SHOCK

May 12, 2011

Body and Disease

Shock

- Definition of shock
- Pathophysiology
- Types of shock
- Management of shock

Shock

- Definition?

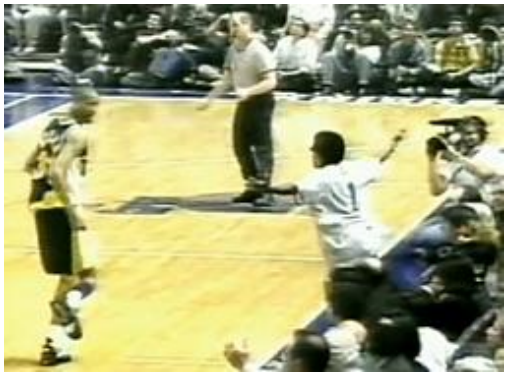
Shock

What common people think shock is....

- What the Duke Community would have experienced if Gordon Hayward of Butler had hit the last second three point shot in the 2010 NCAA Final

Shock

- What Spike Lee said after Reggie Miller dropped 25 points in the fourth quarter on the Knicks in the 1994 NBA Eastern Conference Finals



Shock

- What you get when you walk across the carpet and pet the cats for a long time and touch the doorknob

Shock

- How Pikachu and other electric Pokemon attack other Pokemon characters

Shock

A more scientific definition

- “The rude unhinging of the machinery of life” – Samuel Gross 19th Century surgeon

Shock

- Final common pathway of all life whether it be from myocardial infarction, microbial sepsis, pulmonary embolism, trauma, anaphylaxis

Eventually the body will wind up in shock.

Shock – Pure definition

- Inadequate perfusion at the cellular level

Remember this slide and the Krebs cycle, you make a total of 38 ATP

Shock

- No substrate for aerobic cellular metabolism
- Mitochondria do not have adequate supplies of glucose and oxygen to create ATP

In essence, your cells die

Shock

- Diminished oxygen delivery puts limits on oxygen consumption
- Energy output becomes dependent on anaerobic metabolism

Fick Principle

Normally you make 38 ATPs, if you loose oxidative phosphorylation and drop down to glycolysis, you only get 2 ATPs. You're down to like 5% efficiency.

Shock

- Decreased ATP
- Increased lactate production

Adding more poisons to the system

Shock

On the cellular level:

- Loss of cellular integrity
- Normal ion gradients damaged
- Intracellular fluid increases

Shock

- Loss of Na-K-ATPase pump
- Acidosis
- Calcium influx

Shock

- End result – cellular edema and energy deficit lead to cell death and organ dysfunction

Organs collapse

Shock

Circulatory level

- Vascular endothelial cell damage leads to cytokine and immunomodulator release
- Microcirculation damage from continued substrate interruption
- Multiple organ system failure

Shock

Later on you get this:

- Mechanical obstruction of the microcirculation with a loss of lumen diameter and increased osmotic concentration of intravascular material

Shock

Now your organs can't function

- Organs are not able to autoregulate flow
- Liver failure – deficiency of clotting factors
- Decreased renal perfusion – increase in intra and extravascular volume, electrolyte abnormalities
- Decrease in lung compliance

Factors made by liver: II, VII, IX, and X

Shock

- Cytokine activation
- Complement activation

Spiral downhill...

Shock – Treatment Focus

Maximize these three things for treatment:

Remember:
CO = SV x HR
You need to augment either one or both of these.

- Oxygen delivery
- Cardiac output
- Oxygen carrying capacity of the blood

Give O2, IV fluids

Treatment

- ABCs
- Vascular access
- Fluid resuscitation
- Vasopressors
- Consider antimicrobials

Make sure they have an airway, can breath, and can circulate something

Recognition of Shock

- High index of suspicion
- Constant reevaluation

BIG CONCEPT:
This is how shock presents: There's something out of place, something that doesn't fit in.

You always need to be thinking about it.
"There's something weird happening that maybe I need to act on now"

Recognition of Shock

Poor man's oximeter reading: mental status.

If you can stay alert and functioning, your brain is probably getting enough glucose and oxygen.

- Level of consciousness
- Vitals – respiratory rate and effort, heart rate, pulses
- Skin perfusion

As you walk into the patient's room, you can tell if they're "shockish" or not.

Recognition

- Level of consciousness
- Early – normal or anxious
- Late – agitated, confused, lethargic

Patients can act a little antsy about things, because they're not perfusing. Don't dismiss behavior changes too readily because it could be shock.

Recognition

- Respiratory rate and effort
- Effortless tachypnea

Effortless tachypnea = rapid breathing without using accessory muscles, without exertion

Why?
When you breathe fast, you're trying to get rid of CO_2 (by product of being acidotic)

If you see a patient breathing 30 w/o batting an eyelash, THINK that something's out of place...

Recognition

- Heart rate

- Increased
- Decreased

Usually increased

If you cant increase SV, you have to increase HR

Some instances it can be decreased

Recognition

- Pulses

- Weak and thready Not a good thing.
- Early septic shock - bounding

Recognition

- Skin perfusion

- Cool, delayed capillary refill time
- Early septic shock – warm, brisk capillary refill time

Hypovolemic Shock

Can be caused by a number of things...

- Hemorrhage

i.e. bleeding out because of trauma, GI bleed, etc.

- Dehydration

Vomiting, not tolerating POs

- Plasma losses from burns
- Diabetic ketoacidosis

You lose fluids because glucose makes blood osmotic and brings stuff out, you don't tolerate PO because you're throwing up, and you're tachypnic

Sensible fluid losses: you're aware of it (GI tract losses, urine)

Insensible fluid losses: you're not aware of it (via skin and lungs)

Hypovolemic Shock

- IV Fluids
- Blood – type specific cross match preferred
- Hypoglycemia

Watch out for this if their calorie count is down or negative.

Whenever you put in an IV, check bedside glucose. Its better to find out early before they go into late stage shock

Distributive Shock

- Decreased vascular tone leads to relative vascular volume depletion
- Intravascular volume is not distributed properly

Distributive Shock

- Decreased vascular tone results in pooling of blood in the large veins
- Decreased venous return results in decreased preload
- Cardiac output falls

Distributive Shock

Usually caused by:

- Anaphylaxis
- Spinal cord injury

Loss of sympathetic tone going to heart, lose vascular tone.

Distributive Shock

- Initial treatment with IV fluids
- After two to three boluses, consider vasoactive medications

Epinephrine
norepinephrine
dopamine

Vasoactive meds don't respond to medications.

Distributive Shock

- Dopamine or norepinephrine
- Alpha adrenergic properties cause systemic vasoconstriction

How to chose vasoactive meds: start with a low dose and go higher, unless shock is really bad.

Distributive Shock

- Anaphylaxis
- Epinephrine IM faster and more consistent uptake
- Antihistamines
- Steroids

Better than IV

If you give via IV, you get problems tachycardia and dysrhythmia

H1/H2 Block

Obstructive Shock

- Blood is unable to enter or leave the heart even with normal intravascular volume and cardiac function

Obstructive Shock

- Cardiac tamponade
- Tension pneumothorax
- Pulmonary hypertension
- Coarctation of the aorta

One of your big players responsible

Air in spaces where it should be. Compresses structures and doesn't let them function properly

Gives an obstructive pattern for blood flow

Obstructive Shock

If you remove pericardial fluid in someone with chronic issue (like SLE), the heart might not be able to adjust

- Cardiac tamponade
- Fluid in the pericardial sac causes increasing pressure around the heart
- Increased right atrial pressure causes a decrease in blood return to the heart
- Decreased ventricular filling results in decreased stroke volume

Obstructive Shock

- Most causes of obstructive shock cannot be treated pharmacologically
- Look for emergent interventions

"Stick a needle into something"

Cardiogenic Shock

Probably will see this kind of frequently

- Intrinsic dysfunction of the heart and decreased myocardial contractility causes decreased cardiac output

Cardiogenic Shock

- Myocarditis
- Cardiomyopathies
- Trauma
- Dysrhythmias

Cardiogenic Shock

- Treatment is different than other forms of shock
- Increased intravascular volume and an increase in systemic vascular resistance increase afterload, increasing the work of the heart

For treatment, you want to reverse these things

Cardiogenic Shock

- Milrinone
- Increases inotropy, diastolic relaxation, and peripheral vasodilation
- Dobutamine
- Beta adrenergic with no alpha effects
- Increases inotropy
- Decrease in afterload and blood pressure

Why wouldn't you want vasoconstrictors here?
Because it would increase afterload/BP.

Septic Shock

- Most controversial
- Most studied
- Least understood

No one really knows what to do.

Septic Shock

- Multiple organ system derangement
- Can be considered a form of distributive shock
- Also a combination of distributive, hypovolemic, and cardiogenic shock
- Host response to the insult is the key factor

Septic Shock

- Infectious agent Bacterial, viral, parasitic or fungal
- Mediators produced by the infectious agent
- Response of the immune system to the infectious agent

Septic Shock

- Responses are usually the result of mediator release
- Interleukin-1, tumor necrosis factor-alpha, cytokines, platelet activating factor

Immunology's coming back to haunt us!

Septic Shock

Two classifications of septic shock:
Warm and Cold

- Warm shock
- Early septic shock - a hyperdynamic cardiovascular state with bounding pulses and warm extremities
- Despite increased cardiac output, they may be profoundly hypotensive because of decreased systemic vascular resistance
- Wide pulse pressure

BP = 110/40?
Warning sign!

Vital signs are important: if you get a weird vital, you have to address it and figure out what's going on.

Hopefully you stop the shock at the warm stage, and don't get to cold...

Septic Shock

- Cold shock
- Cool, clammy or mottled skin with diminished or absent pulses and delayed capillary refill time
- Cardiac output falls as a result of increased systemic vascular resistance, decreased preload, and myocardial depression

At this point, its almost irreversible.

Septic Shock

- ABCs
- Vascular access
- Fluid resuscitation
- Packed red cells
- Fresh frozen plasma

Kitchen sink: throw everything you can think of at them and hope something works.

Septic Shock

- Vasopressor therapy
- Dopamine
- Norepinephrine
- Epinephrine

Triple pressors = using all three

Once you're using triple pressors = bad prognosis

Is there any immune therapy to treat this? There's a lot of work done, but no consensus. Maybe steroids, but maybe not.

Ripe area of research for critical care/ emergency medicine junkies.

Septic Shock

Review...

- Alpha –smooth muscle contraction in arterioles and bronchiole muscles
- Leads to vasoconstriction, increasing blood pressure and afterload

Septic Shock

You'll have to know this forever...

- Beta -1 receptors mediate contractility (inotropy) and heart rate (chronotropy)

- Beta -2 receptors cause smooth muscle relaxation with arteriole vasodilation and bronchiole relaxation

Septic Shock

- Dopamine
- Combined alpha and beta adrenergic effects
- Increase systemic vascular resistance with vasoconstriction
- Increase cardiac output with increased contractility and heart rate

What amounts of DA do you give?
Start low, and work your way up. Start at 5 and work up to 10. Then add epi if it doesn't work, then add NE

Septic Shock

- Norepinephrine
- Primarily an alpha agonist
- Systemic vasoconstriction
- Anaphylaxis, spinal shock, early septic shock

Septic Shock

- Epinephrine If you're using this, it's pretty bad.
- Potent alpha and beta effects
- Severe septic shock, post-cardiac arrest

Septic Shock

- Antibiotics
- Steroids

Some say yes for steroids, some say no

Diagnostic Studies

- Orders?

Diagnostic Studies

- Blood gas/Shock Panel
- Bedside glucose
- Lactate
- Cultures
- CBC, chemistries, coags, type and cross
- CXR, ECG, Echo

You can get this really quickly. VERY IMPORTANT. You always want to order one in a shock patient

Gives you ions, pH, glucose, Ca++, lactate

Also quick and an easy test.

Part of shock panel

Remember to never delay ABX to wait for a culture.

"Other important stuff."

Q: What's the best test for liver function?

A: PT, because of factors II, VII, IX
if liver's damaged, you cant produce these factors.

If transaminase goes up and then down, everything's been fried and there's nothing left to kill in the liver

- A 25 year old male is brought in by EMS after sustaining multiple gunshot wounds to the chest and abdomen 15 minutes ago.
- Vitals BP 70/50, HR 120, RR 35
- Listless, responds to verbal stimuli
- Cool and clammy skin

Hypovolemic shock

Tx: Fluids, O2

- A 28 year old construction worker was pinned under debris and was extricated three hours later
- Vitals BP nonpalpable, HR 120, RR 10 and shallow
- Cyanosis peripherally
- Unresponsive to verbal stimuli

- Absent breath sounds in the right chest
- Asymmetric chest wall movement
- Tracheal deviation to the left
- Distended neck veins

He's in ***obstructive shock*** because he has tension pneumothorax

Tx: needle decompression and follow with a chest tube

- An 18 year old male with a history of nut allergies is brought in by EMS for severe respiratory difficulty, and a diffuse red rash.
- Vitals BP not obtainable, HR 120, RR 35

He's in ***anaphylactic shock***

Tx: Epi, steroids, H1/H2

- A 5 year old male is brought in by EMS for altered mental status, fever, and a petechial rash on the lower extremities.
- Vitals BP 60/palp, HR 140, RR 25 and shallow

He's in ***septic shock***

Tx: give ABX, pressors, fluids, tube him, get him to the unit.

Q: As for dosing epi?
A: Just start low and dial up the dose until you get a response

Q: why do you want to decrease afterload/BP in cardiogenic shock?
A: You want to decrease myocardial O2 consumption, reduce work for the heart.