

Vesalius SCALpel™ : Trauma (non-abdominal)

blunt trauma

no vital signs in the field, 0% survivors, ER thoracotomy futile

hemorrhage

duration of hypotension major predictor of mortality

permissive hypotension: maintain mean arterial pressure (MAP) ~90 to prevent

pressure-induced rebleeding from sealed vessels

class of hemorrhage (70Kg ~ 5L circulation blood volume)

I 15% = 750cc

II 15-30% = 750 to 1500cc (increased pulse, diastolic/narrowed pulse pressure)

III 30-40% > 1500 to 2000cc (hypotension)

IV > 40% > 2000cc

urine output diminishes from class II onward

transfusion: trauma pts < 55 without heart disease Hb 7-9 is adequate

shock

Df: inadequate tissue perfusion to maintain normal metabolic and nutritional functions

cellular hypoperfusion/hypoxia/injury

release of cytokines, superoxide radicals, prostaglandins, adhesion molecules

extensive injuries can lead to SIRS, MOF

normal systemic parameters

cardiac index (CI): > 4.5L/m/M²

mixed venous O₂ saturation: > 70%

O₂ delivery: > 600 mL/m/M²

O₂ consumption: > 170 mL/M²

right ventricular end diastolic volume index (RVEDVi) > 100mL/M²

gastric mucosal pH > 7.35

enteral feeding as effective as H₂ block and ppi's in lowering gastric pH

radiation

whole body gamma, neutron, high energy X-ray

hi sensitivity tissues: WBCs, hematopoietic, GI epithelium

intermediate: vascular endothelium, pulmonary endothelium, hepatocytes

resistant: myocytes, nerve

3 syndromes

1 hematopoietic (1-4 Gy): pancytopenia, opportunistic infection, thrombocytopenia (& bleeding)

2 GI (8-10 Gy): severe nausea/vomiting, watery diarrhea (acute onset, mucosal separation day 5), massive fluid loss, acute renal failure, death (in addition to type 1 changes)

3 neurovascular (15-50 Gy): extreme nervousness, confusion, massive vasodilatation, interval partial recovery @ 6h, then rapid mortality @ 72h

blast injury

primary injury: shock wave causes air embolus, pulmonary contusion, diffuse parenchymal hemorrhage
often late manifestations 12-24h

tympanic membrane most sensitive screening exam (retinal air embolus rarer)

initial CXR may be clear; decreasing pulse ox sat may be 1st sign pulmonary injury

secondary injury (body thrown against solid objects)

more likely causes of abdominal solid organ injury than primary shock wave

tertiary (flying debris)

neuro

traumatic brain injury (TBI)

severe: GCS 8 or below (coma)

control hypotension to minimize secondary injury, maintain oxygen saturation

ventriculostomy to monitor ICP, drain CSF if necessary

multimodality monitoring to intervene before secondary injury occurs

if ICP exceeds 20, institute measures to decrease, maintain cerebral perfusion pressure (CPP=MAP-ICP) >60

pressors OK in volume resuscitated patients

sedation and analgesia decrease metabolic demands

elevate head of bed 35-40 degrees

mannitol: osmotic diuresis, increases serum osmolality, draws fluid from brain

saline resuscitation increases vascular volume, cerebral perfusion and decreases blood viscosity improving cerebral circulation

hyperventilation to decrease pCO₂ no lower than 35, causes mild cerebral vasospasm, decreased cranial blood volume

further drop leads to ischemia

preexisting warfarin anticoagulation increases mortality of intracranial injury 4-5X

emergency reversal with rFVIIa in 9h (v 32h FFP and vit K)

Cushing's response: hypertension, bradycardia

definition of death

traditional cardiorespiratory criterion indicating loss of brain stem integrating function: ceases and does not resume communication, movement or breathing

'68 criteria:

presence of a cause compatible with brain death

absence of complicating medical condition

absence of drug intoxication, poison

no voluntary or involuntary movement except spinal reflexes

apnea with high pCO₂ without toxic or drug etiology

apnea test: hypercapnea (pCO₂ 60) with normal pO₂ should stimulate respiration

body temperature at least 36 degrees centigrade

below core temp of 32 lose brain stem reflexes
systemic blood pressure > 90
positive fluid balance > 6h
(EEG not sufficient, nuclear scan measure of cerebral blood flow not studied yet)

spinal cord injury

M 4:1, age 20-35

CT superior to multiplanar views for C-spine Dx

most contusion, compression, stretch, not transection

secondary injury: mitochondrial dysfunction, cyclooxygenase, lipoxygenase, nitric oxide synthetase activity, reactive oxygen species damage of cell membrane by lipid peroxidation

methyl prednisolone unlikely to improve function

neurogenic shock

complete cord transection, loss of sympathetic tone, hypotension

persistent bradycardia, absence of reflexes lasting days to weeks

complete transection above T6 20-70% incidence of autonomic dysreflexia (6% in 1st mo)

stimulus below the level of injury can provoke BP variability, hypertension, intracranial hemorrhage, seizure, death

afferent impulses result in generalized sympathetic response, vasoconstriction below the level of injury

stimuli: removal of hardware, bowel/bladder distention, pressure sores, menstruation, meds

Rx: remove trigger, short acting antihypertensive meds, phosphodiesterase inhibitor (sildenafil)

XS parasympathetic response after injury causes vasodilation, flushing, piloerection, nasal congestion, sweating, headache

peripheral nerve: digital nerve repair has better prognosis than larger nerves

extremity

mangled extremity severity score components: injury, ischemia, shock, age

score > 7 poor viability

no system reliably predicts long term functional outcome

energy imparted directly correlates with height of amputation

elderly may lack the energy to use prosthesis

absolute indications for amputation

ischemic limb/unreconstructable vasculature

hypothermia, acidosis, coagulopathy

massive crush

myoglobin & cytokine release lead to

SIRS, renal failure, ARDS

damage control when emergency craniotomy, thoracotomy, laparotomy necessary

avoid prolonged salvage attempts at vascular repair, unstable fracture

crush injury

exacerbated by reperfusion injury

CK level proportional to rhabdomyolysis

increased capillary permeability after reperfusion, edema, exacerbates hypovolemia from other injuries

phosphate and potassium released from injured cells, arrhythmias, death

35% incidence renal failure, multifactorial

hypovolemia: pre-renal azotemia

vasoconstrictors

myoglobin precipitation in tubules, direct toxicity

acid urine exacerbates precipitation

Rx: vigorous volume resuscitation, dilutes myoglobin load

mannitol, osmotic diuresis

bicarbonate to alkalinize urine

(no loop diuretic/lasix: acidifies urine and decreases vascular volume)

extremity vascular

explore without a-gram: obvious hemorrhage, expanding hematoma, distal ischemia

pulse deficit, ABI < 1 associated with significant vascular injury

most popliteal injuries associated with pulse deficit, so no angio

angio penetrating shoulder because collaterals mask injuries

vascular

minor intimal injury: < 50% diameter, no flow limitation, allow spontaneous healing

spasm adjacent to soft tissue injury no Rx

intervention (most injuries):

occlusion causing ischemia: to OR unless adequate collaterals

persistent hemorrhage: OR or interventional

AV fistula: coil or ligate, enlarge with time

large pseudoaneurysm: OR or US-guided compression, percutaneous thrombolysis

blunt carotid injury

hyperextension (most common), rotation: neuro and vascular complications

stretch carotid over TV processes C1-2 causing dissection

hi risk for blunt carotid injury: Horner's, basal skull fx, diffuse axonal injury, near hanging, LeFort II & III (not I), direct blow

hours to days: head/neckache, focal motor/sensory deficit

early Dx and Rx decreases stroke

delayed presentation up to 1y

4 vessel angio gold standard, duplex US

IV unfractionated heparin

LMWH at venous thrombolysis levels inadequate

if heparin contraindicated: antiplatelet (ASA, plavix)

deterioration in neuro status to OR or interventional

f/u testing > 10d for progression/evolution

5-40% mortality, 12-80% neuro morbidity/sequellae

25% of stenoses recanalize, 75% of thromboses

aortic injury

endovascular repair option in the face of other life threatening injuries

proximal subclavian injury stent avoids morbidity of trap door

popliteal trauma

mechanisms: tibial plateau fx, posterior knee dislocation, hyperextension/stretch
popliteal artery intimal tear, thrombosis; venous injury
priorities: stabilization, prompt reperfusion
if long delay temporary shunt, anticoagulate
31% amputation rate pulseless distal limb

thoracic

cause of 25% of trauma mortality

rib fx elderly: 2X risk morbidity and mortality; 19% increase for each rib

most elderly not candidates for epidural

hemothorax: initial output > 1,500cc to OR

innominate artery most common vascular injury in blunt trauma: median sternotomy

tension pneumo: kink superior and inferior vena cava, circulatory collapse

aorta

if cross clamp necessary limit to 30m to avoid spinal cord ischemia

10-15% paraplegia

cardiac contusion: echocardiogram, enzymes

more common than valvular damage

troponin I specific for cardiac injury

rises by 4-10 hours, 50% sensitivity within 4h, 100% @12

confirmatory, too late for emergency screening

peaks 4-8d

peak correlates with subsequent cardiac events

conservative Rx

severe can go into CHF, need aortic balloon pump

thoracotomy

R anterolateral rarely used

double lumen tube prevent blood and secretions compromising non-involved side

penetrating, tamponade, usually R vent injury

clinical or FAST Dx

needle pericardiocentesis temporizing only, not necessary for diagnosis

sternotomy for definitive repair of injury

flail chest: primary problem is pulmonary contusion

pain control: intercostal block, epidural

mechanical ventilation if necessary

diaphragmatic rupture

blunt 3X incidence of penetrating, usually (70%) L

80% present with dyspnea

X-ray NG tube above diaphragm

acute: explore through abdomen for associated abdominal injuries (85%), most spleen

chronic/late: repair through thorax because of adhesions

musculoskeletal

hip dislocation

CT little useful information before reduction

15% nerve injury, most sciatic neuropraxia

timing of reduction (1st few hrs) influences late osteonecrosis femoral head (10-50%)

transfusion reaction

earliest sign of transfusion reaction in the OR is diffuse bleeding

References:

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