

## Guideline on the assessment and management of intra-abdominal hypertension

AIM: To provide guidance on the management of IAH and ACS.  
SCOPE: All adult ICUs within Brighton and Sussex University Hospitals

*Suggested Assessment Flowchart,*  
taken from WSACS, the Abdominal Compartment Society (2013)

### INTRA-ABDOMINAL HYPERTENSION ASSESSMENT ALGORITHM

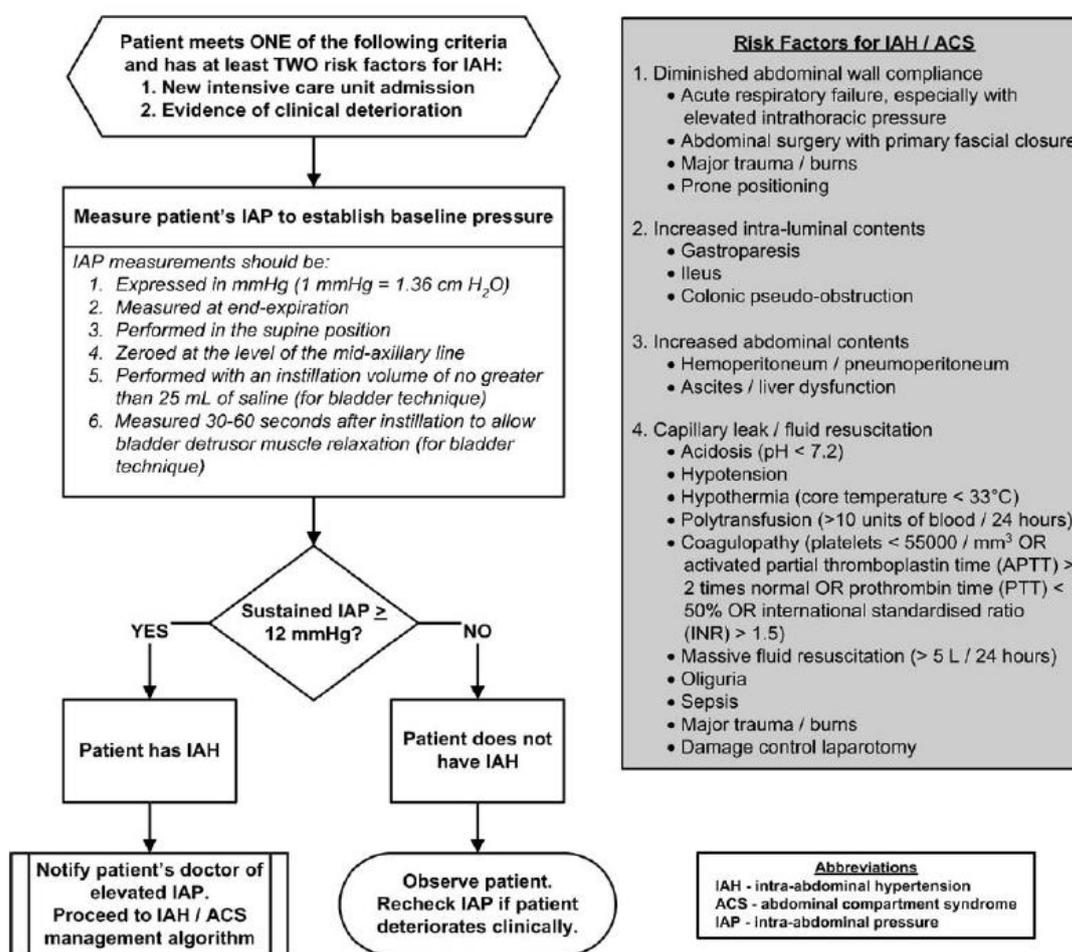
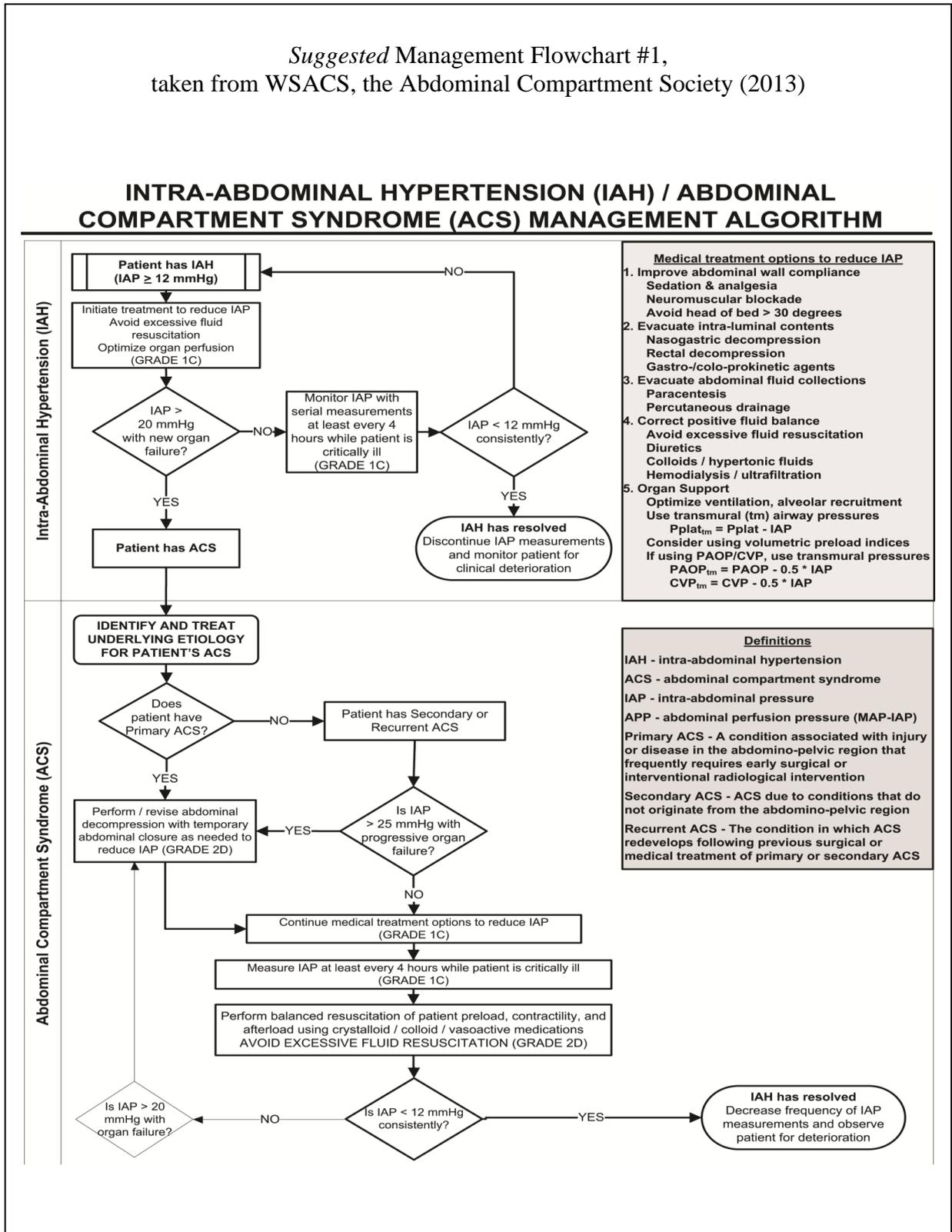


Fig. 1 Intra-abdominal hypertension assessment algorithm



**The  
Abdominal  
Compartment  
Society**

*Suggested Management Flowchart #1,*  
taken from WSACS, the Abdominal Compartment Society (2013)



## 1. INTRODUCTION

The aims of these guidelines are to identify the correct technique for measurement of intra-abdominal pressure in BSUH critical care units, and to suggest options for subsequent management of intra-abdominal hypertension and the abdominal compartment syndrome. Although much of the guidance is taken from WSACS publications, clinicians should use their own judgement as to the correct management for the patient in front of them.

## 2. PROCESS

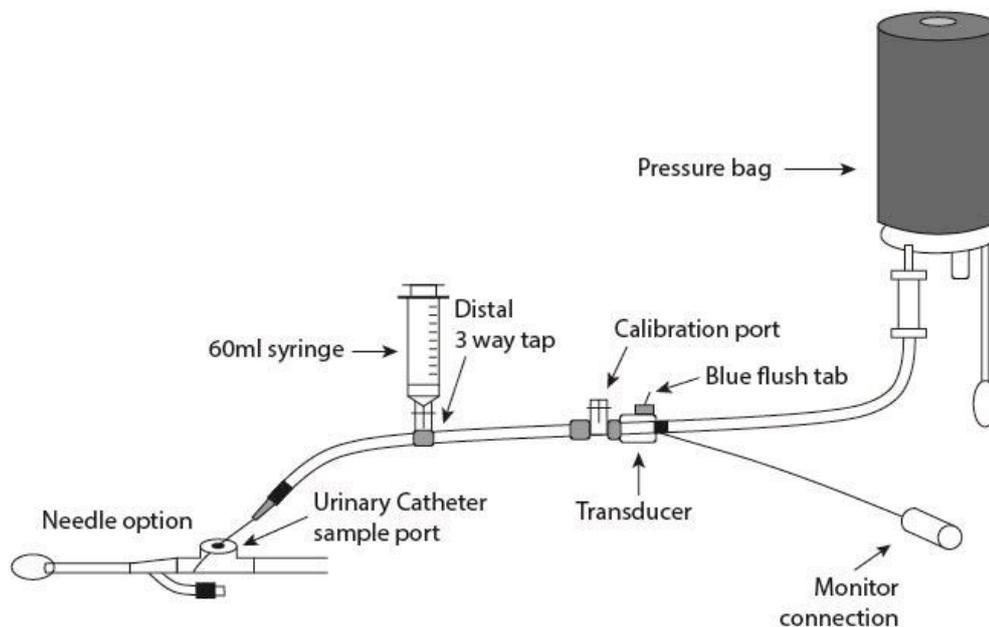
Recommendation (Action)	Justification (Rationale)
Consider risk factors for the development of IAH and ACS	<p><i>Diminished abdominal wall compliance</i> Abdominal surgery Major Trauma/ Major burns Prone positioning</p> <p><i>Increased intra-luminal contents</i> Gastroparesis/gastric distension/ileus Colonic pseudo-obstruction / volvulus</p> <p><i>Increased intra-abdominal contents</i> Acute pancreatitis Haemoperitoneum/pneumoperitoneum or intra-peritoneal fluid collections Intra-abdominal infection/abscess/tumours Liver dysfunction/cirrhosis with ascites</p> <p><i>Capillary leak/fluid resuscitation</i> Acidosis / hypothermia Damage control laparotomy Massive fluid resuscitation or positive fluid balance</p> <p><i>Others/miscellaneous</i> Age, bacteremia Coagulopathy Mechanical ventilation Obesity or increased body mass index Sepsis / shock or hypotension</p>

Recommendation (Action)	Justification (Rationale)																		
<p><b>Table 2</b> Physiological changes associated with intra-abdominal hypertension and abdominal compartment syndrome<sup>1,2</sup></p> <table border="1"> <thead> <tr> <th data-bbox="204 331 635 365">Organ system/physiological change</th> <th data-bbox="667 331 802 365">IAP range*</th> <th data-bbox="1002 331 1114 365">Explanation</th> </tr> </thead> <tbody> <tr> <td data-bbox="204 387 635 589"> <b>Cardiovascular</b>                      ↑ Central venous pressure, pulmonary artery pressure, pulmonary capillary wedge pressure, systemic vascular resistance                      ↓ Cardiac output (more pronounced with hypovolemia)                      ↓ Blood pressure or normal blood pressure                      ↓ Venous return from lower extremities (risk of deep venous thrombosis)                 </td> <td data-bbox="667 387 802 421">Low to moderate</td> <td data-bbox="826 387 1289 566">Increased abdominal pressure prevents venous return (preload reduction) and impedes arterial outflow (increase in afterload). Transmitted back pressure from the abdominal cavity falsely elevates central venous pressure, pulmonary artery pressure, pulmonary capillary wedge pressure, pulmonary vascular resistance, and systemic vascular resistance.</td> </tr> <tr> <td data-bbox="204 611 635 723"> <b>Pulmonary</b>                      ↑ Intrathoracic pressures                      ↑ Peak inspiratory pressures                      ↓ Tidal volume → hypercarbia + ↓ Pao<sub>2</sub>                      ↓ Compliance                 </td> <td data-bbox="667 611 802 645">Moderate to severe</td> <td data-bbox="826 611 1289 678">Increased intra-abdominal pressure causes increase in intrathoracic pressure and limits diaphragm excursion, resulting in hypoventilation and hypoxia.</td> </tr> <tr> <td data-bbox="204 745 635 813"> <b>Renal</b>                      ↓ Renal blood flow → ↓ glomerular filtration rate → ↓ urine output                 </td> <td data-bbox="667 745 802 779">Low to moderate</td> <td data-bbox="826 745 1289 813">Increased intra-abdominal pressure compresses the renal parenchyma, reducing blood flow and urine output.</td> </tr> <tr> <td data-bbox="204 835 635 902"> <b>Neurological</b>                      ↑ Intracranial pressure                      ↓ Cerebral perfusion pressure                 </td> <td data-bbox="667 835 802 869">Low to moderate</td> <td data-bbox="826 835 1289 902">Increased intra-abdominal pressure impedes venous outflow from the brain, increasing cerebral venous congestion.</td> </tr> <tr> <td data-bbox="204 925 635 1037"> <b>Gastrointestinal or hepatic</b>                      ↓ Celiac and portal blood flow                      ↓ Lactate clearance                      ↓ Mucosal blood flow → ↓ intramucosal pH (acidosis)                 </td> <td data-bbox="667 925 802 958">Low to moderate</td> <td data-bbox="826 925 1289 969">Increased intra-abdominal pressure reduces perfusion to the abdominal organs.</td> </tr> </tbody> </table> <p>*IAP range indicates range of intra-abdominal pressures at which initial effects on organ system occur; ↑, increase; ↓, decrease.</p>		Organ system/physiological change	IAP range*	Explanation	<b>Cardiovascular</b> ↑ Central venous pressure, pulmonary artery pressure, pulmonary capillary wedge pressure, systemic vascular resistance ↓ Cardiac output (more pronounced with hypovolemia) ↓ Blood pressure or normal blood pressure ↓ Venous return from lower extremities (risk of deep venous thrombosis)	Low to moderate	Increased abdominal pressure prevents venous return (preload reduction) and impedes arterial outflow (increase in afterload). Transmitted back pressure from the abdominal cavity falsely elevates central venous pressure, pulmonary artery pressure, pulmonary capillary wedge pressure, pulmonary vascular resistance, and systemic vascular resistance.	<b>Pulmonary</b> ↑ Intrathoracic pressures ↑ Peak inspiratory pressures ↓ Tidal volume → hypercarbia + ↓ Pao <sub>2</sub> ↓ Compliance	Moderate to severe	Increased intra-abdominal pressure causes increase in intrathoracic pressure and limits diaphragm excursion, resulting in hypoventilation and hypoxia.	<b>Renal</b> ↓ Renal blood flow → ↓ glomerular filtration rate → ↓ urine output	Low to moderate	Increased intra-abdominal pressure compresses the renal parenchyma, reducing blood flow and urine output.	<b>Neurological</b> ↑ Intracranial pressure ↓ Cerebral perfusion pressure	Low to moderate	Increased intra-abdominal pressure impedes venous outflow from the brain, increasing cerebral venous congestion.	<b>Gastrointestinal or hepatic</b> ↓ Celiac and portal blood flow ↓ Lactate clearance ↓ Mucosal blood flow → ↓ intramucosal pH (acidosis)	Low to moderate	Increased intra-abdominal pressure reduces perfusion to the abdominal organs.
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<p><b>Measure intra-abdominal pressure (IAP) in patients at high risk of, or suspected to have, abdominal compartment syndrome (ACS).</b></p>	<p>The physiological consequences of increased intra-abdominal pressure include the following :</p>																		
<p><b>Assemble the necessary equipment to measure IAP (via urinary catheter)</b></p>	<p>Invasive monitoring capability                      500ml Normal saline and pressure bag                      Standard pressure transducer and line with 2 x 3-way taps                      Transducer connection cable                      50 ml Luer-Lock syringe                      Clamp                      2% chlorhexidine swab                      18G (green needle)</p>																		

## Procedure

1. Wash hands and follow universal precautions. If the patient is awake, explain the procedure. If the patient is sedated, ensure adequate sedation to minimize movement.
2. Assemble and pressurize the transducer system

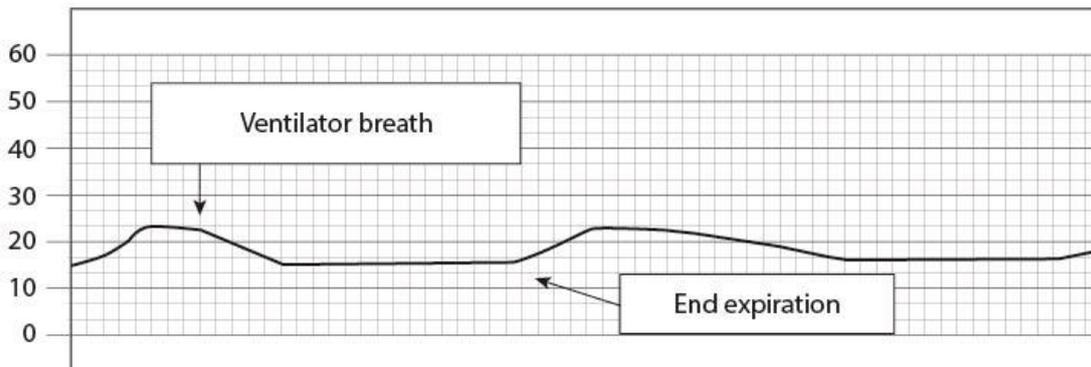
Figure 2



3. Attach the 50 ml syringe to the distal 3-way tap and attach the needle to the end of the transducer tubing (Fig 2)
4. Connect the transducer to the pressure module of the monitoring system and ensure a waveform appears on the screen. Select a scale in the <math><50\text{ mmHg}</math> range. Zero the pressure module at the mid-axillary line of the patient & mark for future reference.
5. The patient should be placed supine, or if there is a clinical contra-indication, as flat as possible. The position of the patient during the measurement should be noted in the care record and further measurements performed in the same position.

6. Clamp the bladder drainage system just distal to the connection of the catheter to the drainage bag.
7. Clean the sampling port on the catheter with a 2% chlorhexidine wipe and aseptically insert the needle into the port.
8. Turn the 3-way tap attached to the syringe off to the patient and open to the pressure bag and syringe. Use the fast-flush pigtail catheter to fill the syringe to 25 ml.
9. Turn the 3-way tap off to the pressure bag and inject the 25ml of saline into the bladder.
10. Using the proximal 3-way tap level and zero the transducer at the mid-axillary line.
11. Allow 30-60 seconds for the pressures to equalise and then measure the intra-abdominal pressure from the waveform on the monitor. Measurements should be at end-expiration as in the example below where the intra-abdominal pressure is 15mmHg. (Fig 3)

Figure 3

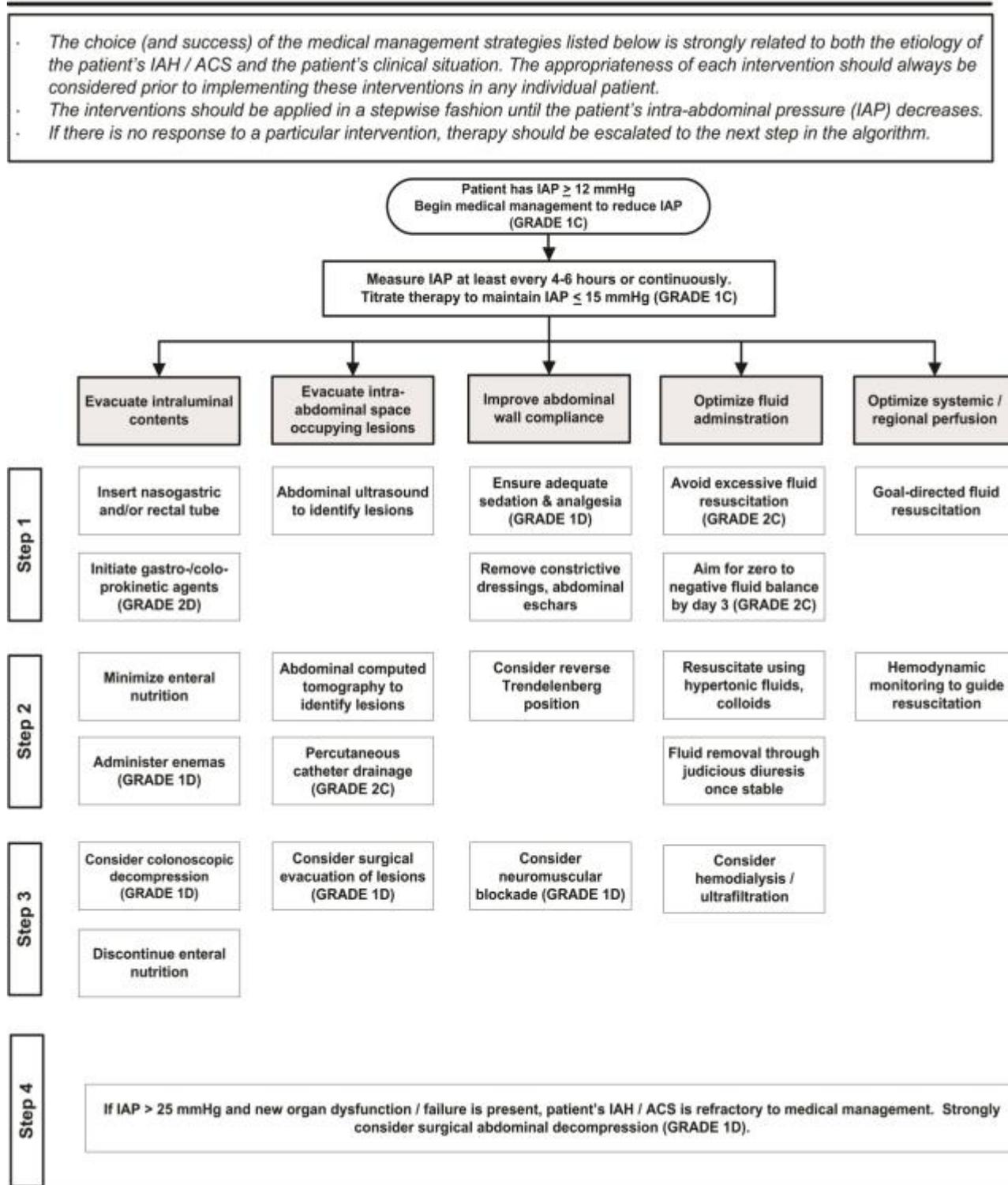


12. Once the measurement has been obtained, remove the needle from the sampling port and unclamp the drainage system.
13. Record the intra-abdominal pressure on MetaVision and remember to subtract the 25ml of saline from the hourly urine output.
14. Report intra-abdominal pressure to medical staff. Suggested management of the patient is outlined in the IAH/ACS management algorithm.

Recommendation (Action)	Justification (Rationale)
<b>Inform senior medical team on critical care unit if IAH is identified, and consider referral to surgical team.</b>	Although flowcharts from WSACS are included in this guideline, the treating critical care doctor must decide the correct management strategy, in consultation with the critical care consultant +/- surgical team.
<b>Take action to improve organ dysfunction by reducing IAP where possible.</b>	An evidence-based scheme is suggested by WSACS, as below.

*Suggested* Management Flowchart #2,  
taken from WSACS, the Abdominal Compartment Society (2013)

## IAH / ACS MEDICAL MANAGEMENT ALGORITHM



### 3. GLOSSARY

### 4. REFERENCES

1. Hunt, L., et al. 2012. A comparison of fluid instillation volumes to assess intra-abdominal pressure using Kron's methods. *Journal of Trauma & Acute Care Surgery* 73 (1): 152-155.
2. Kirkpatrick, A.W., et al. 2013. Intra-abdominal hypertension and the abdominal compartment syndrome: Updated consensus definitions and clinical practice guidelines from the world society of the abdominal compartment syndrome. *Intensive Care Medicine* 39 (7): 1190-1206.
3. Lee, R.K. 2012. Intra-abdominal hypertension and abdominal compartment syndrome a comprehensive overview. *Critical Care Nurse* 32 (1): 19-32.
4. Malbrain, M.L., et al. 2013. Intra-abdominal hypertension: Definitions, monitoring, interpretation and management. *Best Practice & Research Clinical Anaesthesiology* 27 (2): 249-270.
5. Malbrain, M.L. and J.DeWaele. 2013. *Intra-abdominal hypertension core critical care*. Cambridge: Cambridge University Press
6. Min Yi., et al. 2012. The evaluation of the effect of body positioning on intra-abdominal pressure measurement and the effect of intra-abdominal pressure at different body positioning on organ function and prognosis in critically ill patients. *Journal of Critical Care* 27 (2): 222.e1-6.
7. Kirkpatrick AW, et al (2013) Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. *Intensive Care Med* (2013) 39:1190–1206

### 5. ONLINE RESOURCES

[WSACS Consensus Guidelines Summary | WSACS](#)

The use of this guideline is subject to professional judgement and accountability. This guideline has been prepared carefully and in good faith for use within the Department of Critical Care at Brighton and Sussex University Hospitals. The decision to implement this guideline is at the discretion of the on-call critical care consultant in conjunction with appropriate critical care medical / nursing staff.