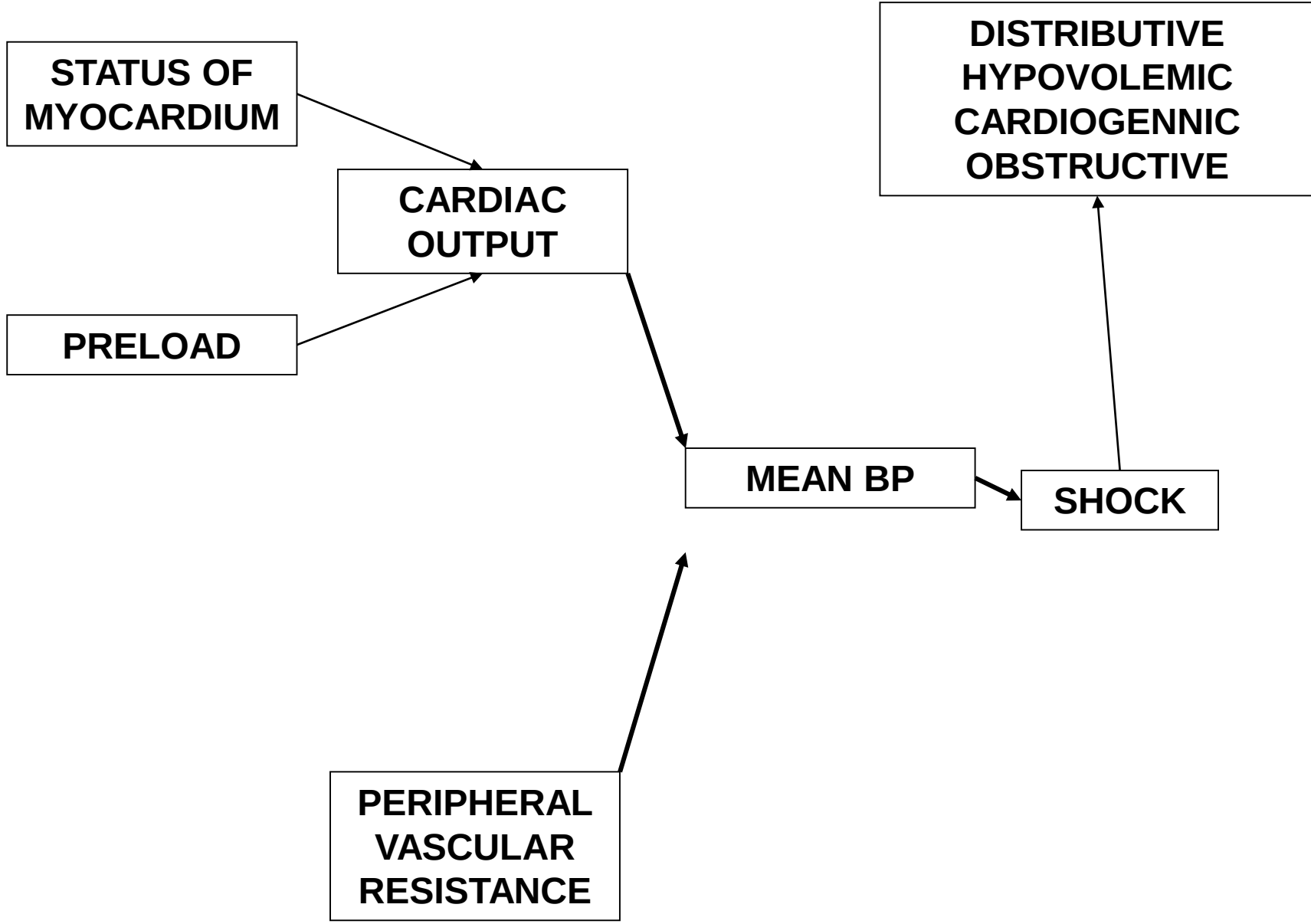
1. Distributive (66 %, from that 62 % septic/4 % anafylactic, neurogennic …)
2. Cardiogenic (16 %)
3. Hypovolemic (16 %)
4. Obstructive (2 %)
5. Mixed (1+2+3)

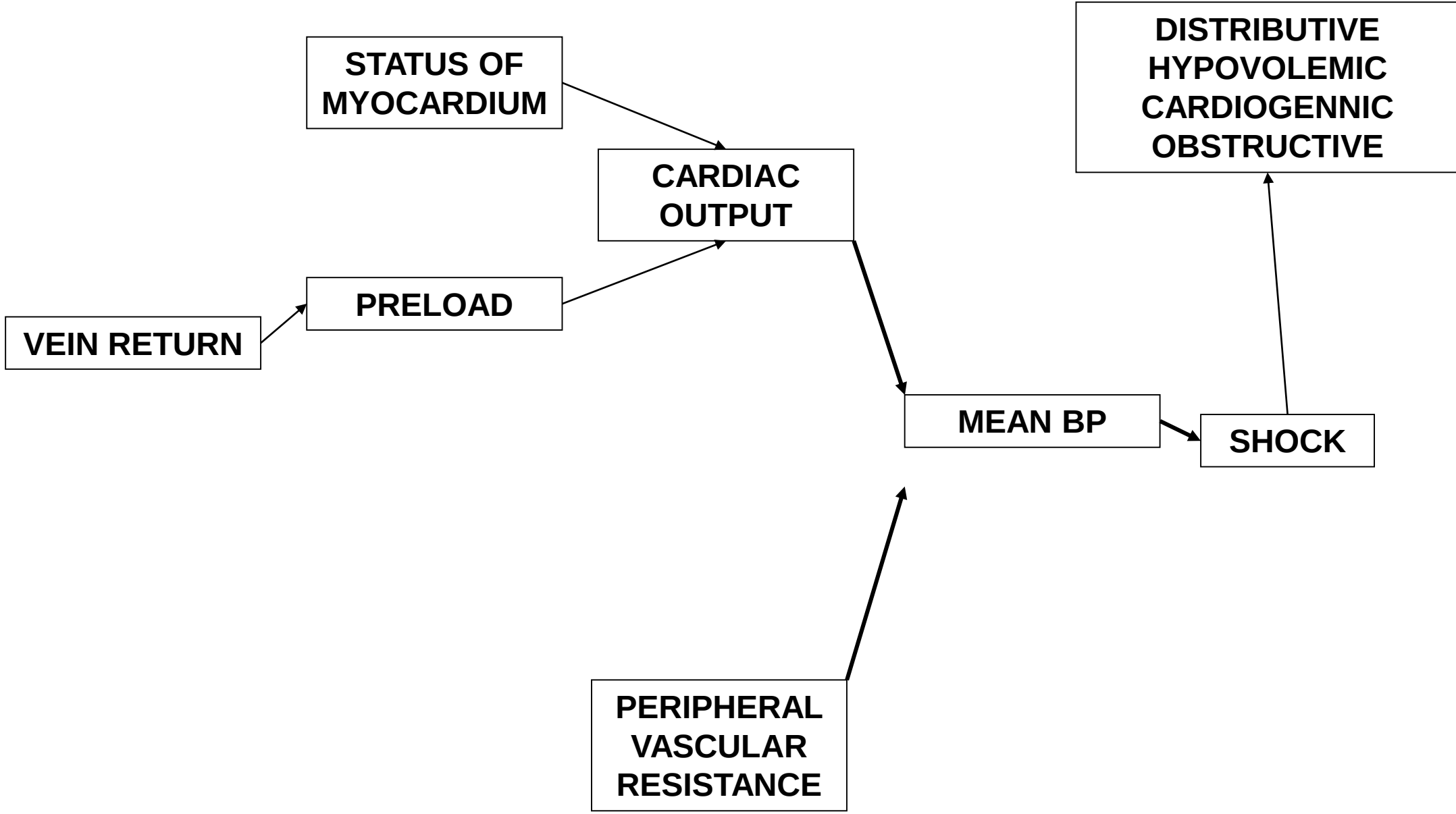
Classification - causes:

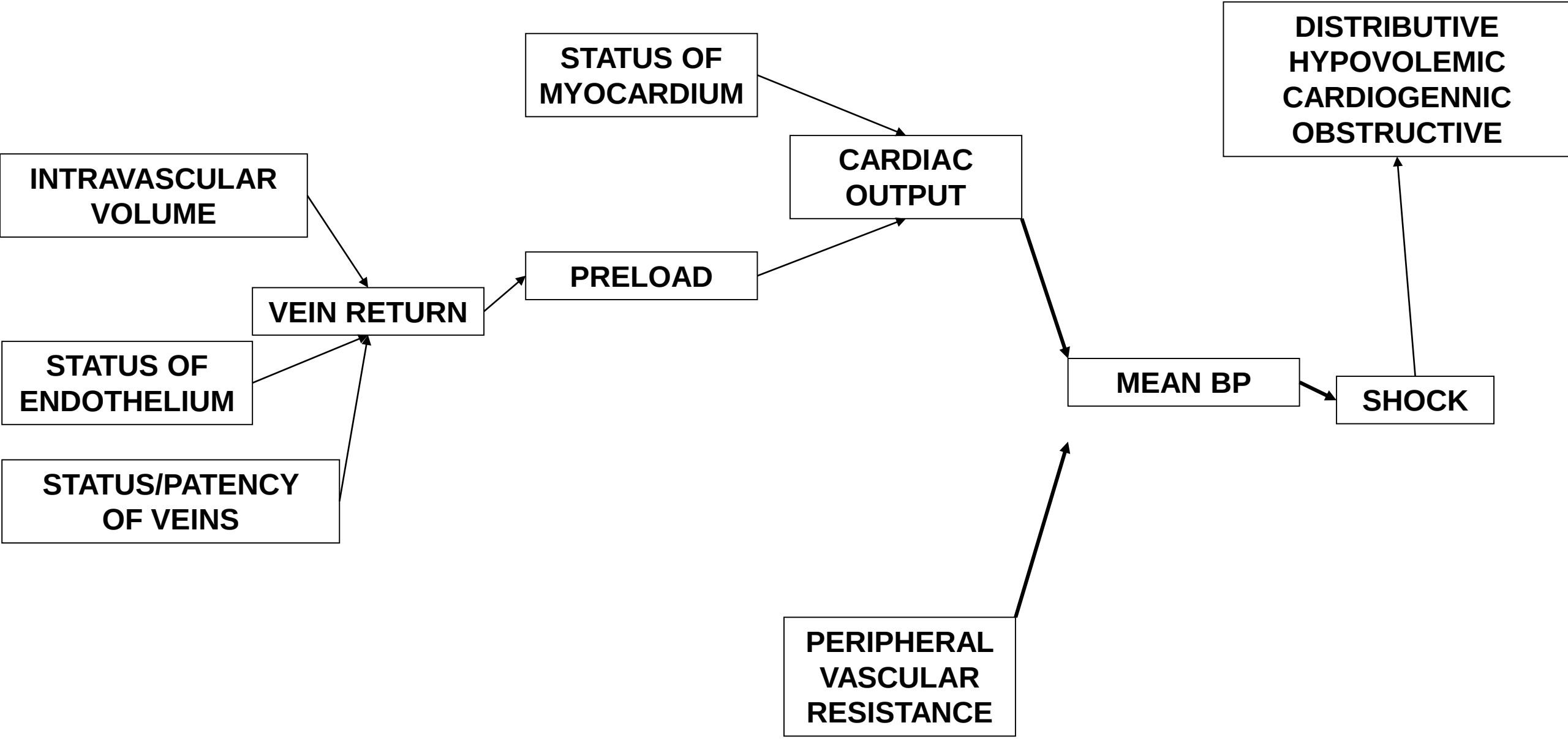
- **Distributive** (G- sepsis, alerg. anafylaxis, spine injury, drugs in anesteziology, …)
- **Cardiogenic** (MI, end stage heart failure …)
- **Hypovolemic** (GIT hemorrhage, trauma, pancreatitis, …)
- **Obstructive** (pulmonary embolism, cardiac tamponade, myxoma, dissection of asc. aorta, …)

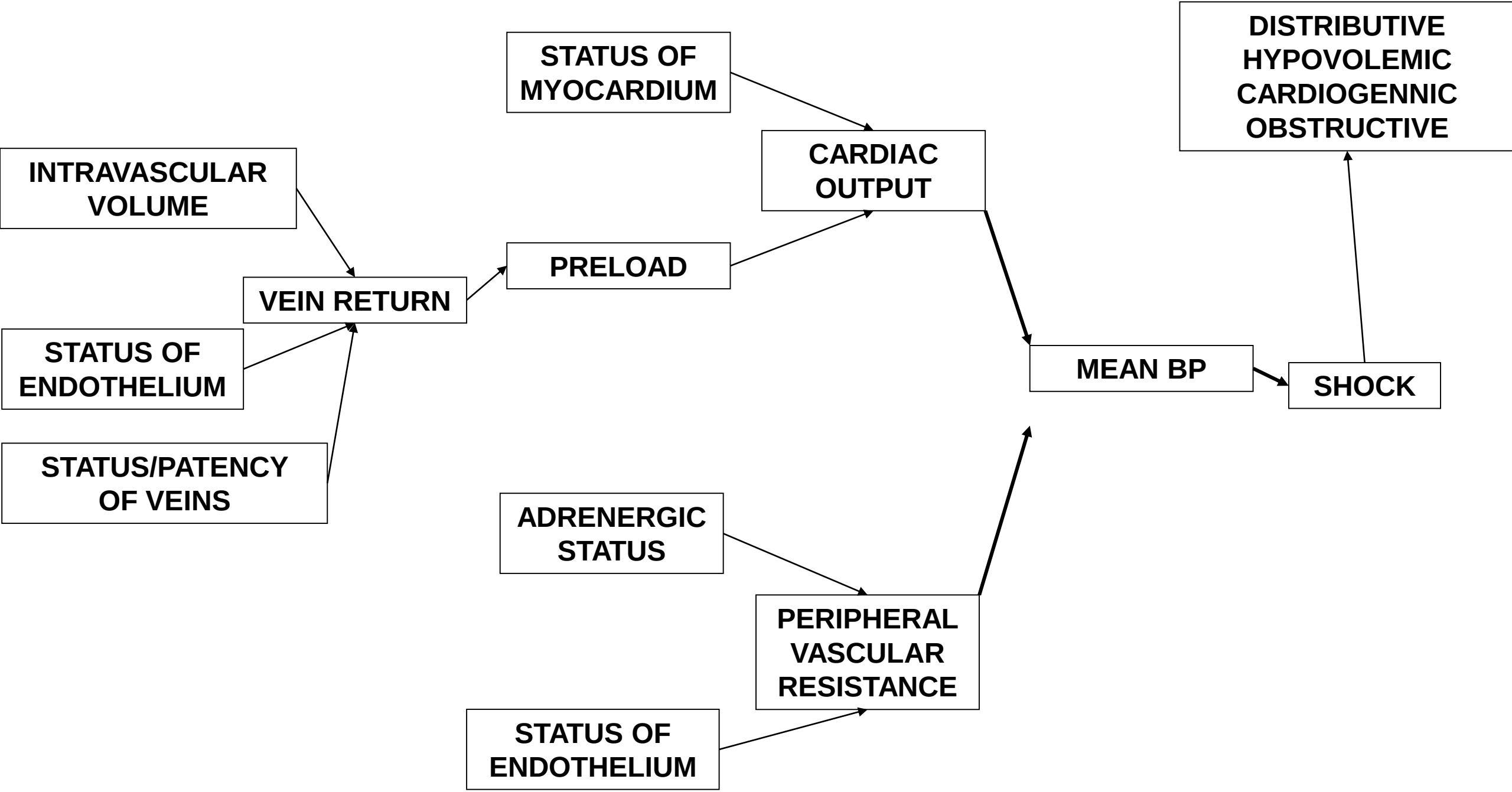

DISTRIBUTIVE
HYPOVOLEMIC
CARDIOGENNIC
OBSTRUCTIVE
MEAN BP
SHOCK

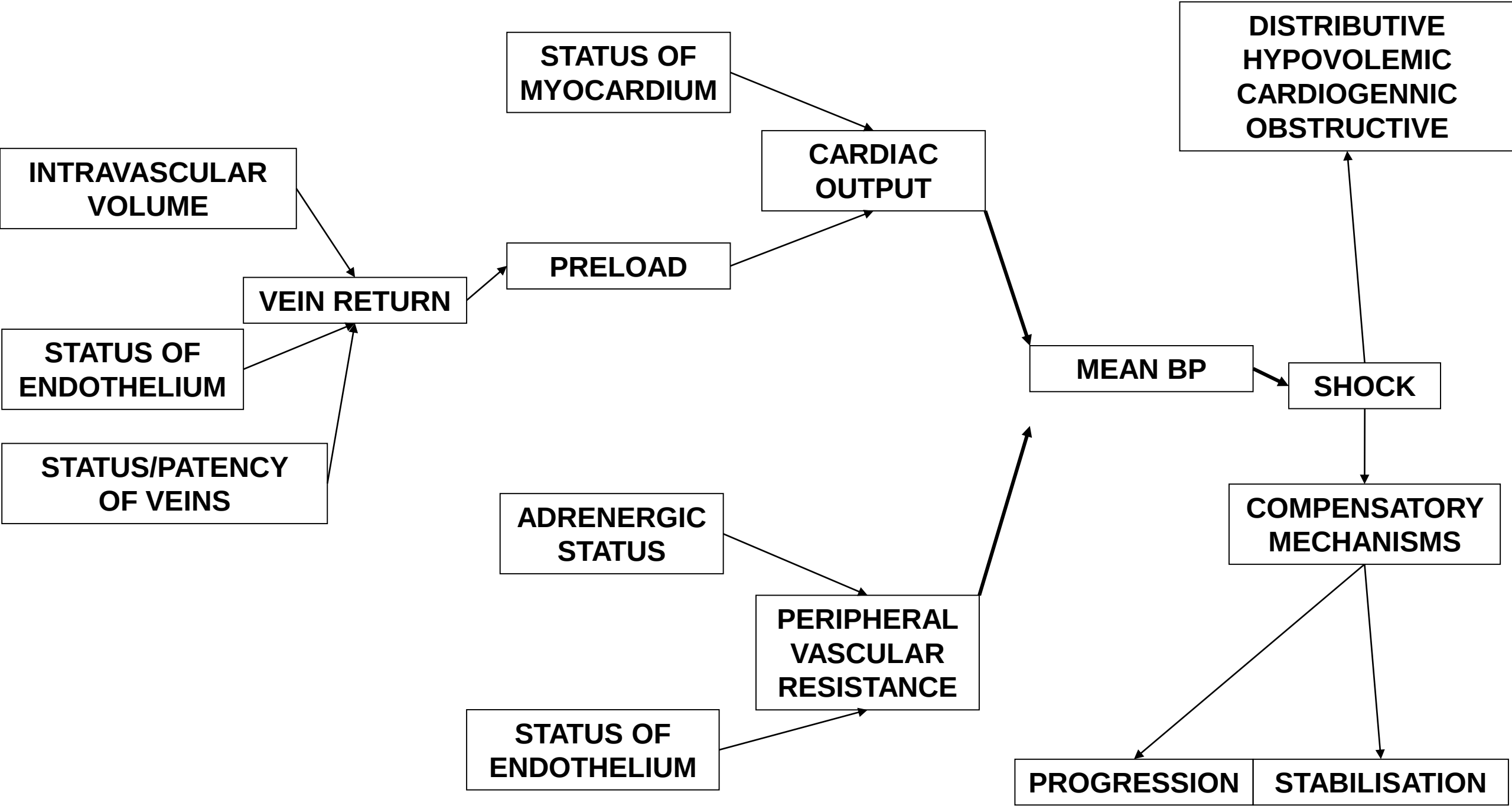

DISTRIBUTIVE
HYPOVOLEMIC
CARDIOGENNIC
OBSTRUCTIVE
CARDIAC OUTPUT
MEAN BP
SHOCK
PERIPHERAL VASCULAR RESISTANCE

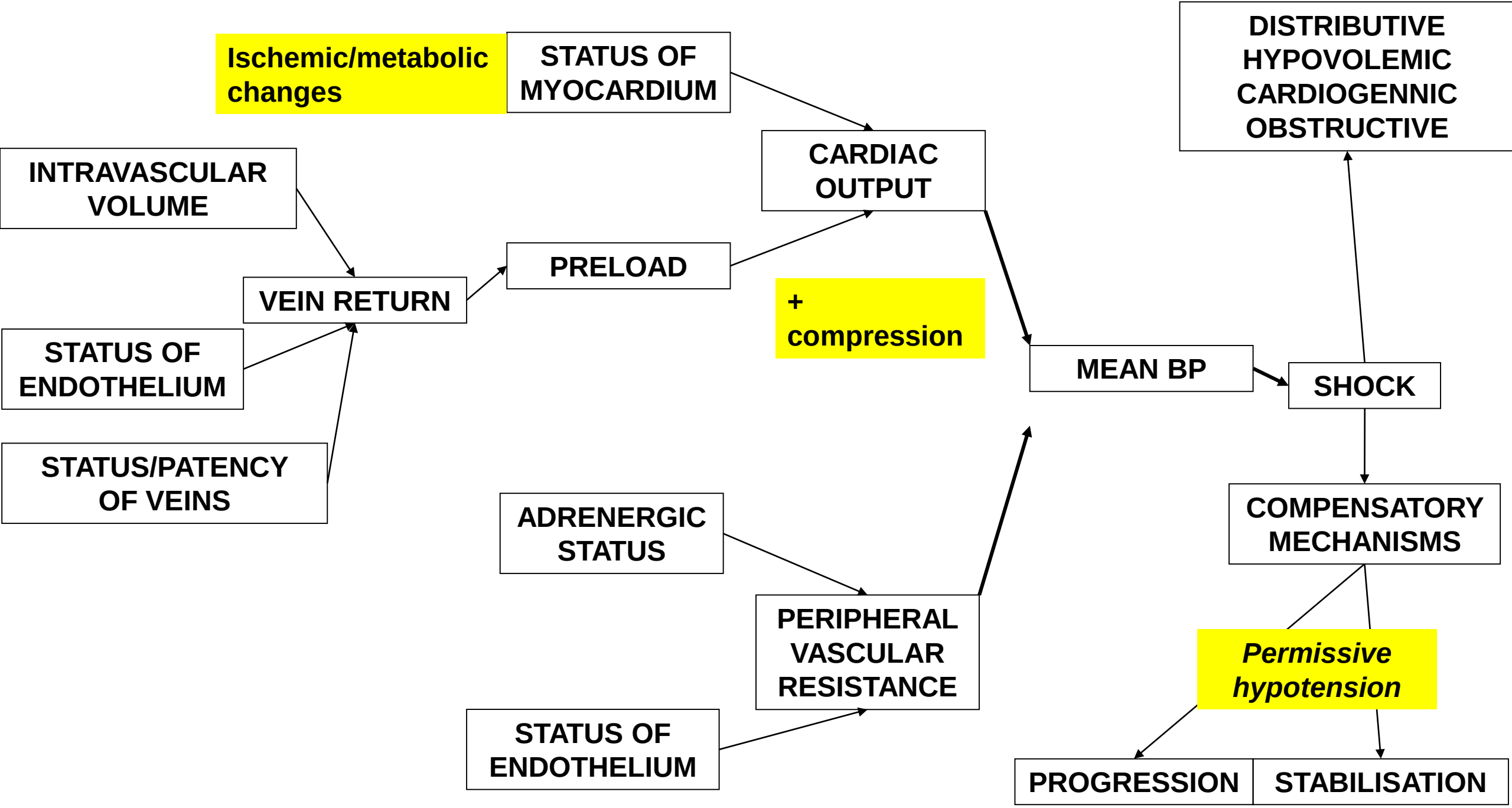

STATUS OF MYOCARDIUM
CARDIAC OUTPUT
PRELOAD
MEAN BP
SHOCK
DISTRIBUTIVE
HYPOVOLEMIC
CARDIOGENNIC
OBSTRUCTIVE
PERIPHERAL VASCULAR RESISTANCE


STATUS OF MYOCARDIUM
CARDIAC OUTPUT
DISTRIBUTIVE
HYPOVOLEMIC
CARDIOGENNIC
OBSTRUCTIVE
VEIN RETURN
PRELOAD
MEAN BP
SHOCK
PERIPHERAL VASCULAR RESISTANCE


DISTRIBUTIVE
HYPOVOLEMIC
CARDIOGENNIC
OBSTRUCTIVE
STATUS OF MYOCARDIUM
INTRAVASCULAR VOLUME
CARDIAC OUTPUT
PRELOAD
VEIN RETURN
STATUS OF ENDOTHELIUM
STATUS/PATENCY OF VEINS
MEAN BP
SHOCK
PERIPHERAL VASCULAR RESISTANCE


DISTRIBUTIVE
HYPOVOLEMIC
CARDIOGENNIC
OBSTRUCTIVE
STATUS OF MYOCARDIUM
INTRAVASCULAR VOLUME
CARDIAC OUTPUT
PRELOAD
VEIN RETURN
STATUS OF ENDOTHELIUM
STATUS/PATENCY OF VEINS
MEAN BP
SHOCK
ADRENERGIC STATUS
PERIPHERAL VASCULAR RESISTANCE
STATUS OF ENDOTHELIUM


DISTRIBUTIVE
HYPOVOLEMIC
CARDIOGENNIC
OBSTRUCTIVE
STATUS OF MYOCARDIUM
CARDIAC OUTPUT
INTRAVASCULAR VOLUME
PRELOAD
VEIN RETURN
STATUS OF ENDOTHELIUM
STATUS/PATENCY OF VEINS
MEAN BP
SHOCK
COMPENSATORY MECHANISMS
ADRENERGIC STATUS
PERIPHERAL VASCULAR RESISTANCE
STATUS OF ENDOTHELIUM
PROGRESSION
STABILISATION


Ischemic/metabolic changes
STATUS OF MYOCARDIUM
DISTRIBUTIVE
HYPOVOLEMIC
CARDIOGENNIC
OBSTRUCTIVE
INTRAVASCULAR VOLUME
CARDIAC OUTPUT
PRELOAD
VEIN RETURN
+ compression
STATUS OF ENDOTHELIUM
MEAN BP
SHOCK
STATUS/PATENCY OF VEINS
COMPENSATORY MECHANISMS
ADRENERGIC STATUS
PERIPHERAL VASCULAR RESISTANCE
Permissive hypotension
STATUS OF ENDOTHELIUM
PROGRESSION
STABILISATION

SHOCK – compensatory mechanisms

1. **Increased adrenergic activity/tonus – catecholamines (rcps. alfa, beta1, beta2)**
2. **RAAS – Na retention, increased intravascular volume**
3. **ADH/vasopresine – retention of H_2O, thirst, vasoconstriction in splanchnic aa.**
4. **Cytokines – increased activity of procoagulant/inflammatory markers**
5. ***endorfines, encefalines, …***

SHOCK – compensatory mechanisms

Alfa – vasoconstriction

Beta 1 – increased heart rate

Beta 2 – bronchodilation, vasodilation (striated muscles)

2. RAAS – …

3. ADH/vasopressin – …

4. *Endorfines, encefalines – pain relieve*

SHOCK – compensatory mechanisms

<u>Priorities: to manage blood flow to central nervous system and myocardium (+ *striated muscles*)</u>

x

Reduction of flow to kidneys, skin, GIT: clinical signs

(oliguria/anuria, cold sweated skin, hepatopathy - laboratory, …)

SHOCK – compensatory mechanisms

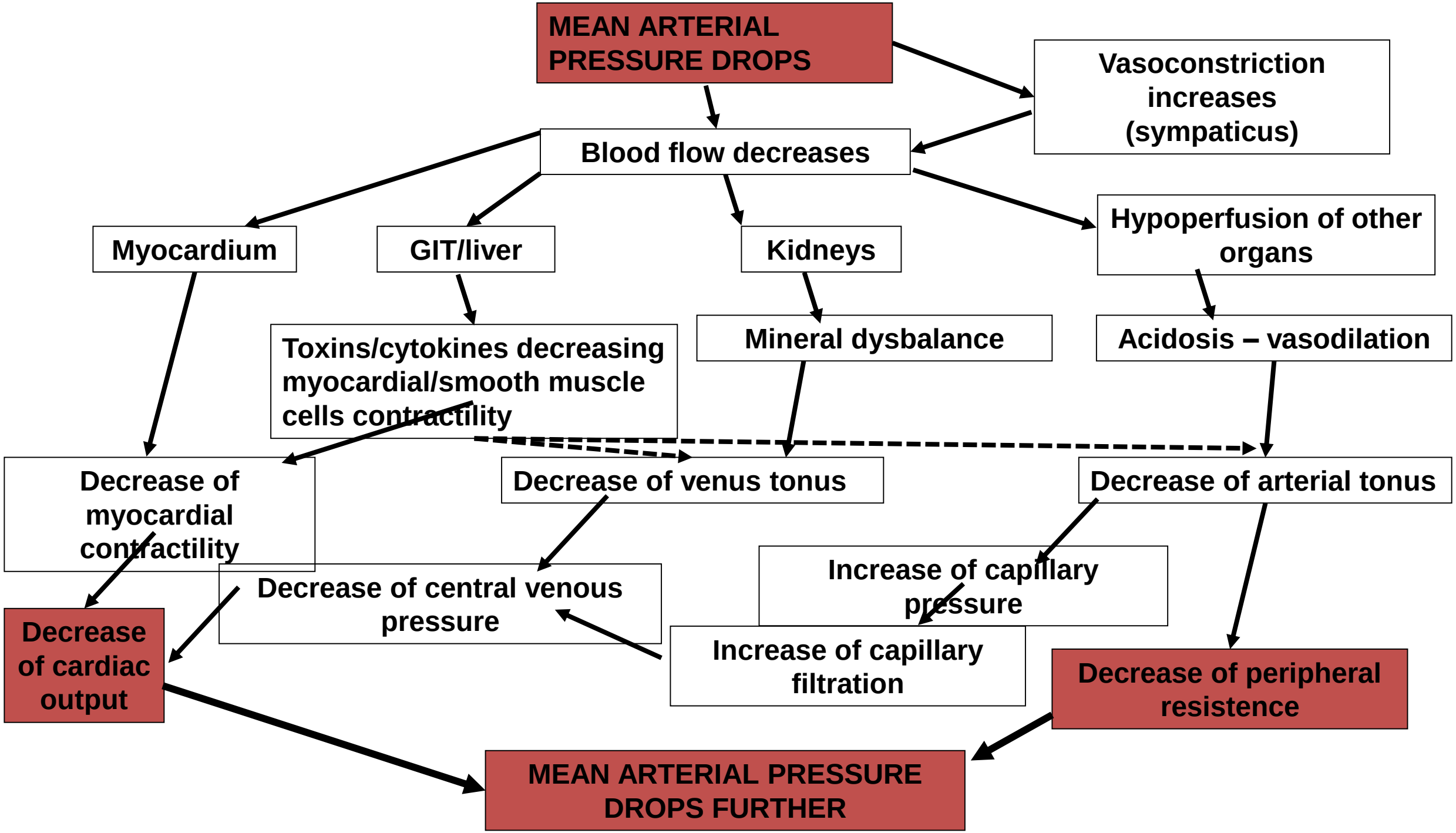
<u>Priorities: to manage blood flow to central nervous system and myocardium (+ *striated muscles*)</u>

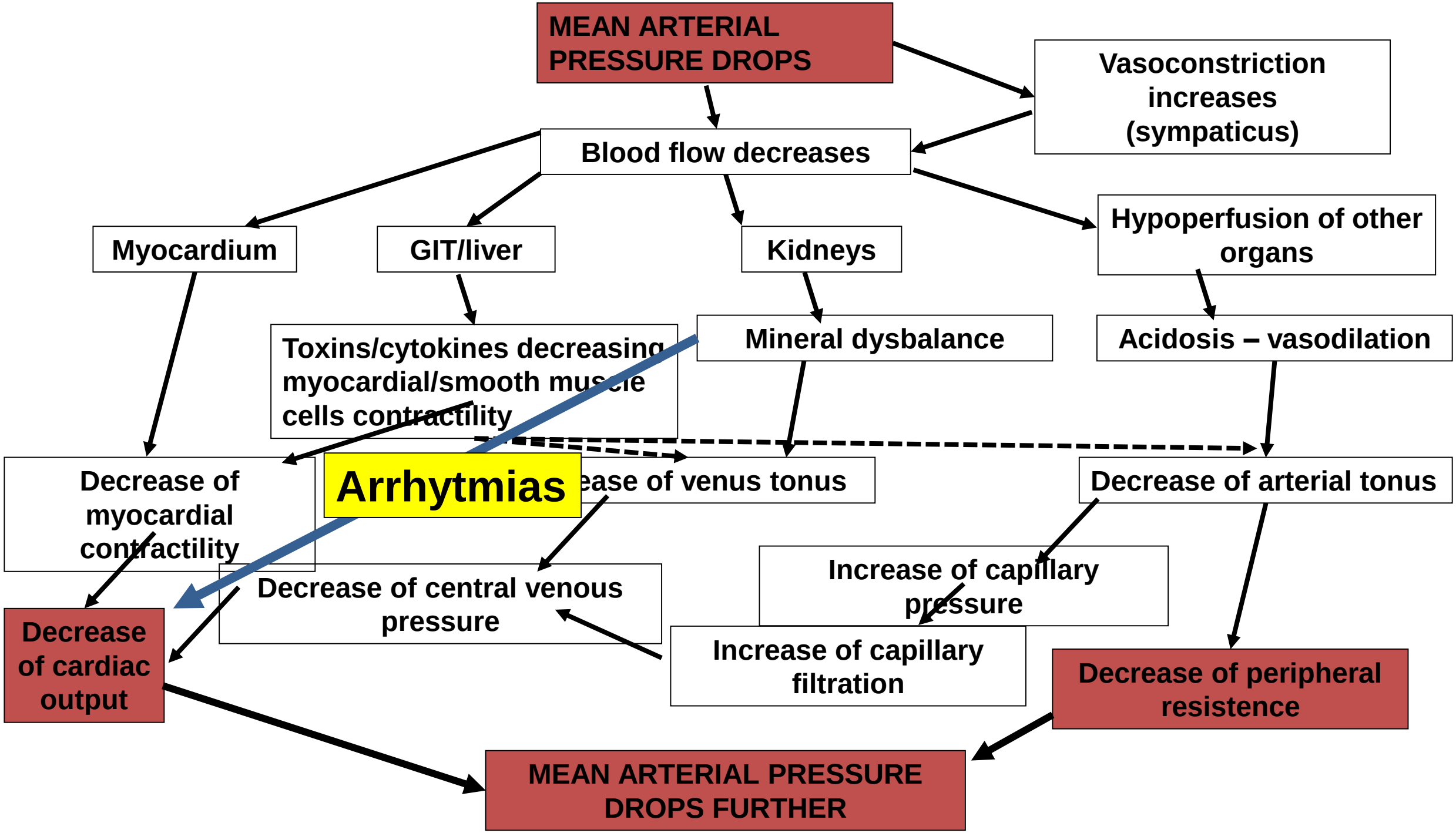
x

Reduction of flow to kidneys, skin, GIT: clinical signs

(oliguria/anuria, cold sweated skin, hepatopathy - laboratory, …)

Compensatory mechanisms lead to decompenstations (through cytokines, ..) – with ensuing cardiodepressive effects


MEAN ARTERIAL PRESSURE DROPS
Vasoconstriction increases (sympaticus)
Blood flow decreases
Myocardium
GIT/liver
Kidneys
Hypoperfusion of other organs
Toxins/cytokines decreasing myocardial/smooth muscle cells contractility
Mineral dysbalance
Acidosis – vasodilation
Decrease of myocardial contractility
Decrease of venus tonus
Decrease of arterial tonus
Decrease of central venous pressure
Increase of capillary pressure
Decrease of cardiac output
Increase of capillary filtration
Decrease of peripheral resistence
MEAN ARTERIAL PRESSURE DROPS FURTHER


MEAN ARTERIAL PRESSURE DROPS
Vasoconstriction increases (sympaticus)
Blood flow decreases
Myocardium
GIT/liver
Kidneys
Hypoperfusion of other organs
Toxins/cytokines decreasing myocardial/smooth muscle cells contractility
Mineral dysbalance
Acidosis – vasodilation
Arrhytmias
Decrease of myocardial contractility
ease of venus tonus
Decrease of arterial tonus
Decrease of central venous pressure
Increase of capillary pressure
Decrease of cardiac output
Increase of capillary filtration
Decrease of peripheral resistence
MEAN ARTERIAL PRESSURE DROPS FURTHER

SHOCK – irreversibility

Collaps of peripheral circulation, loss of function/increase of permeability of capillaries, dilation (resistence arterioles), dysfunction/afunction of life sustaining organs

<u>Clinical signs:</u> Unmeasurable blood pressure, cardiac arrest (electro-mechanical dissociation)

! Despite measurable mean blood pressure (60-70 mm Hg) during aggressive treatment (catecholamines): increase of lactate, progression of renal dysfunction, increasing acidosis,

Paradoxical improvement of metabolic parameters: increase of Sa02 , decrease of lactate - ?

SHOCK – DIAGNOSIS

History (chest pain, dyspnea, fever, ….)

Physical examination (vital signs – HR, BP, fever, RR, Sa02, diuresis…)

<u>Laboratory</u>

Other/noninvasive tests

<u>Invasive tests</u>

SHOCK – DIAGNOSIS

Underlying condition (ischemic heart disease, heart insufficiency, …)

Triggers (infection, hemorrhage, arhytmia, iatrogenic …)

First treatment of symptoms + looking for the cause/triggers

SHOCK – DIAGNOSIS

Kidneys !!!: diuresis lower than 30 ml/h

Decrease of BP (cave hypertonsive patient) – changes of pulse pressure (increased x decreased cardiac output)

Tachycardia (cave previous treatment betablockers)

Cold, sweaty skin (not in distributive shock)

Impaired consiousness

Laboratory: increased lactate, acidosis,

+ spec. tests – inflammatory markers, blood count …

SHOCK - DIAGNOSIS

Interventional :

Intraarterial measurement of blood pressure, pO2, pC02,

pH, HCO3. ..

Direct measurements of central venous pressure

+

Echocardiography

SHOCK - MANAGEMENT

1. **Prevention: ATB, revascularisation procedures, supplementation of fluids, ...**
2. **VIP (Ventilation, Infusions, Pump – heart**
3. **Manage the cause**

SHOCK - Ventilation

Intubate early

Oxygenate (40-50 % O_2),

= decreased work of respiratory muscles,

improved oxygenation

ARDS – smaller volumes, PEEP (?), …

SHOCK - Fluid replacement (critical)

1. **Appropriate fluid policy (crystaloids, colloids, …)**
2. **Rate of replacement - 300-500 ml/30 minutes, then according to patient status**
3. **Goal(s) – improvement of BP (70 MBP), CVP (10 cm H_2O)**
4. **Correction of overshooting (pulm. oedema, compartment sy)**

SHOCK - Vasoactive drugs

1. **Noradrenaline i.v. (0,1-2,0 ug/kg/min)**
2. ***Angiotensin II i.v. (cave thrombosis)***
3. **Isoproterenol – bradycardia**
4. **Dopamine – alfa x beta stimulation ?**
5. **Vasopressine – septic shock**
6. **Dobutamine – does not increase BP, but improves myocardial contractility**
7. **Inhibitors of PDE III** (amrinone, milrinone, …)

SHOCK – instruments – cardiogenic shock …

Mechanical/instrumental treatment (cardiogennic shock:

IABC (intraarterial baloon contrapulsation)

ECMO (extracorp.membrane oxygenator)

LVAD (HM II/III – left ventr. ass. device)

+ Experimental:

Inhibitors of inflammatory cytokines, …

CRITICAL CARE MEDICINE

Simon R. Finfer, M.D., and Jean-Louis Vincent, M.D., Ph.D., *Editors*

Circulatory Shock

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

N Engl J Med 2013;369:1726-34.
DOI: 10.1056/NEJMra1208943

SHOCK – factors modifying prognosis

Age

Underlying diseases – ischemic heart disease,

diabetes mellitus, …

Stage of shock (reversiblity) – timing of treatment

Critical status of microcirculation (DIC)

DIC
(disseminated intravascular coagulation)

- **Syndrom accompanying another serious disease**
- **Impairment of antithrombotic endothelial function, activation of monocytes/macrofages:**

Thrombi/fibrin deposists in microcirkulation

Mechanical damage of thrombocytes and erythrocytes

Activation and consumption of coagulation factors/out of control fibrinolysis (Tissue factors, f. XII – Hageman f. …)

DIC

Provoking factors:

Release of tissue factors (extrinsic pathway):

Abruption of placenta, embolization of amniotic fluid, retention of dead fetus, abortion in II. trimester

Trauma, burns

Malignancies

Direct damage of endothelium (intrinsic pathway):

Gram-negative sepsis, parasites, … HUS,

DIC

- **Clinical manifetation:**

1. **Chronic (laboratory signs)**

2. **Acute:**

Tissue ischemia (acrocyanosis, peripheral gangrenes, renal/liver insufficiency, …)

Serious bleeding after minimal injuries - i.v., …

DIC

- **Diagnosis:**

1. **Risk factors …**
2. **Clinical manifestation – ischemia x hemorrhage**

3. Laboratory findings: anemia (schistocytes), thrombocytopenia, prolonged PT, aPTT, decrease of fibrinogen, increase of FDP (fibrin degradation products, D-dimers, lactate (?).

DIC

- **Management:**

1. **Find and remove the cause**

2. **Manage bleeding/ischemic complications:**

Replacement of coagulation ff. (fresh frozen plasma, thombocytes, coagulation ff.)

Heparin – chronic DIC (malignancies), combination with replacement of coagulation ff. – monitor FDP, lactate, ..

CONCLUSIONS:

- **Reveal and manage risk of shock**
- **Reveal evolving shock (diuresis!)**
- **Monitor/correct vital functions look for the cause**
- **In evolving shock apply more aggressive approach (intubation, ventilation, hydration, catecholamines).**