Intra-Abdominal Hypertension: What’s in YOR Belly?

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Content Outline

Pathophysiology of IAH

Direct or Indirect injury at the cellular injury = release of mediators of SIRS

Initial findings are subtle – lactate levels increase 4-8 hours before clinical symptoms

Compensatory mechanisms keep cellular dysfunction local – when fails go global

Patients at Risk for IAH:

Capillary Leak – Fluid Resuscitation
Acidosis - pH > 7.2
Hypotension
Hypothermia
Polytransfusion - > 10 units blood / 24 hrs
Coagulopathy
Massive fluid resuscitation - > 5l / 24 hrs
Pancreatitis
Sepsis
Damage control laparotomy

Grades of IAH

• Grade I  12 – 15 mmHg
• Grade II  16 – 20 mmHg
• Grade III  21 – 25 mmHg
• Grade IV  > 25 mmHg

• Acute – develops within hours of a trauma or several days as a result of sepsis, capillary leak or critical illness
• Chronic – develops over months or years chronic ascites, obesity, pregnancy, tumors.

Identification – Measure bladder pressures

Treat IAP/APP per treatment algorithms

Algorithms for identification – treatment at WSACS.org