Shock
Review of Types , Stages, and Treatment Principles

B. pp 303-311; 312-319
Paramedic Care I pp. 203 - 218
K. p. 213 - 223 (skim)
C. pp 53 -69 (drug doses)

Objectives

Upon completion of this lecture, the learner should be able to:

- describe the basic pathophysiology of shock
- review the determinants of cardiac output and blood pressure
- review how preload, afterload, and contractility affect stroke volume
- review the stages of shock and some of the types of shock

Objectives, continued

- review the pathophysiology of respiratory failure, hypovolemia, vascular failure, and cardiac failure.
- describe the compensatory mechanisms triggered by shock
- describe interventions that improve circulation and O_2 delivery.

Priority of Fluid and Electrolyte Derangement in Disease

Stratification

- A / B: oxygenation and ventilation
- C: circulation
- then equilibrium of acid-base status

Rate of Correction and Priorities

Rate

- rate of correction usually should mirror the rate of derangement

Priorities

1. volume - adequate tissue perfusion [C]
   (C depends on A + B)
2. pH
3. potassium, calcium, and magnesium
4. sodium and chloride

Initial Treatment of Shock Presumed to be due to Fluid Loss

COMEBIG

– 2 LB IVs NS WO

Shock

- Shock can be defined as :
  circulatory failure to adequately perfuse and oxygenate the tissues.
- Most signs and symptoms of shock represent compensatory measures used to maintain oxygen delivery to the vital organs : heart, lungs and brain.

Shock - Introduction , cont.

Prehospital management of shock :

- Importance of the recognition of the early stages of shock
- Importance of airway management and high flow oxygen administration
- Importance of definitive care of underlying cause

Cardiovascular Physiology

- **Cardiac Output**
  - CO = HR x SV
- **Blood Pressure**
  - BP ≈ CO x SVR
  - BP ≈ HR x SV x SVR

Cardiovascular Physiology

Factors affecting cardiac output and blood pressure

- HR
- PVR
- SV

Factors affecting stroke volume

- preload
- afterload
- contractility

CV Physiology , cont.

- PVR - blood vessels
  - Vascular muscles under control of the sympathetic nervous system
  - Most of the blood volume (2/3) is in the veins and venules (during normal circulation)
    - venoconstriction will increase venous return (preload) to the heart

CV Physiology , cont.

- Oxygen transport
  - hemoglobin : necessary for O_2 transport
  - not just volume (such as NS)
- Blood
  - hemoglobin
  - delivers O_2 and nutrients to the tissues and transports CO_2 + wastes away
  - average blood volume ~ 70mL/kg or 5 L

CV Physiology , cont.

Acid - Base (oxyhemoglobin dissociation curve)

- Uptake and release of oxygen:
  - Acidosis shifts the curve to the right: O_2 more easily released into tissues
  - Alkalosis shifts the curve to the left: less O_2 is released into tissues

Factors Affecting Perfusion and O_2 Delivery

- heart rate
- stroke volume
  - preload
  - afterload
  - contractility
- hemoglobin concentration
- acid-base status
- oxygen concentration

CV Physiology , cont.

Oxygen Delivery

- in normal circulation
  - 20% of delivered O_2 is extracted by tissues
- in shock
  - tissue O_2 extraction may increase to 50%

Oxygen Delivery , cont.

Oxygen Delivery
- Requires normal A + B
- In inadequate A + B
  - reduced amount of hemoglobin -O_2 delivered to tissues

Types of Shock

- Pump (cardiogenic - MI, TCA, tension pneumo.)
- Pipes (septic, spinal, metabolic acidosis,…)
- Water (hypovolemic - hemorrhagic, dehydration)

Types of Shock

- Hypovolemic (Perfusate Failure)
  - Loss of blood (and Hb) to transport O_2
- Vessel Failure
  - Loss of sympathetic muscle tone
- Cardiac Failure
  - Loss of rate
  - Loss of muscle
  - Loss of CO (valvular injury)
  - Loss of venous return (obstruction)

Compensatory Mechanisms

Sympathetic Nervous System Activation

- Epinenephrine
  - restlessness
  - increased HR
  - increased RR
  - increased PVR
    - skin changes

Compensatory Mechanisms , cont.

Neuroendocrine Response

- Vasopressin (ADH) is released by pituitary
  - increased PVR
- Thirst is stimulated

Decompensation

Compensatory mechanisms overwhelmed

- O_2 delivery begins to fall precipitously
- Staged dysfunction, then death
  - cells
  - tissues
    - brain [confusion]
    - skin [cyanosis]
    - muscle [poor tone]
  - organs
  - organ systems
  - organism

Stages of Shock

STAGES : I – IV		collated with *sx. + sn.s*	collated with blood loss
Mild (early)	I	"compensated" restlessness increased heart rate increased respiratory rate skin changes: pale,…	~ 10 % (500* mL)
Moderate	II	still "compensating" thirst decreasing respiratory effort [narrowed pulse pressure]	~ 20 % (1000 mL)

* assuming a blood vol. of 5 L

Stages of Shock

STAGES : I – IV		collated with *sx. + sn.s*	collated with blood loss
Severe (late)	III	"decompensating" confusion drop in BP upon upright oliguria	~ 30 % (1500 mL)
"**Irreversible**"	IV	"decompensated" decreased respiratory effort sustained drop in SBP cardiac and respiratory arrest	~ 40 % (2000 mL)

Improving Circulation and O_2 Delivery

$CO_2M_3E\ BI_{NS}G$

- ABCs
  - Improve oxyhemoglobin saturation
  - Ensure adequate HR
  - Increase hemoglobin level ASAP
    - Increasing hemoglobin from 8 to 12 gram/dl will increase O_2 delivery 50%

Improving Circulation and O_2 Delivery , cont.

- Manipulating Preload (one Stroke Volume determinant)
  - 20-30 ml/kg crystalloid
    - If VS + O_2 sat. improve, bleeding has probably ceased.
    - If VS improve transiently, or not at all, blood loss is probably continuing and the patient needs rapid surgical intervention.
  - Fluid Choice
    - in shock, colloids leak from vascular space
    - crystalloids (NS; RL) : preferable
    - hypertonic saline (3%; 7%);
      blood substitute: experimental

Improving Circulation and O_2 Delivery , cont.

Afterload treatment

- preload is maximized first, afterload is undertaken next
- low SVR (low afterload) shock
  - spinal
  - anaphylactic
  - septic
  - metabolic / toxic

Improving Circulation and O_2 Delivery , cont.

- Afterload increasers
  - Agents that increase SVR
    - Vasopressors
      - Dopamine (alpha; high dose)
      - Norepinephrine (levophed)
      - Vasopressin (ADH; pitressin)
      - Epinephrine
    - Contraindicated in hypovolemic shock

Pressor Agents

- Dopamine
  - *1600* mg/ 500ml @ 30ml/hr for 100 kg:
    - *16* mic./ kg / min
- Epinephrine
  - *2* mg in 500ml @ 30ml/hr
    - *2* mic. / min
      (2

Pressor Agents , cont.

Norepinephrine (Levophed)

- range of drip dose : 0.5 - 30 mic./ min
- *8* mg placed in 500ml (4mg/vial x 2 vials) @ 30ml/hr
  - *8* mic./ min

Pressor Agents , cont.

Vasopressin

- *250* Units in 500ml (10 U. / 0.5ml vial x 25 vials)
  (or equiv amount ; say, 5 vials in 100 cc)
- @ 30ml/hr
- Starting dose * : 0.*250* U / min
  * ***investigational*** (Range of drip dose : 0.01 - 0.5 U / min)
- For persistent VF / VT : IV bolus [40 U IV]

Improving Circulation and O_2 Delivery , cont.

- Increasing Stroke Volume , cont.
  - Contractility
    - Inotropic agents
      - Dobutamine
      - Dopamine

Summary

We have discussed :

- the stages of shock and some of the types of shock
- the basic physiology of circulation: cardiac output blood pressure, and the affect of preload, afterload, and contractility on stroke volume
- the compensatory mechanisms triggered by shock and the importance of recognizing the early stages of shock