Abdominal Compartment Syndrome (ACS) & Intra-Abdominal Hypertension (IAH)
Case: Septic child

5 y.o. female presenting with septic syndrome

- Treatment: Fluids, vasopressors, antibiotics
- 24 hours into therapy develops worsening hypotension, oliguria, hypoxemia, hypercarbia. PIP rises from 20 to 40 cm
- IAP = 26 \rightarrow decompressive laparotomy
- Immediate resolution of renal, pulmonary and hemodynamic compromise
- 7 days later abdomen closed. Alive and well now.

DeCou, J Ped Surg 2000
Case: Complicated pulmonary embolism

46 yo male with PE on SQ enoxaparin

• Acutely decompensated, requiring IVF, vasopressors and blood for retroperitoneal hematoma
• Became anuric, BP dropped again, difficult to ventilate
• IAP measured at 50 mm Hg
• Decompression resulted in immediate resolution of anuria, hypotension and ventilator pressure issues
• Eventually discharged alive and well

Dabney, Intensive Care Med 2001
Case: Chest and Pelvic trauma

54 y.o. male fell 15 feet – broke ribs, pelvis, L-spine

• External fixation of pelvis, posterior spine stabilized
• 2 days later developed increasing pulmonary difficulty and was intubated
• Persistent pulmonary deterioration with hypotension requiring fluids, then dobutamine & adrenaline
• Pulmonary catheter showed good preload, but oliguria developed
• Bladder pressure 46 cm when measured → Decompressed
• Initially had immediate improvement of cardiopulmonary status, but progressively worsened and died 9 days later of MSOF.

Kopelman, J Trauma 2000
Case Points

- Intra-abdominal hypertension and ACS occur in many ICU settings (PICU, MICU, SICU).
- Trauma is not required for ACS to develop.
- Bladder pressure measurement is valuable in assessing whether IAH is contributing to organ dysfunction.
- IAP monitoring allows early detection of IAH, probably allowing clinicians to intervene earlier and avoid morbidity, mortality and prolonged ICU stay associated with delayed diagnosis of ACS.
Outline

• Definition – what is Intra-abdominal Hypertension and the Abdominal Compartment Syndrome?
• Causes
• Recent increase in recognition
• Physiologic Manifestations
• Prevalence
• Outcome
• Treatment
• Detection:
  – Bladder pressure monitoring
Abdominal Compartment Syndrome (ACS):

Definition

“.......the end result of a progressive, unchecked increase in intra-abdominal pressure from a myriad of disorders that eventually leads to multiple organ dysfunction.”

John Hunt, MD
Causes of Intra-abdominal Pressure (IAP) Elevation

- **Retroperitoneal**: pancreatitis, retroperitoneal or pelvic bleeding, contained AAA rupture, aortic surgery, abscess, visceral edema
- **Intraperitoneal**: intraperitoneal bleeding, AAA rupture, acute gastric dilatation, bowel obstruction, ileus, mesenteric venous obstruction, pneumoperitoneum, abdominal packing, abscess, visceral edema secondary to resuscitation (SIRS)
- **Abdominal Wall**: burn eschar, repair of gastroschisis or omphalocele, reduction of large hernias, pneumatic anti-shock garments, lap closure under tension, abdominal binders
- **Chronic**: central obesity, ascites, large abdominal tumors, PD, pregnancy
Recent increases in ACS Recognition
ACS Literature: Publication explosion
Intra-abdominal Hypertension & Abdominal Compartment Syndrome

Physiologic Sequelae
Physiologic Sequelae

Cardiac:

- Increased intra-abdominal pressures causes:
  - Compression of the vena cava with reduction in venous return to the heart
  - Elevated ITP with multiple negative cardiac effects
  - Compression of the aorta and systemic arterial tree

The result:

- Decreased cardiac output
- Increased cardiac workload
- Decreased SVO2, elevations of PAWP and CVP
- Cardiac insufficiency and eventually Cardiac arrest
Physiologic Sequelae

Detailed Cardiac effects - Preload impact:

- IAH pushes diaphragms up, resulting in compression of intra-thoracic organs and reduced intra-thoracic volume.
- This plus positive pressure ventilation lead to elevated intra-thoracic pressure (ITP).
- Elevated ITP impedes blood flow into the thorax.
- Elevated diaphragms compress vena cava as it enters chest.
- Elevated IAP compresses vena cava leading to pooling of blood in the pelvis and legs

**END RESULT:** Dramatic reduction in venous return to the heart (preload).
Physiologic Sequelae

Detailed Cardiac effects - Cardiac contractility:

- Reduction in thoracic cavity volume plus increase in ITP results in increased pulmonary artery pressures and reduced return of blood to left heart.
- Pulmonary hypertension leads to RV dilation, ventricular septal deviation into LV and higher RV wall tension. This leads to increased RV work and oxygen consumption.
- Reduced blood return to left heart plus obstructive impact of ventricular septum leads to reduced cardiac output.

**END RESULT:** Right coronary artery blood flow drop with resultant RV subendocardial ischemia and worsening cardiac dysfunction.
Physiologic Sequelae

Detailed Cardiac effects - Afterload impact:

• IAH causes some direct arterial compression resulting in increased afterload.

• More importantly, reduced cardiac output leads to an elevation of SVR in attempt to maintain blood pressure.

**END RESULT:** Elevated SVR leads to reduced blood flow to organs already suffering from ischemia and venous engorgement. They are now more ischemic and the capillary leak worsens, further exacerbating the syndrome.
Physiologic Sequelae

Detailed Cardiac effects - Hemodynamic monitoring:

- Elevated intra-thoracic pressure directly impacts traditional pressure-based cardiac filling measurements such as CVP and PAOP (wedge).
- These pressure measurements are erroneously elevated and do not reflect actual fluid resuscitation endpoints.

**END RESULT:** Failure to understand this, and reliance on pressure-based cardiac indices will lead to inadequate fluid resuscitation, persistent global organ ischemia and higher instances of MOF and death.
Physiologic Sequelae

Detailed Cardiac effects - Hemodynamic monitoring:

- Volumetric indices such as RVEDVI and GEDVI accurately reflect fluid volume status in the face of elevated IAP and ITP.

**END RESULT:** Focusing volume resuscitation end points on a volume-based index will result in improved cardiac function and reduced organ failure.
Physiologic Sequelae

Pulmonary:

- Increased intra-abdominal pressures causes:
  - Elevation of the diaphragms with reduction in lung volumes

The result:

- Elevated intrathoracic pressure (which further reduces venous return to heart, exacerbating cardiac problems)
- Increased peak pressures
- Reduced tidal volumes
- Barotrauma, atelectasis, hypoxia, hypercarbia
Physiologic Sequelae

Gastrointestinal:

- Increased intra-abdominal pressures causes:
  - Compression of mesenteric arteries
  - Congestion of mesenteric veins and capillaries
  - Reduced cardiac output to the gut

The result:
- Decreased gut perfusion, increased gut edema and leak
- Ischemia, necrosis, cytokine release
- Bacterial translocation
- Development and perpetuation of SIRS
- Further increases in intra-abdominal pressure
Physiologic Sequelae

Renal:

- **Elevated intra-abdominal pressure causes:**
  - Compression of renal veins and arteries
  - Reduced cardiac output to kidneys

**The Result:**

- Decreased renal artery and vein flow
- Renal congestion and edema
- Decreased glomerular filtration rate (GFR)
- Acute tubular necrosis (ATN)
- Renal failure, oliguria/anuria
Physiologic Sequelae

Neuro:

• Elevated intra-abdominal pressure causes:
  – Increases in intrathoracic pressure
  – Increases in superior vena cava (SVC) pressure with reduction in drainage of SVC into the thorax

The Result:
  – Increased central venous pressure and IJ pressure
  – Increased intracranial pressure
  – Decreased cerebral perfusion pressure
  – Cerebral edema, brain anoxia, brain injury
Physiologic Sequelae

Miscellaneous

• Elevated intra-abdominal pressure causes:
  – Reduces perfusion of surgical and traumatic wounds
  – Reduced blood flow to critical organs and tissues

The Result:
  – Poor wound healing and dehiscence
  – Coagulopathy
  – Immunosuppression
Physiologic Sequelae at increasing pressures

0-9mm Hg:

– Cytokine release & capillary leak

– 3rd spacing of resuscitative fluid

– Decreasing venous return and preload

Ridings Surg Forum. 1994

– Early effects on ICP and CPP

Bloomfield Crit Care Med 1997
Physiologic Sequelae at increasing pressures

10-15mm Hg:

– Abdominal wall perfusion decreases 42%
  Diebel Am Surg 1992

– Marked reduction in intestinal and intra-abdominal organ blood flow leading to regional acidosis and free radical formation.
  Schwatre Anesthesiology 2004
  Deibel Trauma 1992

– Bacterial Translocation across bowel wall
  Eleftheriadis World J Surg 1996
  Deibel J Trauma 1997
Physiologic Sequelae at increasing pressures

16-25mm Hg:

– Worsening hemodynamics:
  • Markedly decreased venous return, CO and splanchnic perfusion
  • Increased SVR, CVP, PAWP

– Pulmonary Spiral:
  • Decreased TLC, FRC, RV.
  • Increased vent pressures, hypercapnia, hypoxia-
    – Ridings et al
Physiologic Sequelae at increasing pressures

16-25mm Hg:

- Bowel ischemia
  - Reduction to 61% of baseline mucosal blood flow - Deibel et al
  - Increasing gut acidosis - Timmer, Ivatury et al

- Renal Dysfunction:
  - Oliguria, anuria, etc

- Cerebral perfusion problems
  - Worsening CPP with increasing ICP
Physiologic Sequelae at increasing pressures

26-40mm Hg:

- Hemodynamic collapse, worsening acidosis, hypoxia, hypercapnia, anuria.
- Flow in Celiac A. 58%, SMA 39%, Renal A. 30%
  Barnes, AM J Physiol 1985
- 80% reduction in flow to abdominal wall
  Deibel et al
- Inability to oxygenate, ventilate or resuscitate
Circling the Drain

Intra-abdominal Pressure

Capillary leak
Mucosal Breakdown
Decreased O2 delivery

Free radical formation (Multi-System Organ Failure) Bacterial translocation

Acidosis
Anaerobic metabolism
How good is clinical judgment for detecting elevated IAP?


– Prospective, blinded trial - Staff physician judgment
– Results: Less than 50% of the time was the clinician able to determine when IAP was elevated.

“These findings suggest that more routine measurements of bladder pressure in patients at risk for intra-abdominal hypertension should be performed.”
How common is this syndrome?

Malbrain, Intensive Care Medicine (2004):

– Prospective, multi-center trial
  • 13 ICU’s, 6 countries

– Every patient in ICU with expected stay > 24 hours had IAP measured q6 hours.
  • 97 patients entered
How common is this syndrome?

Malbrain, Intensive Care Medicine (2004):

<table>
<thead>
<tr>
<th>Abdominal pressure:</th>
<th>Total Prevalence</th>
<th>MICU prevalence</th>
<th>SICU prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>IAP &gt; 12</td>
<td>58.8%</td>
<td>54.4%</td>
<td>65%</td>
</tr>
<tr>
<td>IAP &gt; 15</td>
<td>28.9%</td>
<td>29.8%</td>
<td>27.5%</td>
</tr>
<tr>
<td>IAP &gt; 20 plus organ failure</td>
<td>8.2%</td>
<td>10.5%</td>
<td>5.0%</td>
</tr>
</tbody>
</table>
Malbrain, Intensive Care Medicine (2004):

“Our study suggests that there is no specific type of patient or disease or treatment that reliably indicates when IAP needs to be measured, or when measurement is not necessary in a mixed ICU population. Indeed, it seems that .. IAP should be routinely measured.”
Does IAH / ACS affect patient outcome?

Tao, 2003: Diagnosis and management of severe acute pancreatitis complicated with abdominal compartment syndrome.

- 23 cases of severe pancreatitis with ACS
  - 18 cases were emergency decompressed: 16.7% mortality
  - 5 cases were not decompressed: 80% mortality
  - All cases with decompression within 5 hours or less of diagnosis survived.

“Early diagnosis, emergency decompressive celiotomy and temporary abdominal closure .. are the keys to the management of the condition.”
Does IAH / ACS affect patient outcome?

Pupelis, 2002: Clinical significance of increased intra-abdominal pressure in severe acute pancreatitis.

37 cases of severe pancreatitis

- 26 cases with IAP < 25 mm Hg:
  19% SIRS & MODS  0 % mortality  Mean ICU LOS 9 days

- 11 cases with IAP > 25 mm Hg:
  64% SIRS & MODS  36 % mortality  Mean ICU LOS 21 days

“Routine measurement of the intra-abdominal pressure is rational in the clinical setting of the ICU and gives additional criteria for the evaluation of the clinical course and the effectiveness of the treatment.”
Does IAH / ACS affect patient outcome?

Raeburn 2001: The abdominal compartment syndrome is a morbid complication of post-injury damage control surgery.

- 77 patients monitored for IAH / ACS
  - 36% developed IAP > 20 mm Hg:
    - Longer ICU LOS
    - Longer ventilator times
    - Higher MSOF
    - Higher mortality
Does IAH / ACS affect patient outcome?


- 70 patients with monitored for IAH > 25 mm Hg
  - 25 had facial closure at time of surgery:
    - 52% developed IAH > 25
    - 39% Died
  - 45 cases had abdomen left “open”:
    - 22% developed IAH > 25
    - 10.6% Died
Does IAH / ACS affect patient outcome?


Prospective observational study in 108 liver transplants

- 32% developed IAP > 25 mm Hg:
  - Renal failure in 32%; permanent dialysis 9%, higher mortality

- 68% with IAP < 25 mm Hg:
  - Renal failure 8%; permanent dialysis 0%

“The critical IAP values… with the best sensitivity specificity, were 23 mm Hg for postoperative ventilatory delayed weaning (P < .05), 24 mm Hg for renal dysfunction (P < .05), and 25 mm Hg for death (P < .01).”
Does IAH / ACS affect patient outcome?

Sugrue, Arch Surg, 1999: Intra-abdominal hypertension is an independent cause of postoperative renal impairment.

- Large Prospective study investigating IAP monitoring
- 263 patients monitored for IAH > 18 mm Hg
  - 156 cases with IAP < 18: 14.1% renal impairment
  - 107 cases (41%) with IAP > 18: 32.7% renal impairment

This study shows that IAP is “an independent cause of renal impairment, and it ranks in importance after hypotension, sepsis, and age older than 60 years.”
Does IAH / ACS affect patient outcome?

Michael Sugrue, MD – “World expert” in IAH and ACS (over 1800 measurements done, 10 publications)

• “Evidence is clear” regarding renal impairment.

• Not every patient will respond to decompression –
  – About 25-30% benefit if some delay in decompression occurs
  – Probably 60-70% benefit if you decompress early
  – Still has substantial morbidity and mortality.

• Evolving area of research / understanding
Does IAH / ACS affect patient outcome?

Points:

• IAH and ACS increase morbidity, mortality and ICU length of stay
• Early detection and intervention reduces these complications in some patients
• Clinical signs of ACS are unreliable and only show up late in clinical course
• Monitoring early (not waiting for high clinical suspicion) all high risk patients allows early detection and early intervention.
IAH/ACS Management

• Close Monitoring
  – Early identification of those at risk
  – Serial evaluation of bladder pressures, vent settings, hemodynamics, I/O’s, labs
  – Judicious fluid resuscitation
  – Beware of monitoring pitfalls (CVP, SVR, CO, PAOP, peak pressures, UOP, etc are all affected and may be misleading)
IAH/ACS Management

- **Fluids – two edged sword**
  - Fluids will absolutely improve cardiac indices if the patient has inadequate RVEDVI - so early in the course they are necessary.
  - However, over resuscitation will lead to worsened edema and elevations in IAP - close monitoring is needed.

- **Paralytics**

- **Cathartics / enema to clear bowel?**

- **Paracentesis**
  - Need significant free fluid on US
  - Can place temporary catheter

  Latenser et al J Burn Care Rehabil 23:190 2002

- **Decompressive laparotomy**
IAH/ACS Management

Decompression Laparotomy:

• Err on the side of early vs late intervention
  – Less bowel edema or cell damage, better chance of early closure and early recovery.

• Can be performed bedside for unstable patients
IAH/ACS Management

Decompression Laparotomy:

Rigid Abdomen in ACS

Post decompressive laparotomy
IAH/ACS Management

Decompression Laparotomy:

Post-operative dressing

Several days post-op
Decompression Laparotomy:

- Anticipate significant physiologic changes at decompression if you have waited too long
  - Cardiovascular: asystole, dysrhythmias, hypertension—turn down pressors
  - Pulmonary: Barotrauma, hyperoxia-dial down pressures and Fi02, pulmonary embolism
  - Acidosis from reperfusion
  - Renal/Fluids: Ongoing 3rd spacing and need for resuscitation
IAH/ACS Management

Post-laparotomy ACS:

• Same problem-same treatment
  – Be aware that ACS can recur following a decompression laparotomy
  – Score or replace dressing to treat recurrence

Gracias et al Arch Surg 2002;137:1298-1300
Intra-Abdominal Pressure Monitoring
Intra-Abdominal Pressure Monitoring

Bladder pressure monitoring through the Foley catheter is:

- Comparable to direct intraperitoneal pressure measurements, but is non-invasive (Bailey, Crit Care 2000)
- More reliable and reproducible than clinical judgment (Kirkpatrick, CJS 2000; Sugrue World J Surg 2002)
- Becoming a standard method for monitoring abdominal pressures (Fritsch, Crit Care Nurse 2000)
- Allows early detection of intra-abdominal hypertension, allowing intervention before ACS develops (Sugrue, Intensive Care Med 2002)
Intra-Abdominal Pressure Monitoring

• How much fluid should be infused into the bladder?
  – The minimal amount of fluid required to obtain a reliable IAP measurement.
  – Too much fluid leads to bladder over distention and compliance issues (see next slide)
  – Currently it appears that one never needs more than 50 ml in an adult, less is probably adequate
Bladder compliance curve

Non-compliant bladder: Measured pressure increases as volumes exceed 75 ml of infusion.

Compliant bladder: Measured pressure changes very little with higher volumes of fluid infusion.
Fluid-Column Manometry

- Simple method of measuring bladder pressure via fluid column in a Foley catheter.
- Requires disconnection of the Foley to instill saline and careful bending of the Foley to ensure accurate measurement.

Manometry Accuracy Study

<table>
<thead>
<tr>
<th>10 cm H2O column: mean±STDV (range)</th>
<th>20 cm H2O column: mean±STDV (range)</th>
<th>30 cm H2O column: mean±STDV (range)</th>
<th>40 cm H2O column: mean±STDV (range)</th>
<th>50 cm H2O column: mean±STDV (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>10 ml infused</strong></td>
<td>12.0±2.4 (10-15.5)</td>
<td>20.6±2.2 (16.5-25)</td>
<td>22.6±1.6 (19.5-24)</td>
<td>23.6±2.6 (21-26)</td>
</tr>
<tr>
<td><strong>20 ml infused</strong></td>
<td>17.7±7 (10-33)</td>
<td>23.4±4.6 (20-32)</td>
<td>33.6±4 (29-40.5)</td>
<td>38.8±1.1 (37-40.5)</td>
</tr>
<tr>
<td><strong>30 ml infused</strong></td>
<td>20.8±12.4 (10-42)</td>
<td>29.7±9.4 (20-46)</td>
<td>37.6±8.4 (30-54)</td>
<td>41.1±0.9 (40-43)</td>
</tr>
<tr>
<td><strong>40 ml infused</strong></td>
<td>17.7±8 (10-31)</td>
<td>28.1±9.9 (20.5-45.5)</td>
<td>39.5±10.1 (30-57)</td>
<td>41.1±0.7 (40-42)</td>
</tr>
<tr>
<td><strong>50 ml infused</strong></td>
<td>16±9.6 (10-39)</td>
<td>26.3±9.1 (20-42.5)</td>
<td>35.9±9 (29.5-56)</td>
<td>40.4±0.7 (39-41)</td>
</tr>
</tbody>
</table>

Inadequate volume results in falsely low pressure when pressure is elevated.

Siphon effect results in falsely elevated pressure when pressure is low.
Fluid-Column Manometry

Problems:

Failure to pay extreme attention to detail may lead to errors

- Siphon effect leads to false elevations
- Pinching of tube can lead to inability to equilibrate
- Failure to hold tube vertical can lead to inaccuracies
- Inadequate volume of infusion will lead to falsely low measurements

Need to infuse urine back into patient
“Home Made” Pressure Transducer Technique

Home-made assembly:

- Transducer
- 2 stopcocks
- 1 60 ml syringe,
- 1 tubing with saline bag spike / luer connector
- 1 tubing with luer both ends
- 1 needle / angiocath
- Clamp for Foley

Assembled sterilely in proper fashion
“Home Made” Pressure Transducer Technique

**PROBLEMS:**

- Home-made:
  - No standardization
  - Sterility issues

- Data reproducibility / variability issues and costs / morbidity of inaccurate information

- Requires recurrent penetration of closed urinary drain system with needle or some form of connection. Leaks, re-zeroing problems

- Time consuming data acquisition limits utility
“Home Made” Pressure Transducer Technique

QUESTIONS TO CONSIDER:

• Who is “building” your home-made devices
  – Are all nurses adept? Is it done in sterile fashion

• How reproducible is the data you obtain
  – Are there variations? Does this lead to confusion regarding care and therapy?

• How often are you measuring IAP?
  – Most only measure 2-4 time a day due to hassle, but IAH occurs rather quickly in many cases

• What are the hidden costs in delayed or missed diagnosis, time requirements of homemade device, etc
What is the big deal about data reproducibility?

• Reliable, reproducible data is imperative to properly manage critically ill patients whose physiology is in a state of rapid fluctuation.

• Inaccurate or flawed data may lead:
  – to errors of omission (failure to recognize a disease process, leading to failure to intervene)
  – errors of commission (inaccurate identification of a disease process that does not exist, leading to unnecessary interventions).
What is the big deal about data reproducibility?

Failure to identify IAH / ACS may lead to:

– Organ failure &/or Death
– Prolonged ICU length of stay

Misidentification of IAH / ACS may lead to:

– Use of inaccurate CVP, cardiac data with resultant errors in fluid/pressor management
– Unnecessary decompressive laparotomy.

In either case, substantial increases in morbidity and cost of care may occur.
AbViser

Intra-Abdominal Pressure Monitoring Kit

Closed system in-line with the Foley catheter.

- Once attached it is left in place during entire time IAP is measured.
- A simple valve twist allows fluid infusion and pressure measurement.
- Twist back to drain.
- 30 seconds to measure IAP
AbViser
Intra-Abdominal Pressure Monitoring Kit

Advantages:
• Standardized measurement
• No reproducibility errors
• Ease & simplicity of use
• Time savings
  – 30 seconds to get data.
• Closed system
• No needles
• No contamination risks
Study Aims: Evaluate intra and inter-observer variability when intra-abdominal pressures are measured using the AbViser - a newly developed intra-abdominal pressure monitoring kit.

Two phases:
- **Bench top model** - eliminates all uncontrolled variables to ensure device is accurate
- **Clinical setting** - validates device data reproducibility in real environment.
AbViser Reproducibility Study: Methods

**Bench-Top:**

**Gold standard pressure** -

Water column depth created to mimic pressures of 5, 10, 15, 20, 25, 30 and 40 mm Hg

- 1.36 cm water equals 1 mm Hg

**Study variable measured** -

Transduced pressure recorded on hemodynamic monitor using AbViser intra-abdominal pressure monitoring kit.

- 11 participant, 5 measurements each

**Clinical Inter-observer variability:**

"Gold standard pressure" -

Primary nurse measured IAP on patient using AbViser, then drained bladder

**Study variable measured** -

Second nurse measured IAP on same patient within 20 minutes of first measurement and the measurements were compared.
**AbViser Reproducibility Study: Results**

**Bench-top data:**
- No differences greater than 2 mm Hg occurred at any pressure.
- **Mean Differences**
  - Actual vs. Measured Pressure
    - 0.206 (95%CI 0.165, 0.248)
  - (Volume = 50 ml)

**Clinical Data:**
- Measurement differences greater than 3 mm Hg were uncommon.
- **Mean differences**
  - First nurse vs. second nurse
    - 0.265 (CI -0.217 to 0.748)
    - Limits of agreement (Reference Range for difference): -3.096 to 3.626
AbViser: Reproducibility Study

Bench - 15 mm Hg

Clinical - Interobserver
Conclusions

The AbViser, a newly developed intra-abdominal pressure monitoring kit:

- Provides very accurate pressure measurements when compared to a known gold standard.
- Provides reproducible pressure measurements without little or no intra or interobserver variability both in an intra-abdominal pressure model as well as in a clinical setting.
- As would be predicted, slight increases in variability occur in the clinical setting, but these are rarely of clinical significance.
University of Utah: IAP monitoring algorithm

- Entry criteria defined in table
- Nurse is empowered to enter any patient fulfilling these criteria

Indications for IAP monitoring:

Sepsis / SIRS / Ischemia Reperfusion
- Sepsis and resuscitation with > 6 liters crystalloid/collod or > 4 units blood in 8 hours\textsuperscript{1,3}
- Pancreatitis\textsuperscript{4, 5}
- Peritonitis\textsuperscript{2, 3, 6}
- Ileus / Bowel obstruction\textsuperscript{2, 3, 7, 8}
- Mesenteric ischemia / necrosis\textsuperscript{9}

Visceral compression / Reduction
- Large ascites/ peritoneal dialysis\textsuperscript{3, 6, 10, 11}
- Retroperitoneal / abdominal wall bleeding\textsuperscript{12, 13}
- Large Abdominal tumor\textsuperscript{6, 10}
- Laparotomy closed under tension\textsuperscript{14, 15}
- Gastrochisis / Omphalocele\textsuperscript{16-18}

Surgical
- Intra-operative fluid balance > 6 liters\textsuperscript{19}
- Abdominal aortic aneurysm repair\textsuperscript{20-23}

Trauma
- Shock requiring resuscitation (ischemia-reperfusion)\textsuperscript{3, 24, 25}
- Damage Control Laparotomy\textsuperscript{26-28}
- Multiple trauma with or without abdominal trauma requiring resuscitation with > 6 liters crystalloid/collod or > 4 units blood in 8 hours\textsuperscript{24, 25, 27, 29, 30}
- Major burns (> 25%)\textsuperscript{11, 31-34}
University of Utah:
IAP monitoring algorithm

- IAP monitoring Q1-2 hours for first 12 hours
  - IAP consistently <12 mm Hg
    - Reduce frequency of IAP measurements to Q4-6 hours for additional 24 hours
  - IAP 12 to 15 mm Hg
    - Continue close monitoring
  - IAP 15-20 mm Hg with no evidence of organ dysfunction/ischemia
  - IAP >20 mm Hg OR IAP >15 mm Hg with evidence of organ dysfunction/ischemia
    - Consider Medical Management of IAH
      - Neuromuscular blockade
      - Paracentesis if free fluid documented
      - Other options:
        - Gastric suction
        - Rectal tube with enemas
  - “Additional Insult” - Patient develops new indications for IAP monitoring
    - IAP pressures remain <12 mm Hg
      - Discontinue IAP monitoring
  - Notify House Officer
    - MD CONSIDER SURGICAL DECOMPRESSION:
      - Through abdominal wall if this is initial decompression
      - Through Abdominal dressing if recurrent decompression

Page 1 of 2 (References on back)
QUESTIONS?