TRENDS IN TRAUMA RESUSCITATION

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DISCLOSURES

“NO RELEVANT FINANCIAL RELATIONSHIP(S) EXIST.”
OBJECTIVES

DEFINE RESUSCITATION

ENDPOINTS OF RESUSCITATION

ACUTE COAGULOPATHY OF TRAUMA-SHOCK

THROMBOELASTOGRAPHY

MASSIVE TRANSFUSION

TRANEXAMIC ACID

SHOCK

INADEQUATE CELLULAR PERFUSION TO MAINTAIN CELL LIFE
RESUSCITATION

TO RESTORE CONSCIOUSNESS, VIGOR OR LIFE

END POINTS OF RESUSCITATION

BLOOD PRESSURE
MENTAL STATUS
PULSE
LACTATE
BASE DEFICIT
CVP
UOP
CARDIAC INDEX
SCVO₂/SVO₂
PULMONARY ARTERY OCCLUSION PRESSURE
THERE ARE NONE
MUST USE ALL AVAILABLE DATA
THE WHOLE IS GREATER THAN
THE SUM OF ITS PARTS
DYNAMIC PROCESS THAT
WARRANTS **CONSTANT**
RE-EVALUATION

OXYGEN DELIVERY

\[ DO_2 = CO \times C_A \cdot CO_2 \]
\[ = [(HR \times SV)] \times [(HG \times 1.34 \times S_A \cdot O_2) \]
\[ + (P_A \cdot O_2 \times 0.003)] \]

NOTICE THERE IS **NO PRESSURE**
VARIABLE
KEY POINT

PRESSURE ≠ FLOW ≠ PERFUSION

FINALLY

OXYGEN DELIVERY ≠ OXYGEN CONSUMPTION
LACTATE
OXYGEN DEBT...
MADE IN ANAEROBIC CONDITIONS...
RIGHT?

LACTATE
LACTIC ACIDOSIS MAKES US SICK...
RIGHT?
Fig. 10. Substrates and products of the ATPase reaction. This reaction is referred to as a hydrolysis reaction (ATP hydrolysis) due to the involvement of a water molecule. An oxygen atom, 2 electrons, and a proton from the water molecule are required to complete the free inorganic phosphate product of the reaction. The remaining protons from the water molecule is released into solution. Arrows pointing away from a bond represent bond/group removal. Arrows pointing to a bond represent addition of an atom/group.

LACTATE

BUFFER FOR INTRACELLULAR ACIDITY
USED FOR FUEL
WHEN CELL CAN’T MAKE ENOUGH LACTATE:

- PH DROPS MORE
- CELL DIES
- LACTATE SPILLS OUT (SOME IS TRANSPORTED OUT PRIOR VIA H+/LACTATE TRANSPORTER)
- HYDROGEN ION DERIVED FROM HYDROLYSIS OF ATP


LACTATE

LACTATE DERIVED FROM CONVERSION OF PYRUVATE VIA LDH

- OCCURS WHEN INSUFFICIENT O₂ PRESENT (SHOCK) TO ALLOW MITOCHONDRIA TO OXIDIZE GLUCOSE TO ATP
- ALSO OCCURS IN PRESENCE OF ADEQUATE OXYGEN

LACTATE OR LACTIC ACID

$P_{K_A}$ OF LACTIC ACID IS 3.85

LACTATE TO LACTIC ACID IS IN A RATIO OF 3548:1 AT $pH$ 7.4

ACID LOAD COMES FROM HYDROLYSIS OF ATP->ADP->AMP


LACTATE

INDIRECT MARKER OF SHOCK

MORE INDICATIVE OF ADRENERGIC DRIVE!

AKA...SOMETHING BAD IS PROBABLY GOING ON....
HEMORRHAGIC SHOCK RESUSCITATION

CRYSTALLOIDS ARE OUT
COLLOIDS ARE OUT
PERMISSIVE HYPOTENSION IS IN
BLOOD AND PLASMA ARE IN
COAGULOPATHY GUIDED RESUSCITATION

HEMORRHAGIC SHOCK RESUSCITATION

STOP THE BLEEDING
GIVE THEM WHAT THEY NEED....
AND NOT A DROP MORE
ACUTE COAGULOPATHY OF TRAUMA-SHOCK (ACoTS)

SYNDROME OF COAGULOPATHY THAT FAVORS BLEEDING PRESENT IN 25-30% OF TRAUMATICALLY INJURED ON PRESENTATION 1-2

8X AND 4X-INCREASED RISK OF MORTALITY AT 24 HOURS AND 30 DAYS.

MORE TRANSFUSION, LONGER ICU AND HOSPITAL LOS, MORE MOF3-4

REVERSAL REQUIRES FACTOR DRIVEN RESUSCITATION

4. INTENSIVE CARE MEDICINE. (37)4.572-82. 2011.

ACoTS

ALL MECHANISMS NOT KNOWN YET

DEPENDS ON:
DEGREE OF TISSUE INJURY
DEGREE OF HYPOPERFUSION

2 COMPONENTS ARE:
ACTIVATION OF PROTEIN C (APC) – BLOOD LOSS CAUSING HYPOPERFUSION

HYPERFIBRINOLYSIS – TISSUE DAMAGE CAUSES RELEASE OF TPA (TISSUE PLASMINOGEN ACTIVATOR)

APC
INACTIVATES FACTOR VIII AND V
INCREASES FIBRINOLYSIS
CONSUMES:
PLASMINOGEN ACTIVATOR INHIBITOR
THROMBIN ACTIVATABLE FIBRINOLYSIS INHIBITOR
5. MINERVA ANESTESIOLOGICA. 77;3:349-59.2011

ACoTS
WORSENED BY BUT NOT CAUSED BY:
DILUTION – CRYSTALLOID AND COLLOID
HYPOTHERMIA
ACIDEMIA
ACoTS

SHOULD BE CONSIDERED IN ALL:

SEVERELY INJURED PATIENTS
HIGH ENERGY TRAUMA
CLINICALLY ILL
EVIDENCE OF SHOCK


ACoTS

Trauma → Hemorrhage

Inflammation
Other Diseases
Medications
Genetics

Resuscitation

Shock

Acidemia
Fibrinolysis
Factor Consumption

Hypothermia

COAGULOPATHY

J TRAUMA. 2008;64:1211–1217
ACoTS AND SHOCK

ACoTS IS DOSE DEPENDENT

ACoTS SEVERITY BASED ON:

SEVERITY OF HYPOPERFUSION

BD > 6 MMOL/L\textsuperscript{7}
PT/PTT > 1.5 X NL\textsuperscript{8}


PERMISSIVE HYPOTENSION

CLOT LYSIS WHEN SBP > 80 MMHG

PERMISSIVE HYPOTENSION
SBP 80-90 MMHG
AND/OR
MAP 50 MMHG


WHAT ABOUT TBI?
HYPOTENSION INCREASES TBI MORTALITY
SBP TARGETED FLUID RESUSCITATION
WILL NOT IMPROVE SBP DURING ACTIVE HEMORRHAGE

NEED LOTS OF HEMORRAGHGE
ONLY WHEN ~50% BLOOD VOLUME LOST
= FALL IN SBP


GIVE MORE VOLUME
WHO CARES?

WORSE COAGULOPATHY
WORSE SIRS
MORE ARDS
MORE ACS
MORE PULM EDEMA
MORE DEATH

THROMBOELASTOGRAPHY

MECHANICAL GRAPHICAL DISPLAY OF CLOT FORMATION AND STABILITY

TAKES 30-45 MINUTES

REAL TIME DISPLAY OF CLOT

REQUIRES EQUIPMENT AND TRAINING FOR GRAPH INTERPRETATION

TEG

INCREASED R TIME → FFP
DECREASED ANGLE → CRYOPRECIPITATE
DECREASED MA → PLATELETS (CONSIDER DDAVP)
FIBRINOLYSIS → TRANEXAMIC
WHAT ABOUT INR?

IF INR > 1.5 IT IS VALUABLE

WILL MISS FIBRINOLYSIS

ONLY ASSESSES FIRST 60 SECONDS OF CLOTTING IN PLASMA

BLOOD IS WARMED TO RUN THE TEST

NOT A TRUE REFLECTION OF HEMOSTATIC ENVIRONMENT
TRANEXAMIC ACID

ANTIFIBRINOLYTIC

INHIBITS PLASMINOGEN ACTIVATION

DECREASES PLASMIN ACTIVITY

REDUCES CLOT LYSIS

TRANEXAMIC ACID

CRASH-2 TRIAL

LOWERED MORTALITY

GIVE WITHIN 3 HOURS OF INJURY

RECOMMENDED FOR ANY PATIENT YOU FEEL IS AT RISK FOR BLEEDING

IF YOU HAVE ACCESS TO TEG – USE IT TO GUIDE YOUR ADMINISTRATION

HYPERFIBRINOLYSIS

INCREASED RISK OF DEATH

TREATMENT INCLUDES TRANEXAMIC ACID

Fibrinolysis (UK, SK, or t-PA)
Presence of t-PA
R ~ Normal;
MA = Continuous decrease
LY30 > 7.5%; WBCLI30 < 97.5%;
Ly60 > 15.0%; WBCLI60 < 85%
WHAT DO WE DO?

STOP BLEEDING

USE HIGH RATIO TRANSFUSION
(1:1:1)

ALLOW PERMISSIVE HYPOTENSION*

* OUTSIDE OF HEAD INJURY

WHAT DO WE DO?

AVOID/CORRECT:

HYPOTHERMIA
ACIDEDEMIA
DILUTION
HYPOCALCEMIA
MASSIVE TRANSFUSION

ARBTRARY DEFINITION
2-3% OF CIVILIAN TRAUMA
MIMICS WHOLE BLOOD TRANSFUSION
1:1:1
PLASMA:PLATELETS:PRBCS
NO BEST RATIO OUTSIDE OF 1:1:1


HEMOSTATIC RESUSCITATION
USE OF AGGRESSIVE RATIOS OF BLOOD AND PRODUCTS TO ATTEMPT TO REVERSE ACoTS
UNABLE TO COMPLETELY REVERSE WITHOUT ARREST OF HEMORRHAGE

24. BARANIUK. INJURY. 45(9):1287-95.
PREDICTING MT

LOTS OF SCORING SYSTEMS
MCLAUGHLIN
TASH
ABC
PWH

NOT ALL PATIENTS HAVE THE DATA TO USE THESE

CURRENTLY LACK A GOLD STANDARD PREDICTOR

STABLE PATIENTS

DON’T NEED:

BLOOD
CRYSTALLOID
COLLOID

IT’S OK TO NOT INTERVENE
SUMMARY

STOP BLEEDING
ALLOW HYPOTENSION
MINIMIZE CRYSTALLOID AND COLLOID
ACoTS INCREASES MORTALITY
RELATED TO DEGREE OF HYPOPERFUSION AND INJURY

SUMMARY

EARLY USE OF COMPONENT BLOOD PRODUCT RESUSCITATION
TRANEXAMIC ACID GOOD AND GIVE EARLY
TEG CAN GUIDE BLOOD RESUSCITATION
LACTATE A MARKER OF SEVERITY OF ILLNESS
REFERENCES