Care of Complex Surgical Patients Clinical Guidelines



Royal College of Surgeons in Ireland November 2005



**RCSI Clinical Guidelines Committee** Mr Ronan Cahill, Prof Arnold Hill, Prof H Paul Redmond.

Care of Complex Surgical Patients Clinical Guideline.



## Foreword

## **Prof HP Redmond**

The Clinical Guidelines Committee is pleased to present Guidelines on the Care of Complex Surgical Patients, prepared by an ad hoc working party of the RCSI Clinical Guidelines Committee. The production of these guidelines has arisen out of the perceived requirement for them by the Irish Higher Surgical Training Group.

As is always the case with clinical guidelines it should be emphasised that they are not strict protocols and at all times leave scope for independent decision making by the clinician. Furthermore, it is the intention of the Committee to review and renew these guidelines as new evidence in the topics chosen becomes available.

These guidelines represent a new departure for the Clinical Guidelines Committee. They are broad-based in their coverage of a variety of important topics and have been authored by a large cohort of our Irish Higher Surgical Trainees, all of whom I would like to commend for the huge effort they have put in to preparing these guidelines in a timely fashion. A special word of thanks goes to Mr Paul Balfe, Mr Joe Dowdall, Mr Brian Manning, Mr Paul Ridgeway, and Mr Conor Shields, who in addition to contributing chapters also acted in an editorial advisory capacity. As always the working party acknowledges with gratitude the considerable efforts and contributions made to this document by Ms Grainne Donovan, RCSI. A very special note of gratitude must go to Mr Ronan Cahill for his remarkable vision, enthusiasm and leadership in driving forward this initiative. Finally I hope very much that these pocket size guidelines will be of practical value to trainees and consultants alike.

Professor H Paul Redmond Chairman Clinical Guidelines Committee November 2005



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## Acknowledgements

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Ms Grainne Donovan, R.C.S.I.

In addition to contributing chapters, the following also acted in an editorial advisory capacity:

Mr Paul Balfe, Mr Joseph Dowdall, Mr Brian Manning, Mr Paul Ridgway, Mr Conor Shields.

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## Introductior

## **RCSI Guidelines Committee**

As a first impression, some may question the credentials of non-consultants as primary authors of RCSI Guidelines on the "Care of Complex Surgical Patients". Indeed, on a broader issue, there may be misgivings on the usefulness of creating guidelines in the first place. The address of both issues lies in the consideration of the current role and function within Irish medicine of both Clinical Guidelines and the Senior Specialist Registrars (SSpRs) in surgery.

While most professional societies now recognise the value of guidelines, they also realise that they cannot be based on research evidence alone. Often, however, their systems of production are criticised as lacking both transparency and reliability and because they fail to take into account the level of available resources in individual institutions. For these reasons, their critics argue, guidelines are often unrepresentative and so will never achieve comprehensive and timely coverage of the health issue they intend to address.<sup>1</sup> SSpRs in surgery represent a group of senior clinical decision makers whose job is continuously located at all direct points of delivery of patient care throughout a hospital. Furthermore, they have gained their clinical training and experience in a variety of units throughout the country. They therefore possess first hand working knowledge of the facilities available and are all too aware of the shortcomings of care inherent to the Irish hospital system. Finally, individual SSpRs are both exposed to, and actively involved in, the academic endeavour and clinical practice of a range of surgical sub-specialities at the same time as they are developing a focussed sub-specialist interest. Their invited submission of topics covering frequently encountered, important surgical issues is therefore to be welcomed with interest.

What we have not done in this format is to try and formally develop consensus opinion about management issues in a specifically Irish context. Instead, trusting in their training and mentored – professional development as a surrogate, we have allowed the authors themselves to make value judgements on the applicability of the evidence base available to their topics in order to develop reasoned and rational approaches to a variety of complex surgical conditions. Any unit or indeed single practitioner may have opinions that differ to that of the authors and these guidelines are not intended to

<sup>&</sup>lt;sup>1</sup> Raine R+: Developing clinical guidelines: a challenge to current methods. *BMJ* 2005; 331: 631-3.



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replace or override working protocols in any individual department. Indeed, the Guidelines Committee would welcome the submission of alternative approaches to management. An open discussion can only improve the document for its second edition and may indeed ultimately deliver widespread consensus on these important issues.

RCSI Clinical Guidelines Committee November 2005



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## Eoghan Condon

## INTRODUCTION

"Hypovolaemic shock" refers to the clinical condition that manifests when intravascular volume depletion has resulted in impaired organ function due to inadequate perfusion. The two most common causes of this in the nontrauma setting are gastrointestinal (GI) haemorrhage and ruptured abdominal aortic aneurysm (rAAA). The most important points in the approach to the hypovolaemic patient involve an emphasis on a definite strategy to make a diagnosis and achieve haemorrhage control, awareness of the mean arterial pressure (MAP) as indicator of perfusion and the judicious rather than traditional over-zealous use of fluids where hypotension occurs.

## **INITIAL ASSESSMENT – "ABC"**

*Airway* – Assessment is important and intubation in order to protect the airway may be required in the presence of persistent haemataemesis (e.g. secondary to oesophageal varices) due to a high associated risk of aspiration.

*Breathing* – The chest should be examined and oxygen administered to ensure oxygen saturation levels >90%.

*Circulation* – Blood pressure and pulse are assessed. Two large bore (16 G at least) intravenous cannulae should be inserted into wide calibre veins (e.g. antecubital fossa). If this is not possible, femoral or central venous access should be established. The rate of fluid resuscitation varies as per the clinical need (higher infusion rates require a pressure bag be applied to the infusate).

The normal pattern of clinical response to intravascular compromise is that tachycardia precedes hypotension (the initial chronotropic response of the heart can maintain blood pressure in face of the loss of approximately 30% of the circulating blood volume). Reduced urinary output (below 0.5 ml/kg/hr) and evidence of confusion or agitation indicates malperfusion of critical organs and occurs as a result of reduced MAP (i.e. intravascular volume loss in excess of 30%). Coma may supervene when blood loss exceeds 40% of baseline and so the window of therapeutic opportunity between these two stages is narrow. Early transfusion of packed red cell concentrate (RCC) should be considered in any patient with hypotension and oliguria (see Table 1).



After initial resuscitative efforts, patients often separate into responders, transient responders, or non-responders with respect to the initial bolus of fluid given. Based on these observations a decision can be made re immediate surgical/endoscopic intervention or observation/radiological investigation in the first instance.

- A. Responders have had a significant haemorrhage but are no longer bleeding. Their vital signs have normalised. Investigations may proceed non-urgently.
- B. Transient responders are continuing to haemorrhage. The vital signs were restored initially but then tachycardia +/ hypotension re-occurs. These patients require urgent intervention to stop the bleeding rather then continuing resuscitation alone. Indeed, it may be detrimental to these patients' outcome to administer further large boluses of fluid in an effort to normalise vital signs. It is advisable instead to maintain MAP between 80-90 mmHg with either blood or crystalloid fluid at a rate of 125 200mls per hour titrated against clinical response.
- C. Non-responders have had massive haemorrhage or are suffering continuing bleeding. They require immediate intervention. Fluids should be delivered in boluses of 25mls/min to achieve a MAP of 80-90mmHg while transfer to theatre is arranged. Cross matched blood is the fluid of choice. No delay for solely diagnostic procedures is acceptable.

Although early animal models of shock showed a benefit with aggressive volume replacement, these studies were based on "fixed volume deficits" (that is the animal underwent phlebotomy to reduce circulating blood volume but were at no risk of subsequent bleeding). Later work examining more clinically relevant models showed that "low-dose" fluid resuscitation was preferable. This method of resuscitation is referred to as permissive hypotension and has found resonance clinically in the preoperative resuscitation of patients with rAAA and severe trauma. It prevents the so called "pop the clot" phenomenon, where attempts to restore BP without intervening to achieve haemostasis actually accentuates haemorrhage.<sup>1</sup>

<sup>&</sup>lt;sup>1</sup> Dutton RP+: Hypotensive resuscitation during active haemorrhage: impact on in-hospital mortality. *J Trauma* 2002; 52: 1141-6.



While these initial resuscitative measures are ongoing, a patient or relevant collateral history can be obtained to aid in making the diagnosis. It is critical to elicit whether the following symptoms are present:

- 1. Bleeding haemataemesis or melaena (likely upper GI cause) or haematochezia (likely lower GI or brisk upper GI aetiology) or vaginal (ectopic pregnancy).
- 2. Abdominal pain
  - I. radiates to back (rAAA, perforated or bleeding DU).
  - II. diffuse abdominal pain (peritonitis from a perforated abdominal viscus, pancreatitis).
- 3. Chest pain pneumothorax, myocardial infarction, dissection of thoracic aortic aneurysm.
- 4. AMPLE history
  - a. A Allergies.
  - b. M Medications: NSAIDs (DU pathology), anticoagulants, steroids, immunosuppressants.
  - c. P Past medical history, previous AAA repair (aortoduodenal fistula), alcohol abuse, cirrhosis (oesophageal varices).
    - Pregnant?
  - d. L Last meal?
  - e. E Elicit a history from ambulance crew or relatives.

Abdominal examination is performed to elicit the presence of rebound tenderness or guarding (peritonitis, pancreatitis), an abdominal mass (rAAA) or distension. A rectal examination should also be performed in every patient to examine for the presence of fresh or altered blood.



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**N.B.** Other conditions such as visceral perforation (oesophageal/peptic ulcer/colonic), pancreatitis, septic shock or myocardial infarct may present similarly to haemorrhagic shock and must also be borne in mind as differential diagnoses especially in the absence of convincing evidence of bleeding. Beware of placing too much emphasis on a vague collateral history or reports of "coffee ground" vomiting.

## Other measures

Phlebotomy should be performed and the samples sent urgently for:

A *full blood count* to determine Hb (although this may be unreliable early after the bleeding commences as the drop in Hb concentration is evidenced only after haemodilution secondary to replenishment of plasma volume occurs) and platelet count (a low platelet count suggests thrombocytopaenia or hypersplenism secondary to portal hypertension and cirrhosis). Low MCV and MCH levels consistent with iron deficiency anaemia may indicate a chronic element to the blood loss.

A *coagulation screen* to determine liver synthetic function as vitamin K and/or clotting factors (e.g. octoplas/fresh frozen plasma) may be necessary to normalize a bleeding tendency in certain patients (e.g. those with significant alcohol intake or known cirrhosis).

*Urea and electrolytes* – to establish baseline renal function. Urea may be elevated in patients with upper GI bleeding due to digestion of the "meal" of blood in the gut.

*Liver function tests* – to determine baseline liver function.

CPK/troponin - if any suggestion or risk of cardiac dysfunction.

Pregnancy test (in females).

*Blood cross-match sample* – in general patients with significant bleeding potential should have 4 units of type specific blood on stand by at all times. While there is some controversy over the appropriate level to transfuse at, in general, the Hb should be kept above 8 g/dl in most patients and over 10 g/dl in those with significant cardiorespiratory impairment. Pooled platelets, fresh frozen plasma and/or factor concentrates may be given as per clinical need. Haematology input should be sought early in this regard. In general, platelet



counts should be maintained above  $50 \times 10^{9}$ /L (each pool increases the platelet count by approximately  $8 \times 10^{9}$ /L) and the INR <1.2.

It is essential that the lab and portering staff are informed directly about the urgency of the situation.

A **urinary catheter** (16-18 g Foley catheter) is inserted if there is a necessity to assess urine output on an hourly basis as a measure of the adequacy of the resuscitative process. In less urgent settings, allowing micturation into a graduated container can provide the same information without the need for urethral instrumentation. In the absence of any draining urine, check that the balloon is indeed in the bladder as it is unusual for shocked patients to be completely anuric with no residual urine.

A **nasogastric tube** should be placed and aspirated. Aspirated blood may be diagnostic for a bleeding peptic ulcer although the absence of fresh blood from the nasogastric tube by no means excludes this (c. 10% of patients with bleeding duodenal ulcers may not show blood on gastric aspiration). Guiac testing (*Faecal Occult Blood* testing) of the aspirate is of no clinical utility and indeed is often misleading – it should *not* be performed.

An **electrocardiogram** should also be performed as cardiac ischaemia may complicate hypovolaemia.



## SECONDARY ASSESSMENT

- Frequent repeat clinical assessment (BP, pulse rate, O<sub>2</sub> saturations) is a crucial aspect of the care and resuscitation of (potentially) unstable patients as clinical deterioration may occur precipitously and mandate urgent intervention as a life-saving measure.
- Fluid prescription of small aliquots administered regularly with close monitoring of response being used to guide further prescription is preferable to "guestimating" total requirements ahead of time.
- While urine output is a useful measure of renal perfusion outside of a high dependency setting, changes in values tend to lag behind actual intravascular levels. Therefore, accurate pulse and blood pressure measurement along with, where necessary, central venous pressure assessment is a better means of assessing short-term response to treatment.

All hypovolaemic patients being initially managed conservatively should have a definite plan in place to make a definitive diagnosis at the earliest opportunity as well a "Plan B" in case of sudden deterioration in clinical status. This may include a decision not to pursue invasive intervention (e.g. surgery or intubation) in cases deemed unsalvageable – however, it is essential that such a strategy is deliberately decided upon by a senior decision maker early in the care of the patient with full involvement of the next of kin where appropriate.

	Class 1	Class 2	Class 3	Class 4
Blood Loss (mL)	Up to 750	750-1500	1500-2000	>2000
% loss of blood volume	Up to 15%	15-30%	30-40%	>40%
Pulse Rate (bpm)	<100	>100 >120 >140		>140
Blood Pressure	Normal	Normal	Decreased	Decreased
Respiratory Rate	Normal or Increased	Decreased	Decreased	Decreased
Urine Output (mL/h)	>35	30-40	20-30	14-20
CNS/MentalSlightlyMildlyStatusanxiousanxious		Mildly anxious	Anxious, confused	Confused, lethargic
Fluid Replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

#### Table 1: Clinical correlates by the degree of fluid and blood losses<sup>2</sup>

Note: The patient may have normal vital signs even with a 15% loss of blood volume (Class I shock). Oliguria, as a sign of reduced renal perfusion pressure, may not occur until class 3 haemorrhage has developed.

<sup>&</sup>lt;sup>2</sup> American College of Surgeons Committee on Trauma: Advanced Trauma Life Support Course Manual. Chicago, Ill: American College of Surgeons; 1997.



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# 2. Upper Gastrointestinal Haemorrhage

## **Paul Balfe**

## INTRODUCTION

Patients with upper gastrointestinal (GI) haemorrhage present with haematemesis, malaena or both. The causes by site and frequency (where definitely established) are set out in Table 1. However, the source of bleeding may prove elusive in up to 15% of cases despite experienced upper GI endoscopy. In the elderly population, the widespread use of aspirin for cardiovascular prophylaxis/treatment and NSAIDS for arthritic conditions accounts for a significant proportion of presentations in this demographic. In many cases (and in up to 50% of patients with variceal bleeding), the initial haemorrhage stops spontaneously at least temporarily. It is essential however to proceed with diagnostic and therapeutic procedures at the earliest opportunity.

The management of acute upper GI haemorrhage can be divided into two phases:

Phase 1 Resuscitation and control of haemorrhage.

Phase 2 Post stabilisation management of the pre-disposing condition.

- e.g. Varices modification of lifestyle risk factors,  $\beta$ -blockers etc.
- e.g. PUD modification of lifestyle risk factors, eradication of H. Pylori etc.

The emphasis here is mainly on Phase 1. There exists an abundance of literature on the issue of acute upper GI haemorrhage. Drawing on this, Algorithms 1 and 2 have been developed which represent sensible, well practiced workable approaches to this very common clinical presentation. The algorithms are accompanied by keynotes that explain or expand on specific areas. In terms of definitive haemorrhage control, oesophago-gastro-duodenoscopy (OGD) represents the first-line diagnostic and therapeutic intervention.

## Table 1: Causes of upper gastrointestinal bleeding

Organ	Cause	% Frequency
	Oesophagitis	12
Ossenhagus	Varices	5
Oesophagus	Mallory-Weiss Tear	5
	Cancer	2
	PUD	22
Stomach	Gastritis	7
	Cancer	2
Duodenum	PUD	29



Upper Gastrointestinal Haemorrhage

#### Algorithm 1: Initial Management





#### Keynotes accompanying Algorithm 1

- 1. Full clinical examination may reveal stigmata of liver disease.
- 2. Age older than 60 years is an independent marker for a poor outcome. The mortality rate increases to 12-25% in those older than 60 years compared to a mortality rate of less than 10% for patients younger than 60 years. The American Society for Gastrointestinal Endoscopy (ASGE) has found a mortality rate of 3.3% for patients aged 21-31 years, a rate of 10.1% for those aged 41-50 years, and a rate of 14.4% for those aged 71-80 years.<sup>1</sup> Only relatively few deaths are however due to actual exsanguination.
- Aggressive fluid resuscitation should be avoided especially in cases of suspected varices and a "low normal" blood pressure accepted.
- 4. In the initial, active phase of variceal haemorrhage, it has been shown that the immediate institution of antibiotic prophylaxis has a significant beneficial impact on short-term survival. The current UK guidelines recommend the use of ciprofloxacin at a dose of 1g/day.
- 5. For known variceal bleeders, Terlipressin or Octreotide infusions are preferred over vasopressin as the latter has a less selective effect in terms of splanchnic vasoconstriction and may lead to cardiac angina and arrythmias.
- 6. The nature and volume of blood products ordered in the first instance will depend on the degree of haemorrhage and the suspected pathology e.g. FFP/octoplas may be required in those with impaired hepatic function.
- 7. The requirement for packed red cell transfusion correlates with both the need for surgery and mortality<sup>2</sup> (*see Table 2*).
- 8. The endoscopist performing urgent intervention on unstable bleeders should have provision for immediately proceeding to surgery if attempts at endoscopic control are unsuccessful.
- 9. Rebleeding (a marker for increased mortality and need for surgery) occurs in 2% of patients presenting without shock, in 18% with tachycardia alone and in 48% with shock. Mortality rates correlate with the systolic blood pressure (SBP) at presentation: 8% when SBP>100 mm Hg, 17% when SBP 80-90 mm Hg, &>30% when SBP<80 mm Hg.</p>

<sup>&</sup>lt;sup>1</sup> Peter DJ+: Evaluation of the patient with GI bleeding: An evidence based approach. *Emerg Med Clin North Am* 1999; 17: 239-61.

<sup>&</sup>lt;sup>2</sup> Larson E+: Upper gastrointestinal bleeding: prediction of outcome. *Surgery* 1986; 100: 765-73.



## Table 2: Correlation of RCC requirement with subsequent surgery and mortality

Number of Units Transfused	Need for Surgery (%)	Mortality Rate (%)
0	4	4
1-3	6	14
4-5	17	28
>5	57	43



#### Upper Gastrointestinal Haemorrhage

#### Acid suppression in acute G.I. bleeding

Recent meta-analyses have found proton pump inhibitors (PPI) to be more effective then H2-receptor antagonists in preventing persistent or recurrent bleeding.

High-dose PPI therapy by i.v. bolus followed by continuous infusion decreases rebleeding in patients who have undergone successful endoscopic therapy and should be used to treat patients with high-risk endoscopic stigmata.

Empiric therapy with high dose PPI should be considered for patients awaiting endoscopy but is not a replacement for urgent endoscopy and haemostasis.

Barkun A+: Non-variceal upper GI bleeding consensus conference group. Ann Intern Med 2003; 139: 843-57.



## Algorithm 2a: Identification and Control of Haemorrhage





#### **Upper Gastrointestinal Haemorrhage**

#### Keynotes accompanying Algorithm 2a

- The evidence suggests a class effect for PPI treatment and that improvement in rebleeding rates can be achieved using omeprazole at a dose of 80 mg iv stat followed by infusion (8 mg/hr iv) for 72 hours after endoscopic therapy. Patients may be switched to oral PPI after this time or on resumption of oral intake.
- 'Others' includes cancer, oesophagitis/gastritis etc. In the case of malignancy, the tumour may have ulcerated or may have eroded into a vessel. The level of therapeutic intervention in terms of haemorrhage control will depend on the extent of tumour, patient fitness etc. PPIs +/ – H. pylori eradication represent the mainstay of treatment of haemorrhagic oesophagitis/gastritis.
- 3. Rebleeding occurs in 55% of patients who have active bleeding (pulsatile or oozing), in 43% who have a nonbleeding visible vessel, in 22% who have an ulcer with an adherent clot, and in 0-5% who have an ulcer with a clean base. Recurrent bleeding usually occurs within 72 hours.
- 4. A wide array of methods are employed to achieve endoscopic haemostasis. These fall into four main categories:

Injection:	Adrenaline Alcohol Sclerosant Clotting factors	Topical:	Collagen Clotting factors Cyanoacrylate glue
Thermal:	Laser photocoagulation Diathermy Heater probe Microwave	Mechanical:	Balloon tamponade Endoclips Staples Sutures

No one method or combination of methods has emerged as the ideal. Taking into account the available evidence, the recognition of the wide divergence in availability of resources and the over-riding imperative for practicality in the acute situation, adrenaline injection 1:10 000 injected in 0.5-1 ml aliquots around the base of the vessel is recommended.

- 5. Biopsy of acute gastric ulcers is not indicated in the acute phase, but should be performed at an interval of approximately four weeks in order to determine whether the ulcer is malignant. Duodenal ulcers do not generally require a similar assessment.
- 6. A 'second look' OGD is advisable about five days after the initial therapeutic one. This is particularly important in cases involving a visible vessel in an ulcer bed as there is a higher risk of rebleeding with massive haemorrhage post-discharge more likely to prove fatal.
- 7. Significant rebleeding rates of 10% (80% mortality for rebleeders) can occur in patients who undergo laparotomy and undersewing of the ulcer base and so proton pump inhibition should be continued postoperatively.



## Table 3: Upper GI Bleeding Score to assess probability of need for intervention<sup>3</sup>

All variables are quantifiable before endoscopy is performed.

Variable		Measurement	Score
	18.2 to 22.4 mg/dl	2	
Pland Lines Nitrogen	22.4 to 28 mg/dl	3	
Blood Urea Nitrogen		28 to 70 mg/dl	4
		>70 mg/dl	6
		12 to 13 g/dl	1
	Males	10 to 12 g/dl	3
Haemoglobin		<10 g/dl	6
Females		10 to 12 g/dl	1
		<10 g/dl	6
		100 to 109 mmHg	1
Systolic blood pressure		90 to 99 mmHg	2
		<90 mmHg	3
	Pulse >100 per minute	1	
	Presentation with melaena	2	
Miscellaneous Markers		Presentation with syncope	2
		Hepatic disease	2
		Cardiac dysfunction	2

Score  $\leq$ 4 predicts resolution without intervention Score  $\geq$ 5 predicts need for intervention

<sup>&</sup>lt;sup>3</sup> Blatchford 0+: A risk score to predict the need for intervention in upper gastrointestinal haemorrhage. Lancet 2000; 356:1318-21.



# Table 4: Rockall scoring system for risk of rebleeding and death after admission to hospital for acute upper gastrointestinal bleeding

This system is better at predicting death than rebleeding and has been validated as being accurate in both peptic ulcer and variceal bleeding subgroups. A pre-endoscopy score of o-3 is associated with a mortality of 3.2% while a score of 4-7 indicates a risk of 22.4%. A completed postendoscopy score of less the three is associated with an excellent prognosis while a score greater then eight is associated with a high risk of mortality. Those scoring <2 have a rebleed rate of 5% and total mortality rate of 0.1% and so may be appropriately managed as outpatients.<sup>4</sup>

Variable	Score				
	0	1	2	3	
Age (yrs)	<60	60-79	>80		
Shock	No shock (SBP >100, pulse <100)	Tachycardia (pulse >100, SBP >100)	Hypotension (SBP <100, pulse >100)		
Co-morbidity	Nil major		Cardiac failure/ Ischaemic heart disease	Renal failure/ liver failure/ disseminated malignancy	
Diagnosis	Mallory Weiss tear/no lesion and no stigmata of recent haemorrhage	All other diagnosis			
Stigmata of recent haemorrhage	None or dark spot		Blood in upper GI tract/Adherent clot/Visible or spurting vessel		

<sup>&</sup>lt;sup>4</sup> Rockall TA+: Variation in outcome after acute upper gastrointestinal haemorrhage. National Audit of acute upper gastrointestinal haemorrhage. *Lancet* 1995; 346: 346-50.



## Algorithm 2b: Identification and Control of Haemorrhage





#### Keynotes accompanying Algorithm 2b

- Approximately one-third of patients with varices will develop haemorrhage. A patient known to have varices who presents with GI haemorrhage is as likely to be suffering from peptic ulcer bleeding as variceal bleeding.
- 2. Each episode of variceal haemorrhage is associated with a 20-30% risk of death.
- After an acute variceal haemorrhage, bleeding resolves spontaneously in c. 50% of patients. Bleeding is least likely to stop in patients with large varices and a Child-Pugh class C cirrhotic liver.
- 4. Once the bleeding stops, patients remain at increased risk of rebleeding for about 6 weeks although the risk is greatest during the first 48 hours after the initial haemorrhage.
- 5. Avoid over-resuscitation in patients with variceal bleeding as rebound portal hypertension can lead to early rebleeding.
- 6. Clotting factors often need to be administered to normalise the INR. Platelet transfusions are reserved for those with counts below 50x10<sup>9</sup>/L in an actively bleeding patient.
- 7. Terlipressin (2mg stat then 1-2 mg every 4-6 hours by slow i.v. bolus x 72 hrs) has been associated with a 34% relative risk reduction in mortality.<sup>5</sup> The dose may be tapered prior to discontinuation. An alternative agent is octreotide infusion: 25-50 μg/hr for 2-5 days.
- While banding is favoured over injection in the acute setting, it may not always be available or the operator may not be sufficiently experienced in its use. Sclerotherapy should then be utilised.
- 9. Patients with valvular heart disease and those with ascites should receive antibiotics at the time of endoscopic sclerotherapy to prevent bacterial endocarditis and spontaneous bacterial peritonitis, respectively.
- 10. If bleeding persists (or recurs within 48 hours of the initial episode) despite pharmacologic therapy and two endoscopic therapeutic attempts at least 24 hours apart, patients should be considered for surgical salvage. When bleeding occurs more than 48 hours after the initial episode, endoscopic therapy should be attempted again.
- 11. The two most commonly used tubes for balloon tamponade are the Sengstaken-Blakemore tube and the Minnesota tube both of which have oesophageal and gastric balloons that are inflated to produce a compressive effect on variceal flow after insertion (the latter also has a internal oesophageal aspiration port which obviates the need for an additional nasogastric tube). If inserting, consider endotracheal intubation to protect the patient's airway. Although tamponade effectively controls acute variceal haemorrhage, a meta-analysis of studies comparing it to drug therapy and endoscopic sclerotherapy revealed no overall survival advantage in the balloon group.<sup>6</sup> It is therefore a temporising measure and so requires a definitive plan of care/transfer before its removal.

<sup>&</sup>lt;sup>5</sup> Ioannou G+: Terlipressin for acute upper oesophageal haemorrhage. Cochrane Database Syst Rev 2003 (1). CD002147.

<sup>&</sup>lt;sup>6</sup> Kupfer Y+: Acute GI bleeding in the intensive care unit. The intensivist's perspective. *Gastroenterol Clin North Am* 2000; 29: 275-307.



Upper Gastrointestinal Haemorrhage

## Table 5: Endoscopic classification of varices

<b>Oesophageal –</b> classified by size (the greater the size the more likely they are to bleed)	Gastric – classified by location
Grade 1 – Small, straight	In continuity with oesophageal varices: either along lesser or greater curves extending toward the fundus
Grade 2 – Enlarged, tortuous; occupy <1/3 of lumen	Isolated either to the fundus or elsewhere in the stomach
Grade 3 – Large, coil-shaped; occupy >1/3 of lumen	

## Table 6: Factors associated with risk of bleeding from varices

Varix size and location
Large oesophageal varices
Isolated cluster of varices in fundus of stomach
Variceal appearance on endoscopy ("red signs")
Red wale marks (longitudinal red streaks on varices)
Cherry-red spots (red, discrete, flat spots on varices)
Haematocystic spots (red, discrete, raised spots)
Diffuse erythema
Child-Pugh class C cirrhosis
Presence of tense ascites



## Table 7: Factors associated with risk of variceal rebleeding

Early	Late
Age over 60 years	Severe liver failure
Renal failure	Continued alcohol abuse
Severe initial bleeding (i.e. Hb<8 g/dL at presentation)	Renal failure
	Large variceal size
	Hepatocellular carcinoma

### Table 8: Balloon tamponade (Sengstaken-Blakemore or Minnesota Tube)

The tip of the tube is first introduced through the nose into the stomach.

The gastric balloon is inflated with 300-350 mL of air and is pulled up into the gastric fundus compressing the gastro-oesophageal junction. The tube is left hang over the headstand of the bed using a 1L bag of fluid as traction.

The oesophageal balloon is then inflated to a pressure of 40 mm Hg. This balloon should be deflated every 4 hours for 15 minutes to avoid oesophageal pressure necrosis. The entire tube should not be in place for more than 48 hrs.

I	Major complication	Minor complications		
Oesophageal necrosis/ulcer	Oesophageal rupture	Duodenal rupture	Chest pain	Alar necrosis
Airway obstruction	Aspiration	Haemoptysis	Balloon impaction and/ or migration	Naso- pharyngeal bleeding
Tracheo- oesophageal fistula	Tracheal rupture	Thoracic lymph duct obstruction	(nausea & vomiting)	



Care of Complex Surgical Patients Clinical Guidelines

# 3. After-crisis Management of Portal Hypertension

## **Noel O'Brien**

## INTRODUCTION

Approximately 70% of cirrhotic patients surviving an episode of acute variceal bleeding will die within one year if untreated.<sup>1</sup> It is thus clear that the post-haemorrhage management of such patients is as critical as their early management. Abstinence from alcohol should be encouraged in all patients with alcoholic cirrhosis. Ultimately, the optimal treatment for many patients in the long term may be liver transplantation and patients who survive a variceal bleed should be considered for this.

## REBLEEDING

Various predictors of the likelihood of re-bleeding have been studied. The most powerful predictor is the actual size of the varices seen at endoscopy, with re-bleeding being more likely with larger varices. Other less significant predictors included the presence of red weal marks on the varices and the severity of liver dysfunction as expressed by the Child-Pugh classification (*see Table 1*). The endoscopic predictors are felt to directly reflect increased Hepatic Venous Pressure Gradient (HVPG).

Rubber band ligation and injection sclerotherapy of varices are generally found to be equivalent in terms of obliterating varices and improving overall survival, though a recent meta-analysis suggested a greater benefit with band ligation. Furthermore, there is a significantly higher incidence of complications such as oesophageal stricture or perforation with injection sclerotherapy. The optimal interval between banding sessions is not clear but most published guidelines recommend a two to three week interval with treatments being continued until the varices have been obliterated. Repeat endoscopy at three and six months is recommended to confirm their resolution with annual surveillance thereafter.<sup>2</sup>

<sup>&</sup>lt;sup>1</sup> Graham, DY+: The course of patients after variceal haemorrhage. *Gastroenterology* 1981; 80: 800-809.

<sup>&</sup>lt;sup>2</sup> Jalan R+: UK guidelines on the management of variceal haemorrhage in cirrhotic patients. Gut 2000; 46 Suppl 3-4:III1-III15.

## MANAGEMENT OF PORTAL HYPERTENSION

It has been shown that treatments that maintain HVPG at less than 12mmHg effectively prevent recurrent variceal bleeding (normal HVPG is <5 mmHg).<sup>3</sup> Measurement of HVPG is however an invasive procedure and not readily available at present, so endoscopic surveillance of varices remains the primary means of monitoring progress. Medical therapies for the prevention of recurrent variceal bleeding include the use of B-blockers, nitrates and prazosin. Propanolol has been shown to significantly reduce the consequences of rebleeding in most studies (risk of rebleeding is reduced by approximately 45% while the risk of bleeding-related mortality is reduced by 50%). However, only one study has demonstrated a reduction in overall mortality.<sup>4</sup> These benefits occur when this agent is used at a dose sufficient to induce a 20% drop in resting heart rate or a rate of less than 55 bpm. At present the evidence for the use of nitrates and other agents is insufficient to recommend their routine use although nitrates such as isosorbide mononitrate may be justified in those who have a contra-indication to  $\beta$ -blockers (e.g. in those with heart block or asthma).

For patients who fail to respond to the above measures and have recurrent variceal bleeding, the options lie between Transjugular Intrahepatic Portosystemic Shunts (TIPS) placed percutaeneously by a radiologist, or a surgically created portosystemic shunt. Advantages of TIPS are that it is less invasive than a surgical procedure and is associated with control of variceal bleeding in up to 95% of patients with active variceal bleeding. However a meta-analysis concluded that, compared with endoscopic therapy, TIPS reduced the rate of re-bleeding but did not improve mortality figures and was associated with an increased rate of hepatic encephalopathy.<sup>5</sup> Most of the failures of TIPS treatment are associated with failure of the shunt due to thrombosis, kinking, retraction or stenosis of the stent. Thus, the use of TIPS should probably be reserved for patients with recurrent variceal bleeding that does not respond to endoscopic and medical therapy.

<sup>&</sup>lt;sup>3</sup> Bosch J+: Current management of portal hypertension. J Hepatol 2003; 38 suppl 1: 554-68.

<sup>&</sup>lt;sup>4</sup> Hayes PC+: Meta-analysis of value of propanolol in prevention of variceal haemorrhage. *Lancet* 1990; 336: 153-6.

<sup>&</sup>lt;sup>5</sup> Papatheodoridis GV+: Transjugular intrahepatic portosystemic shunt compared with endoscopic treatment for prevention of variceal rebleeding: a meta-analysis. *Hepatology* 1999; 30: 612-22.



Care of Complex Surgical Patients Clinical Guidelines After-crisis Management of Portal Hypertension

Surgical portosystemic shunts can be selective such as distal splenorenal shunts or non-selective such as portocaval shunts which decompress the entire portal system. Promising results have been found with the use of partial non-selective shunts using a small diameter (8-10mm) H-graft between the portal vein and the inferior vena cava.<sup>6</sup> These shunts are associated with less post-shunt encephalopathy and achieve control of rebleeding in 97% of patients. However, such results were only shown in a single study to date and need to be corroborated in larger trials prior to recommending this procedure.

In summary, it appears that the optimal follow-up therapy is a combination of band ligation and  $\beta$ -blockers as described above with TIPS and surgical shunt formation being reserved as "salvage therapy" for those who fail to respond.

#### Primary prophylaxis of variceal rebleeding:

Propanolol: commence at 40 mg bd (some patients e.g. the elderly may be started on 20 mg bd). Titrate to give 20% reduction in baseline pulse rate. Monitor for hypotension.

<sup>&</sup>lt;sup>5</sup> Collins JC+: Narrow-diameter portacaval shunts for management of variceal bleeding. World J Surgery 1994; 18: 211-5.



#### After-crisis Management of Portal Hypertension

Score Allotted	1	2	3
Bilirubin (µmol/l)	<34	34-50	>50
Albumin (g/l)	>35	28-35	<28
PT (seconds prolonged) or INR	<4 <1.7	4-6 1.71-2.2	>6 >2.0
Presence of Encephalopathy	None	Mild	Marked
Presence of Ascites	None	Mild ( <i>controlled</i> )	Marked ( <i>refractory</i> )

#### Table 1: Child – Pugh classification of severity of liver cirrhosis<sup>7</sup>

The individual scores are summed and then grouped as: <7 = A; 7-9 = B; >9 = C.

- If there is primary biliary cirrhosis or sclerosing cholangitis then bilirubin is classified as <68=1; 68-170=2; >170=3.
- Class A cirrhosis is associated with a life expectancy of 15-20 years and a perioperative mortality risk for major surgery of c. 10%. Patients with Class B cirrhosis have a perioperative mortality risk of c. 30% while a C classification forecasts a survival of less than 12 months and confers a perioperative mortality risk of c. 80%.
- The risk of bleeding within 1 year is less than 10% for a patient with a Child-Pugh class A cirrhotic liver with small varices but is 76% for a patient with a class C cirrhotic liver with large varices and "red signs".
- The need for liver transplantation should be considered during the initial evaluation of patients with portal hypertension-related upper gastrointestinal bleeding. For patients with Child class A or B cirrhosis with preserved liver function, portal decompression is preferable to transplantation. For patients with Child class C cirrhosis, the TIPS procedure can be used as a temporizing measure to provide a bridge until assessment for transplantation can be arranged.

<sup>&</sup>lt;sup>7</sup> Pugh RN+: Transection of the oesophagus for bleeding oesophageal varices. *Br J Surg* 1973; 60: 649-9.



Care of Complex Surgical Patients Clinical Guidelines

## 4. Emergency Management of Colonic Haemorrhage

## Dara Kavanagh

## INTRODUCTION

Lower gastrointestinal (GI) tract bleeding accounts for 0.7% of acute surgical admissions with an overall mortality risk of 3%.<sup>1</sup> The majority present as a minor self-limiting event – less than 50% of patients presenting with haematochezia have had a significant bleed (i.e. one that causes cardiovascular compromise or a drop in haemoglobin) and only a minority of these suffer a massive life-threatening bleed.<sup>2</sup>

*Haematochezia* is the passage of red or maroon coloured stool per rectum usually (80%) due to colonic site of haemorrhage (20%= brisk upper GI bleed).

*Melaena* is black coloured stool that is also sticky and foul smelling. It is generally a manifestation of upper GI bleeding but can be secondary to colonic lesion with slow transit time.

The colon is the source of bleeding in 77% of cases with haematochezia while 15% originate in the foregut with 1% arising in the small bowel. No identifiable source is found in 7%.<sup>3</sup> Diverticulosis is now recognised as the primary aetiological factor (30%). Other potential aetiologies include: haemorrhoids (14%), ischaemic colitis (12%), rectal ulcers (9%), colitis (9%), postpolypectomy ulcers (8%), neoplasia (6%) and angiodysplasia (5%). Meckel's diverticulum, small bowel tumours and aortoenteric fistulae should also be considered. Diverticular haemorrhage settles spontaneously in 80% allowing semi-elective investigations. However, 20% subsequently rebleed. Once rebleeding has occurred and settled, the rebleeding rate thereafter is 50%. Although diverticular disease typically affects the left colon, acute colonic bleeding is more likely from right-sided diverticula.<sup>4</sup>

<sup>&</sup>lt;sup>1</sup> Vernava AM+: Lower gastrointestinal bleeding. *Dis Colon Rectum* 1997: 40: 846-58.

<sup>&</sup>lt;sup>2</sup> Bramley PN+: The role of an open-access bleeding unit in the management of colonic haemorrhage. Scand J Gastroenterol 1996: 31: 764-9.

<sup>&</sup>lt;sup>3</sup> Jensen DM+: Urgent colonoscopy for the diagnosis and treatment of severe diverticular disease. N Eng J Med 2000: 342: 78-82.

<sup>&</sup>lt;sup>4</sup> Hoedema RE+: The management of lower gastrointestinal haemorrhage. *Dis Col Rectum* 2005: 10: 1-25.



**Emergency Management of Colonic Haemorrhage** 

The systemic sequelae of major haemorrhage (tachycardia and hypotension) relate to the rate and volume of blood loss, and so the primary management objective is to identify the minority presenting with life-threatening acute or chronic haemorrhage.<sup>5</sup> The rate of rebleeding and need for surgical intervention along with overall mortality rate increase with advancing age, comorbidity and male gender.

The priorities of management include simultaneous resuscitation and clinical evaluation followed by identification of the site and cause of the bleeding. The aspirate from a nasogastric tube may indicate the need for gastroscopy. Following administration of an enema, a proctoscopy (c. 15% of significant lower GI bleeds arise from haemorrhoidal tissue) and sigmoidoscopy are performed. If haemodynamic stability is now established a colonoscopy within 12 hours of admission, following purgation, aids diagnosis and localisation of the bleeding site in 90% of patients with true colonic bleeding.<sup>3</sup> If the stigmata of recent or active bleeding (i.e. adherent thrombus or visible vessel) are evident, endoscopic haemostasis including 3-site adrenaline injection followed by application of a thermal probe topically and labeling with Indian ink can be performed. Variable rebleeding rates of 13 to 53% have been reported and many patients require multiple treatments.

If the colonoscopy is non-diagnostic, in the presence of continued bleeding, technetium–labeled red cell scintigraphy may detect bleeding up to 24 hours after injection. It requires a bleeding rate of 0.1 ml per minute. Its value is limited due to limited diagnostic accuracy (<50%).<sup>6</sup> Selective mesenteric angiography facilitates accurate localisation (40-86%) and provides the potentially therapeutic option of selective embolisation. However, it does not detect venous bleeding rate of 1 ml/minute. If a bleeding site is identified at angiography, therapeutic administration of intra-arterial pressors or microcoil embolisation (risk of bowel ischaemia) may be employed. If these measures are unsuccessful and the patient requires laparotomy, the angiographic catheter should remain in situ to offer methylene blue localisation during subsequent surgery.

<sup>&</sup>lt;sup>5</sup> Finlay IG: Lower gastrointestinal bleeding. *Recent Advances in Surgery* 2004: 27; 137-46.

<sup>&</sup>lt;sup>6</sup> Qureshi A+: Lower gastrointestinal bleeding. Surgical emergencies (1998). 14: 158-64. Blackwell science.



Care of Complex Surgical Patients Clinical Guidelines Emergency Management of Colonic Haemorrhage

A minority of patients proceed to surgery (*see Table 1*). Segmental colectomy (reported mortality of 2-10%), can be performed in patients in whom the area of bleeding can be clearly and confidently identified.<sup>6</sup> The availability of on-table endoscopy with transillumination may reduce the need for subtotal colectomy (reported mortality of 27%) which would otherwise be required if the bleeding is definitely colonic but can not be localised to either hemicolon. Subtotal colectomy is associated with a rebleeding rate of <1% compared to a rebleeding rate of 14% with positive angiography and 42% with a negative angiogram in segmental resections.<sup>4</sup>

#### Table 1: Indications for emergency surgery in patients with colonic haemorrhage

#### Indications for emergency surgery:

Clinical deterioration or persistent haemodynamic instability despite resuscitation.

Transfusion requirements exceeding 6 units.

Persistent and/or recurrent bleeding following other therapeutic modalities.


## 5. Oesophageal Perforation

#### **Brian J Manning**

#### INTRODUCTION

Although relatively uncommon, oesophageal perforation is a surgical emergency which, without early aggressive management and expert surgical intervention, is almost invariably fatal.<sup>1,2</sup>

#### AETIOLOGY

Spontaneous post-emetic rupture (Boerhaave's Syndrome) accounts for 15% of cases, foreign bodies 14% and trauma 10%.

Iatrogenic perforation is increasingly common (up to 75% in some series) as diagnostic oesophagoscopy becomes more often performed and as strictures and segments of achalasia are intubated and stented or dilated.

Endoscopic perforation usually occurs at the level of the cricopharyngeus in the cervical oesophagus or, less commonly, at the pre-hiatal level. In two thirds of the remaining cases, the tear is on the left side. In Boerhaave's syndrome, it occurs just above the diaphragm (being on the left side in 90% of patients).

#### PRESENTATION

Early diagnosis is the key to successful outcome, even in the minority of patients who are suitable for non-operative management.

A high index of suspicion must be maintained in any patient who presents with pain or fever following vomiting, oesophageal intubation or trauma.<sup>3</sup> However, vomiting does not always precede spontaneous perforation.

The fasting patient who has suffered a small oesophageal rent at endoscopy might have little if any mediastinal contamination whereas patients who present with Boerhaave's syndrome usually leak large amounts of partially digested material or alcohol.<sup>4</sup>

<sup>&</sup>lt;sup>1</sup> Reeder LB+: Current results of therapy for oesophageal perforation. *Am J Surg* 1995; 169: 615-7.

<sup>&</sup>lt;sup>2</sup> Bufkin BL+: Oesophageal perforation: emphasis on management. Ann Thorac Surg 1996; 61: 1447-51.

<sup>&</sup>lt;sup>3</sup> Weiman DS+: Noniatrogenic oesophageal trauma. Ann Thorac Surg 1995; 59: 845-9.

<sup>&</sup>lt;sup>4</sup> Vial CM+: Boerhaave's syndrome: diagnosis and treatment. *Surg Clin North Am* 2005; 85: 515-24.



The condition is commonly misdiagnosed as pancreatitis, myocardial infarction or dissection of a thoracic aortic aneurysm.

## DIAGNOSIS

A chest radiograph will demonstrate mediastinal emphysema in 40% of patients with mediastinal widening becoming apparent after several hours. Pneumothorax as a result of rupture of the mediastinal pleura is a common finding with pleural effusion secondary to mediastinal inflammation occurring late. In 10% of cases the chest radiography will be normal.

Contrast oesophagography using water soluble contrast (i.e. gastrograffin) must be performed urgently to demonstrate the presence and location of perforation. A negative barium study rules out a rupture.

Contrast enhanced CT or oesophagoscopy is of little value at the diagnostic stage but may be used if the patient is sedated or intubated and unable to swallow.

#### MANAGEMENT

Initial management goals, once the diagnosis is established, includes fluid resuscitation of the patient, establishment of appropriate physiological monitoring and limitation of mediastinal contamination by means of broadspectrum antibiotics, careful nasogastric tube placement and intercostal chest drain insertion when there is a pleural effusion or pneumothorax.

Although some have reported satisfactory results with an 'aggressive conservative' approach (comprising early nasogastric intubation, intercostal tube drainage, repeated radiological imaging and radiologically placed tubes and catheters to drain collections)<sup>5</sup>, more convincing results have been reported following surgical intervention in most series.<sup>6,7</sup> Therefore, only those patients with mild symptoms and minimal signs of sepsis and whose perforations are shown by contrast studies to be contained within the mediastinum and are draining back into the oesophagus should be considered for conservative treatment.

<sup>&</sup>lt;sup>5</sup> Vogel SB+: Oesophageal perforation; aggressive, conservative treatment lowers morbidity and mortality. Ann Surg 2005; 241: 1016-23.

<sup>&</sup>lt;sup>6</sup> Whyte RI+: Intrathoracic oesophageal perforation. The merit of primary repair. J Thorac Cardiovasc Surg 1995; 109: 140-4.

<sup>&</sup>lt;sup>7</sup> Richardson JD. Management of oesophageal perforations: the value of aggressive surgical treatment. *Am J Surg* 2005; 190: 161-5.



The common goal of all surgical interventions is to drain contamination and prevent further soiling with the specific approach being determined by the site of the perforation.

Cervical perforations are usually managed by drainage alone, through an incision along the anterior border of the left sternocleidomastoid muscle extending from the level of the cricoid cartilage to the sternal notch. Access to the prevertebral space allows irrigation of contaminated tissues and insertion of drains. With more extensive soiling, transthoracic drain insertion may also be necessary. Perforation of the thoracic oesophagus is approached on the side of the tear via a posterolateral thoracotomy at the appropriate intercostal level. In most cases, primary repair is possible, in 2 layers, after full exposure of the defect and debridement of necrotic edges if necessary. The technique of closure varies with the choice of pleural flap or muscle flaps from the diaphragm, the intercostal muscles or the rhomboid muscle or of fundoplication allowing repair to be individualized to the nature of the defect and to the surgeon according to preference. Distal obstruction must be relieved at the time of initial surgery and so myotomy (for achalasia) or resection of tumours or strictures may be indicated (with immediate or delayed interposition of stomach, colon or small bowel). Although more favoured in the past, diversion with end or side cervical oesophagostomy still has a role.

#### **DELAYED PRESENTATION**

Mortality increases with time from perforation to surgical closure, owing to the fulminant mediastinal inflammatory reaction that develops in response to extruded secretions and food debris. As time progresses, primary closure of the oesophageal tear become less feasible, as tissues become inflamed and devitalised, and resection with or without oespohagostomy becomes more likely.<sup>8</sup> Mortality rates therefore range from 1-20% in those undergoing early repair to approximately 50% in those with a delayed presentation. Although the poor outcome reported previously in such patients has been used as an argument against operative intervention, late presentation is not a contraindication to surgery and good results have been obtained even after 48 hours.

<sup>&</sup>lt;sup>8</sup> Salo JA+: Management of delayed oesophageal perforation with mediastinal sepsis. Oesophagectomy or primary repair? *J Thorac Cardiovasc Surg* 1993; 106: 1088-91.



Care of Complex Surgical Patients Clinical Guidelines

# 6. Intraperitoneal Hollow Viscus Perforation

### Martin J O'Sullivan

#### **INTRODUCTION**

Hollow viscus perforation (HVP) remains one of the commonest reasons for emergency laparotomy (*see Table 1*) and can be associated with high rates of morbidity and mortality.<sup>1</sup> The focus here is primarily on peptic ulcer disease (PUD) and colonic perforation.

The most important aspect of managing HVP is the recognition that it may be present.

#### Table 1: Common causes of intraperitoneal hollow viscus perforation

Perforated appendicitis
Peptic ulcer disease (duodenal or gastric ulcer)
Colonic (diverticular, carcinoma, inflammatory bowel disease)
Small bowel (IBD, mesenteric ischaemia)
Gallbladder
latrogenic – laparoscopy, ERCP, other endoscopic procedures
Anastomotic leak
Trauma – blunt or penetrating

#### **HISTORY**

- The most characteristic symptom is the suddenness of the onset of the pain (typically at the site of perforation) – indeed the patient can often tell exactly what time the pain commenced.
- The pain rapidly becomes generalised although occasionally in PUD it moves selectively to the right lower quadrant (due to tracking of gastric contents down the right paracolic gutter).

<sup>1</sup> Anderson ID+: Laparotomy for abdominal sepsis in the critically ill. *Br J Surg* 1996; 83: 535-9.



 Up to 90% of patients with PUD will give a history of previous dyspepsia, previous/current treatment for a duodenal ulcer or ingestion of ulcerogenic drugs.

#### SIGNS

- The patient usually is in obvious pain, looks ill (pale & diaphoretic) and is reluctant to move.
- While tachycardia is usually evident, hypotension is a late finding as is a high fever.
- The patient will usually be most tender over the site of the perforation.
- The characteristic abdominal finding is "board-like" rigidity.
- Bowel sounds are usually absent although not inevitably so.
- With time (3-6 hours), the patient may improve as exudation dilutes the peritoneal contamination.
- After approximately six hours, however, signs and symptoms of diffuse bacterial peritonitis supervene.

Note: The signs are less obvious in certain patient groups (e.g. advanced age, immunosuppression, steroids, diabetics, quadriplegics, comatose). A high index of suspicion is therefore required in such patients.

#### **INVESTIGATIONS**

#### **Blood tests**

- Full blood count (leucocytosis, haemoconcentration [dehydration], anaemia [? tumour]).
- Serum electrolytes (dehydration pattern and hypokalaemia).
- Glucose beware of diabetic ketoacidosis, which may also present with severe abdominal pain.
- Amylase pancreatitis is the main differential diagnosis in those with severe epigastric pain. Although the serum amylase will often be elevated in PUD, it is usually not at a level diagnostic of pancreatitis.



- Arterial blood gases (ABGs) and serum lactate especially if mesenteric ischaemia is suspected. However, the specificity of both tests is poor especially early after the onset of the pain.
- Group and cross match (4-6 U).
- Electrocardiogram (ECG) both to out-rule myocardial infarction in the differential and as a preoperative assessment.

#### Imaging

- An erect chest x-ray (CXR) (after sitting upright for 15-20 minutes) may show free air under the diaphragm in 70-80% of perforations. Beware of confusing this with the gastric air bubble or indeed a dilated colon (Chilaiditi's sign). A lateral decubitus film may show free air if the erect CXR is normal.
- Computed tomography (CT) with water soluble contrast is indicated if there is no evidence of pneumoperitoneum on plain radiology. CT may also help to localise the site of the perforation, if this is not clear clinically. This, in turn, may aid operative planning in terms of an appropriate incision. With the advent of CT, upper and lower gastrointestinal contrast studies are seldom required.

In some cases, all investigations may be negative or inconclusive. Clinical findings alone must then govern management. Do not delay surgical intervention if the clinical findings warrant operation in the absence of ancillary support for the diagnosis. It is often better to "look and see" rather than "wait and see".

#### **INITIAL MANAGEMENT**

- Aggressive, initial resuscitation is required and should take place immediately on reviewing the patient. Infusion of three to four litres of i.v. fluids is frequently required for resuscitation.
- A urinary catheter should be placed to monitor hourly urine output. In very ill patients, central venous access may be required.
- A nasogastric tube should always be passed.



- Broad spectrum intravenous antibiotics should be commenced for a therapeutic course. Cefuroxime and metronidazole is satisfactory in most cases. Consider gentamicin (watch renal function) for the more seriously ill.
- A proton pump inhibitor (PPI) should be commenced, especially if an upper GI perforation is suspected.
- Institute appropriate thrombo-embolism prophylaxis.
- Do not forget analgesia. Intravenous morphine may be appropriate.
- Inform the anaesthetist early in management.
- Consider also at this stage where the patient is likely to end up postoperatively – is intensive care, a high dependency unit or an observation ward the most likely destination for the patient?
- Consent for surgery must be sought including the possibility of a stoma pre-operatively.

#### **DEFINITIVE TREATMENT**

- Although not all patients with perforated PUD require intervention (some patients will seal off the perforation with omentum almost immediately and so be suitable for a conservative approach), the standard of care remains prompt laparotomy (after resuscitation) with appropriate repair (usually primary closure with the use of an omental patch).<sup>2</sup>
- Laparoscopy and washout may be sufficient for sealed cases of PUD. It has also been described for cases of perforated diverticular disease where there is little or no contamination.<sup>3</sup> Laparoscopic repair of perforated PUD can be performed if the expertise is available<sup>4</sup>, although a recent review indicated that it may not be as advantageous as initially thought, especially in high risk patients.<sup>5</sup> If there is any doubt during the procedure, do not hesitate to convert to a laparotomy.

<sup>&</sup>lt;sup>2</sup> Barksdale AR+: Current management of perforated peptic ulcer. *Curr Surg* 2000; 57: 594-9.

<sup>&</sup>lt;sup>3</sup> O'Sullivan GC+: Laparoscopic management of generalized peritonitis due to perforated colonic diverticula. *Am J Surg* 1996; 171: 432-4.

<sup>&</sup>lt;sup>4</sup> Lee FY+: Selection of patients for laparoscopic repair of perforated peptic ulcer. *Br J Surg* 2001; 88: 133-6.

<sup>&</sup>lt;sup>5</sup> Lunevicius R+: Systematic review comparing laparoscopic and open repair for perforated peptic ulcer. Br J Surg 2005; 92: 1195-207.



In cases of colonic perforation, resection of the affected bowel is usually required. As primary anastomosis is contra-indicated in the presence of gross contamination diversion by means of a stoma is necessary (i.e. end colostomy in the setting of a perforated sigmoid diverticulum (Hartmann's procedure)).<sup>6</sup> Iatrogenic injury, when recognised early following colonoscopy of an adequately prepared ("clean") colon, may be managed by primary repair without defunctioning depending on the severity of the injury and the degree of contamination present.

## **POST-OPERATIVE CARE**

- Appropriate attention to the haemodynamic state of the patient is required.
- Antibiotics should be continued for a therapeutic course.
- Eradication of H Pylori is recommended in those with duodenal ulcers.<sup>7</sup>
- Early mobilization and chest physiotherapy are crucial elements of postoperative convalescence.
- A high level of vigilance for infectious complications (e.g. abscess) is also necessary.

<sup>&</sup>lt;sup>6</sup> Bausch K+: Surgical management of severe secondary peritonitis. *Br J Surg* 1999; 86: 1371-7.

<sup>&</sup>lt;sup>7</sup> Ng EK+: Eradication of H. pylori prevents recurrence of ulcer after simple closure of duodenal ulcer perforation. Ann Surg 2000; 231: 153-8



## 7. Severe Biliary Sepsis

#### **Paul F Ridgway**

#### **INTRODUCTION**

- Bile is typically sterile and so bacterial colonisation usually only occurs in the presence of outflow obstruction<sup>1</sup> or after instrumentation of the biliary tract (with or without stent/drain placement).
- The common causes of acquired obstruction include choledocholithiasis, malignancy, iatrogenic and, rarely, cholelithiasis (Mirizzi's Syndrome<sup>2</sup>).
- Organisms penetrate an obstructed biliary system via either the portal circulation or by ascending from the duodenum.
- Between 22%<sup>3,4</sup> to over 90%<sup>5</sup> of patients with biliary sepsis present with the classical (Charcot's) Triad of pain, rigors and jaundice.<sup>6</sup> The additional features of cognitive dysfunction and hypotension (Reynold's pentad<sup>7</sup>) imply imminent multiple organ dysfunction syndrome.

#### AETIOLOGY

- Biliary pathogens in community acquired sepsis are similar to isolates found with asymptomatic bactobilia.
- Two-thirds of cases are associated with more than one pathogen in the bile cultures.<sup>8</sup>
- Polymicrobial infection of bile remains the rule even though a single pathogen is isolated in blood cultures in over 90% of patients.

<sup>&</sup>lt;sup>1</sup> Sinanan MN. Acute cholangitis. *Infect Dis Clin North Am* 1992; 6: 571-99.

<sup>&</sup>lt;sup>2</sup> Mirizzi PL. Sindrome del conducto hepatico. *Journal international de chirurgie, Bruxelles* 1948; 8: 731-77.

<sup>&</sup>lt;sup>3</sup> Chew RK+: Acute cholangitis in Singapore. *Ann Acad Med Singapore* 1986; 15: 172-5.

<sup>&</sup>lt;sup>4</sup> Csendes A+: Risk factors and classification of acute suppurative cholangitis. *Br J Surg* 1992; 79: 655-8.

<sup>&</sup>lt;sup>5</sup> Rubin JR+: Diagnosis of choledocholithiasis. *Surg Gynecol Obstet* 1983; 156: 16-20.

<sup>&</sup>lt;sup>6</sup> Charcot J. M. Leçons sur les maladies du foie, des voies biliaires et des reins faites à la Faculté de Médecine de Paris. Paris: Bureaux du Progrés Médical & Adrien Delahaye; 1877.

<sup>&</sup>lt;sup>7</sup> Reynolds BM+: Acute obstructive cholangitis: a distinct clinical syndrome. Annals of Surgery 1959; 150: 299-303.

<sup>8</sup> Leung JW+: Antibiotics, biliary sepsis, and bile duct stones. Gastrointest Endosc 1994; 40: 716-21.



**Severe Biliary Sepsis** 

Pathogen	+ Bile Culture (%)	+ Blood Culture (%)	Pathogen	+ Bile Culture (%)	+ Blood Culture (%)
Escherichia Coli	27	71	Candida sp	4	0
Klebsiella sp	17	14	Citrobacter sp	3	2
Enterococcus sp	17	0	Proteus sp	3	0
Enterobacter sp	8	14	Clostridium sp	2	0
Streptococcus sp	8	0	Acinetobacter sp	1	0
Pseudomonas aeruginosa	7	4	Bacteroides	1	1

#### Table 1: Common biliary pathogens (modified from Leung et al<sup>8</sup>)

#### MANAGEMENT

Antibiotics are utilised to decrease septic complications (classically renal failure and hepatic abscess).<sup>9,10</sup>

- Empiric broad spectrum antibiotics with known biliary penetrance should be initiated once the diagnosis is suspected and blood cultures taken.<sup>11</sup> The regimens should last for 7-10 days.
- Over 80% of cases will respond to antibiotic therapy alone and so emergency drainage procedures may be avoided in those cases where clinical improvement is seen within the first 24-48 hours.<sup>12</sup>
- There is no one regimen that is evidently superlative.
  - Fluroquinolones are particularly useful as they concentrate in the bile even in obstructed systems (usually in levels significantly higher than the minimum inhibitory concentrations for gram negative

<sup>&</sup>lt;sup>9</sup> Lipsett PA+: Acute cholangitis. *Front Biosci* 2003; 8: s1229-39.

<sup>&</sup>lt;sup>10</sup> Lipsett PA+: Acute cholangitis. *Surg Clin North Am* 1990; 70: 1297-312.

<sup>&</sup>lt;sup>11</sup> van den Hazel SJ+: Role of antibiotics in the treatment and prevention of acute and recurrent cholangitis. *Clin Infect Dis* 1994; 19: 279-86.

<sup>&</sup>lt;sup>12</sup> Boender J+: Endoscopic sphincterotomy and biliary drainage in patients with cholangitis due to common bile duct stones. Am J Gastroenterol 1995; 90: 233-8.



bacteria).<sup>8</sup> Single agent Ciprofloxacin 200mg BD intravenous (iv) is comparable to triple agent ceftazidime (1 g bd iv), ampicillin (500 mg qds iv) and metronidazole (500 mg tds iv).<sup>13</sup> Its expected efficacy is over 80%. It is noteworthy that the dosage of ciprofloxacin currently recommended is 400mg BD iv.

- Piperacillin/ Tazobactam has excellent cover against biliary pathogens, however resistance patterns among E.Coli, Klebsiella and Pseudomanads have been reported.<sup>14</sup> It is also queried whether it has good biliary penetrance in an obstructed system. If pseudomonad infection is suspected double antibiotic coverage is desirable.<sup>15</sup>
- Second (e.g. Cefuroxime) and third generation cephalosporins (e.g. Ceftazidime) lack efficacy against Enterococcus infections and are therefore usually used in combination with an aminoglycoside (e.g. gentamicin).
- Care needs to be employed in utilizing aminoglycosides in view of their potentiated nephrotoxicity in the presence of obstructive jaundice.<sup>16</sup>

*Coagulopathy* should be concomitantly addressed by administering parenteral vitamin K (10 mg iv).

*Imaging:* Obstruction of the biliary tree should be determined in the first instance by ultrasonography or magnetic resonance imaging. Computerised tomography is useful as an adjunct where a malignant obstruction is suspected.

*Drainage:* For ethical reasons, no randomised trials have been performed comparing drainage versus observation alone. Options include ERCP with sphincterotomy and/or stenting, percutaneous transhepatic or nasobiliary drainage. The timing of drainage is based upon the severity of infection, persistence of the obstruction and availability of local expertise. Endoscopic sphincterotomy, stenting or percutaenous drainage are mandated to ensure

<sup>&</sup>lt;sup>13</sup> Sung JJ+: Intravenous ciprofloxacin as treatment for patients with acute suppurative cholangitis: a randomized, controlled clinical trial. J Antimicrob Chemother 1995; 35: 855-64.

<sup>&</sup>lt;sup>14</sup> McGrath K+: Infections of the Biliary tract. *Current Treatment Options in Infectious Diseases* 2001; 3: 403-18.

<sup>&</sup>lt;sup>15</sup> van den Hazel SJ+: Biliary excretion of ciprofloxacin and piperacilliin in the obstructed biliary tract. Antimicrob Agents Chemother 1996; 40: 2658-60.

<sup>&</sup>lt;sup>16</sup> Lucena MI+. Aminoglycoside-associated nephrotoxicity in extrahepatic obstructive jaundice. *J Hepatol* 1995; 22: 189-96.



relief of persistent biliary obstruction.<sup>17</sup> Drainage of an obstructed biliary system is desirable at the earliest available juncture (within 72 hrs) where empiric antibiotic therapy does not induce clinical improvement.<sup>18</sup>

Definitive surgical drainage is rarely now required in the emergency setting. It should be undertaken where other methods fail and there is a worsening of the patient's condition. The minimum required is a laparotomy with placement of a drainage tube in the gallbladder. There is probably an increased incidence of complications when laparoscopic cholecystectomy is undertaken as definitive management of choledocholithasis early in the illness.<sup>19</sup>

First principles should be applied for each acute presentation in cases of recurrent cholangitis (3 or more episodes, usually occurring in those with biliary-enteric anastomoses or indwelling stents) with instigation of longer term prophylaxis thereafter.<sup>11</sup> Duration of therapy is unknown but suggested to be 3-4 months.

Iatrogenic/Procedural acquired ascending cholangitis: Endoscopic and operative manipulation of the biliary tree is an increasing cause of ascending cholangitis. Organism profiles are similar to community acquired sepsis, hence antibiotic regimens are similar. Both from cost and clinical standpoints, prophylaxis is desirable where the biliary tract is to be manipulated. Historically, intravenous regimens have been used although two randomised trials suggest oral fluroquinolones are equivalent.<sup>20,21</sup> One small, randomised trial showed a non-significant reduction in post-ERCP cholangitis with iv piperacilliin.<sup>22</sup>

<sup>&</sup>lt;sup>17</sup> Leung JW+:. Urgent endoscopic drainage for acute suppurative cholangitis. *Lancet* 1989; 1: 1307-9.

<sup>&</sup>lt;sup>18</sup> Lillemoe KD. Surgical treatment of biliary tract infections. *Am Surg* 2000; 66:138-44.

<sup>&</sup>lt;sup>19</sup> Thompson BF+: Antibiotic prophylaxis prior to endoscopic retrograde cholangiopancreatography in patients with obstructive jaundice: is it worth the cost? Aliment Pharmacol Ther 2002; 16: 727-34.

<sup>&</sup>lt;sup>20</sup> Mehal WZ+: Antibiotic prophylaxis for ERCP: a randomized clinical trial comparing ciprofloxacin and cefuroxime in 200 patients at high risk of cholangitis. *Eur J Gastroenterol Hepatol* 1995; 7: 841-5.

<sup>&</sup>lt;sup>21</sup> Davis AJ+:. Antibiotic prophylaxis for ERCP: a comparison of oral ciprofloxacin with intravenous cephazolin in the prophylaxis of high-risk patients. *Aliment Pharmacol Ther* 1998; 12: 207-11.

<sup>&</sup>lt;sup>22</sup> van den Hazel SJ+: Piperacillin to prevent cholangitis after endoscopic retrograde cholangiopancreatography. A randomized, controlled trial. Ann Intern Med 1996; 125: 442-7.



- Ascending cholangitis in the immunosuppressed: The latter stages of AIDS are associated with increased incidence of cholangitis.<sup>14</sup> Patients present with pain and fever, although jaundice is less common. Parasites (cryptosporidium) and viruses (CMV) are commonly implicated as well as the pyogenic organisms previously listed. Therapy should include antiviral (gancylcovir) and anti parasitic (albendazole) agents.
- There are no reports that **diabetic** or **steroid-related cholangitis** behaves dissimilarly to community acquired cholangitis.

Summary Recommendations for the Management of Severe Bilary Sepsis	Strength of evidence
Empiric broad-spectrum antibiotics with known biliary penetrance	Grade B
Single agent Ciprofloxacin (400 mg bd iv) is adequate empiric antibiotic therapy	Grade A
Vitamin K (10 mg iv should be administered) to correct/prevent coagulopathy	Grade C
Drainage (preferably by sphincterotomy+/– stenting) is mandated to ensure relief of biliary obstruction	Grade A
lf no clinical improvement occurs with antibiotic therapy, drainage should be undertaken within 48 hrs	Grade C
Single agent oral Ciprofloxacin is adequate prophylactic antibiotic therapy	Grade A



Care of Complex Surgical Patients Clinical Guidelines

## 8. Initial Management of Acute Pancreatitis

Modified from the RCSI Clinical Guidelines on Management of Acute Pancreatitis<sup>1,2</sup>

#### INTRODUCTION

Despite changes in management, the morbidity associated with acute pancreatitis remains high while mortality rates approximate 10% in many series (rising to 30% for those with severe disease). The evidence base for many aspects of acute pancreatitis care is poor, hence, individual clinical judgement remains important.

#### DIAGNOSIS

- Clinical examination in the first 24 hours lacks sensitivity and should be supported by objective, predictive measures (Grade A).
- A four-fold rise in serum pancreatic amylase in an appropriate clinical setting is considered diagnostic (Grade B). The sensitivity of serum amylase decreases with time from onset of abdominal pain and so the level of hyperamylasaemia should be interpreted accordingly. Urinary amylase levels remain elevated longer than serum values and hence may be a useful adjunct to diagnosis. Serum hyperlipasaemia persists longer and is more sensitive as the pancreas is the only source of lipase (Grade A). Patients with recurrent acute or chronic pancreatitis may not have sufficient pancreatic reserve to manifest hyperamylasaemia.
- Urea and electrolytes, glucose, liver functions tests (LFTS), calcium, albumin and arterial blood gases should be measured at baseline to facilitate predicted severity assessment (*see Table 1*). For an Irish and UK population (with predominantly gallstone and alcohol-related disease), the Glasgow (Imrie) score has been validated. No one scoring test has been validated for those with idiopathic disease, however there are no data to suggest that Glasgow scoring is not valid in this group as well. The Ranson criteria were validated only in a North American population

R.C.S.I. Clinical Guidelines Committee. Management of Acute Pancreatitis. Clinical Guidelines. R.C.S.I. 2003.
 Working party for the British Society of Gastroenterology, Association of Surgeons of Great Britain and Ireland, Pancreatic Society of Great Britain and Ireland, Association of Supper GI surgeons of Great Britain and Ireland. UK guidelines for the management of acute pancreatitis. *Gut* 2005; 54 Suppl 3: iii.9.



with alcohol induced pancreatitis. Other valid scoring systems include APACHE II and Atlanta criteria.

- C-reactive protein levels provide an indication of outcome after 48 hours (Grade A).
- Early, elevation in cholestatic liver enzymes suggests a gallstone aetiology.
- Serum calcium and fasting lipids should be examined after convalescence if aetiology unclear.
- An erect chest x-ray should be performed to aid in the exclusion of visceral perforation as a differential diagnosis.
- A minimum of abdominal ultrasonography should be performed to delineate the presence of gallstones regardless of perceived aetiology.
- Abdominal CT should be performed early only in those where the diagnosis is in doubt. Early imaging has not been shown to influence mortality although it maybe used to predict severity<sup>3</sup> (IAP Guidelines<sup>4</sup>) (*see Table 2*). At 5-10 days CT (esp. pancreas protocol with multi-detector scanner) is a useful tool to delineate pancreatic necrosis as well as acute fluid collections.

#### MANAGEMENT

*Mild pancreatitis (Glasgow score 0-1)* – Basic vital signs should be recorded and intravenous fluids administered. Nasogastric drainage is rarely indicated and is necessary only for persistent vomiting. A urinary catheter, antibiotics and CT scanning are not usually necessary. The patient should be advised to fast initially but should be allowed to resume oral intake as pain symptoms abide. A normalising serum amylase in conjunction with a falling CRP are taken as useful biochemical evidence of impending resolution. If gallstone aetiology is confirmed, it is reasonable to undertake cholecystectomy on the same hospital admission (Grade C), although persistent choledocholithiasis should be ruled out (preferably) preoperatively (usually by MRCP).

*Predicted-severe pancreatitis (Glasgow score 3+)* – patients require high dependency/intensive care and hourly monitoring of vital signs (Grade B).

<sup>&</sup>lt;sup>3</sup> Balthazar A+: Acute pancreatitis: value of CT in establishing prognosis. *Radiology* 1990; 174: 331-6.

<sup>4</sup> Uhl W+: IAP Guidelines for the surgical management of acute pancreatitis. Pancreatology 2002; 2: 565-73.



Urinary catheter is mandatory with or with out central venous access and monitoring. Aggressive fluid resuscitation should be undertaken to ensure a urinary output >0.5ml/kg/hour. Regular monitoring of oxygen saturation should be employed.

- Patients predicted to have severe disease should be considered for referral to a specialist unit particularly if multiple fluid collections and/or >30% pancreatic necrosis is present on CT scanning or the patient has multiple organ failure requiring support (Grade B).
- Urgent ERCP (within 72 of the onset of the pain) is necessary in cases of severe pancreatitis that are suspected or proven to be due to gallstones (Grade B). Endoscopic sphincterotomy should be undertaken whether or not persistent choledocholithiasis is demonstrated (Grade C).
- There is no consensus on prophylactic antibiotic regimens in pancreatitis. Indeed, the 2003 Cochrane review highlighted the difficulties in interpreting this data. Prophylactic antibiotics may decrease infection rates where pancreatic necrosis exists, as well as the need for surgical intervention but this has not been shown to affect survival. They may also predispose to increased systemic fungal septicaemia. Data are available on pancreatic penetrance and outcomes for imipenium (widely used in North America), ciprofloxacin (widely used in continental Europe) and cerfuroxime (Ireland and the UK). Where carbepenems are utilised there is a higher rate of fungaemia (up to 33%) and so some centres recommend concomitant empiric antifungal prophylaxis.
- Nutritional support is required in all patients with severe pancreatitis. The enteral route should be used if tolerated (Grade A). Enteral feeding by nasojejeunal tube is generally preferred, although its superiority relative to central venous or indeed nasogastric feeding is not proven.
- Surgical intervention is required in patients with documented infected pancreatic necrosis. Image-guided fine needle aspiration for bacteriology can be used to differentiate between sterile and infected pancreatic necrosis in patients showing signs of clinical deterioration or sepsis. It is unclear whether advances in minimally invasive radiological drainage represent a safe alternative to the gold standard surgical necrosectomy. Concomitant cholecystectomy should be performed at the same time as the necrosectomy if a biliary aetiology has been proven.



#### Initial Management of Acute Pancreatitis

#### Table 1: Scoring systems for severity assessment in acute pancreatitis

Glasgow (Imrie) Criteria	Ranson criteria		
WCC >15x10 <sup>9</sup> /L	At presentation	Within 48 hours	
Blood glucose >10 mmol/L	Age >55 years	Haematocrit fall >10%	
Blood urea >16 mmol/L	WCC >16x10 <sup>9</sup> /L	Blood urea >16 mmol/L	
LDH >600 IU/L	Blood glucose >10 mmol/L	Serum Ca++ <2 mmol/L	
AST >200 IU/L	LDH >350 IU/L	Arterial PaO2 <8 kPa	
Plasma albumin <32 g/L	AST >250 IU/L	Base deficit >4 mmol/L	
Uncorrected plasma Ca <sup>++</sup> <2 mmol/L		Fluid sequestration >6L	
Arterial PaO <sub>2</sub> <8 kPa			

Greater then three criteria by either scoring system predicts severe disease.

#### Table 2: Indicators of disease severity on CT scanning.

CT findings associated with increased severity of acute pancreatitis		
Enlargement of pancreatic gland	Ill-defined margins	
Abnormal enhancement	Pancreatic gas indicative of necrosis/abscess formation	
Thickening of peripancreatic planes	Blurring of fat planes	
Intra – and retro-peritoneal fluid collections	Pseudocyst formation	
Pleural effusions		



Care of Complex Surgical Patients Clinical Guidelines

# 9. Triggers for Operation in Patients with Ulcerative Colitis

### **Conor J Shields**

#### INTRODUCTION

Surgery is an increasingly popular option for patients with ulcerative colitis given the continuing refinement of continence-restoring techniques. Operation may be undertaken in both elective and emergency scenarios, with indications for intervention differing according to specific circumstances and the preferred surgical approaches. While, in total, 33% of patients undergo surgery within 10 years of their diagnosis, 2/3 of these (20% overall) require emergency intervention due to the complications of fulminant disease.

Emergency	Elective
Toxic colitis	Failure of medical therapy
Toxic megacolon	Complications of long term steroid use
Perforation	Nutritional difficulties
Haemorrhage	Growth retardation (children)
	Poor quality of life
	Risk or evidence of dysplasia or cancer

#### Table 1: Indications for surgical intervention in patients with ulcerative colitis

#### **INDICATIONS FOR EMERGENCY SURGERY**

Steroid-refractory **toxic colitis** is a potentially lethal complication of UC. It is manifest clinically by diffuse abdominal tenderness and systemic signs of toxicity such as tachycardia, fever (>38°C), leucocytosis, anaemia, and hypoalbuminaemia. Initial treatment entails fluid resuscitation, nasogastric decompression, high-dose intravenous steroids, broad-spectrum antibiotics and nutritional supplementation (to prevent nutritional deterioration and correct pre-existing deficiencies). Barium enema, narcotics, anticholinergic drugs, NSAIDS and anti-diarrhoeal agents should be avoided because they may precipitate toxic megacolon. Patients should be followed with haematological and biochemical blood tests as well as plain radiographs



**Triggers for Operation in Patients with Ulcerative Colitis** 

of the abdomen and repeated clinical examination on a daily basis. Surgery is indicated for patients who either deteriorate or do not improve within 48 to 72 hours of institution of medical therapy, or if further complications of fulminant disease develop (i.e. toxic megacolon or perforation). Patients who improve initially but remain unwell after 5-7 days of iv corticosteroids may be considered for a trial of cyclosporin (4 mg/kg/d) before surgery. While this may be initially effective for induction of remission, nearly 50% relapse and require an intervention within 3 years. Although infliximab (anti-TNF- $\alpha$  monoclonal antibody) has open-label data to support its application in severe UC, there are no prospective studies yet in fulminant colitis.

Toxic megacolon is a life-threatening (associated mortality=1-3%) variant of toxic colitis associated with decompensation of the colon wall. Total or segmental non-obstructive dilatation occurs due to extension of the mucosal inflammation into the colonic smooth-muscle layer with destruction of ganglion cells and resultant paralysis of the bowel wall. A dilated and oedematous colon is apparent on plain radiographs.

10% of all emergency colectomies for UC are accounted for by **haemorrhage**. These patients are frequently very debilitated and are usually managed best with a subtotal colectomy and end ileostomy (once the possibility of bleeding from the rectum has been discounted). Endoscopic treatment of massive bleeding in UC is not possible because of the diffuse nature of colonic inflammation.

In selecting the operation, one must adopt an approach of an appropriate extent for a debilitated patient that maximises the chance of cure with an acceptable functional outcome. This is a complex decision that requires insight into the clinical condition as well as the practical aspects and sequelae of any proposed intervention.

Subtotal colectomy with end ileostomy is the preferred surgical approach in emergency cases, as it removes the majority of the diseased bowel and avoids a potentially hazardous and fraught pelvic dissection (thereby decreasing the risk of venous bleeding, impotence, and neurogenic bladder) and does not compromise any future attempts at reconstruction. The remaining colon and rectum may be managed as a closed distal stump (with rectal drain in the immediate post-operative phase), or as a mucous fistula in the case of toxic megacolon or a very diseased rectum. Formation of an ileorectal anastomosis is deprecated due to the unacceptably high rate of anastomotic leakage.



Laparoscopic approaches to the toxic colon have been reported, but these reports have been of an anecdotal and "single surgeon" nature. Concerns regarding the length of operation and potential for damage to a fragile and friable colon persist.<sup>1</sup>

Symptoms and signs	Diagnostic abnormalities	Plain film findings	Initial management
Abdominal pain	Anaemia		Stool cultures
Bloody diarrhoea	White cell count (>20x10 <sup>9</sup> /L)	Dilated colon (transverse >6 cm) with loss of haustral	PFA
Tenesmus	Elevated ESR (>40)	patterns	Limited sigmoidoscopy
Fever	Electrolyte	Intravenous fluids	
Tachycardia	derangement (including	gement Oedematous, ding irregular colon with natraemia & thumb printing kalaemia)	RCC transfusion
Dehydration	hyponatraemia & hypokalaemia)		Rest bowel (fasting)
Weight loss	Metabolic alkalosis	Pneumatosis coli (rare)	Intravenous steroids (hydrocortisone 100 mg iv qds)
Orthostatic hypotension	Hypoalbuminaemia		Board spectrum antibiotics
Distended, tympanic abdomen			Avoid antidiarrhoeals & NSAIDs
Mental obtundation			Nasogastric tube

#### Table 2: Fulminant colitis and toxic megacolon<sup>2</sup>

<sup>&</sup>lt;sup>1</sup> Blumberg D+: Surgery for ulcerative colitis. *Gastroenterol Clin N Am* 2002; 31: 219–35

<sup>&</sup>lt;sup>2</sup> Cheung O+: Inflammatory bowel disease emergencies. *Gastroenterol Clin N Am* 2003; 32: 1269-88.



## 10. Acute Mesenteric Ischaemia

#### **Emmet Andrews**

### INTRODUCTION

Acute mesenteric ischaemia (AMI) is a life-threatening vascular emergency that requires early diagnosis and treatment in order to prevent/treat bowel necrosis and avoid patient death.<sup>1</sup> AMI is not an isolated clinical entity, but a complex of diseases, including acute mesenteric arterial embolus and thrombus, mesenteric venous thrombus, and non-occlusive mesenteric ischaemia (NOMI). It remains a difficult clinical problem with a high mortality rate of 70% to 90%, primarily due to delayed diagnosis. However, the contemporary management of AMI incorporates (as appropriate) effective anticoagulation, early operation for the purpose of revascularisation and/or resection of nonviable bowel with liberal use of relaparotomy to attempt to improve patient outcome.<sup>2</sup>

### AETIOLOGY

Three processes cause AMI by means of arterial inflow insufficiency:<sup>3</sup>

- Emboli (28%) that originate from another site (usually heart or thoracic aorta) and lodge typically in the Superior Mesenteric Artery (SMA) (usually just distal to the origin of the middle colic artery which therefore permits some limited perfusion of the proximal small intestine).
- 2. Thrombosis (64%) of an artery with pre-existing atherosclerosis. Acute thrombosis of the SMA occurs with sudden occlusion of a severely stenotic atherosclerotic lesion, typically at the origin of the artery resulting in severe ischaemia of the entire small bowel and a very poor prognosis.
- Non-occlusive mesenteric ischaemia (8%) caused by severe, prolonged visceral vasoconstriction in the peripheral arteries of the intestine without actual occlusion of the proximal arteries. Most commonly seen with pharmacological vasoconstrictors in critically ill patients. Mortality is 80%.

<sup>&</sup>lt;sup>1</sup> Park WM+: Contemporary management of acute mesenteric ischaemia: Factors associated with survival. J Vasc Surg 2002; 35: 445-52.

<sup>&</sup>lt;sup>2</sup> Yasuhara H. Acute mesenteric ischaemia: the challenge of gastroenterology. *Surg Today* 2005; 35: 185-95.

<sup>&</sup>lt;sup>3</sup> Oldenburg WA+: Acute mesenteric ischaemia: a clinical review. *Arch Intern Med* 2004; 164: 1054-62.



#### Acute Mesenteric Ischaemia

Venous thrombosis (most often complete occlusion of the superior mesenteric vein) may also cause bowel ischaemia by affecting inflow by means of outflow obstruction and accounts for 5-15% of cases of AMI. It may be primary (idiopathic) (prothrombotic states and haematological disorders) or secondary (previous abdominal surgical procedures, inflammation, abdominal infection, pancreatitis, oral contraceptives, malignant tumours) in nature. In many cases, multiple risk factors are present synchronously. The mortality rate among patients with acute mesenteric venous thrombosis ranges from 20 to 50%.<sup>4</sup> AMVT has a high rate of recurrence most commonly within the first 30 days after initial presentation.<sup>5</sup>

## **CLINICAL PRESENTATION**

- Patients typically present with acute onset of severe abdominal pain that is often not well localised.
- Duration of the pain before presentation is usually short (<12 hours).
- Other symptoms include nausea, vomiting and diarrhoea.
- The classical physical finding is minimal abdominal tenderness despite severe abdominal pain (pain out of proportion to physical findings).
- Bowel sounds may be present.
- Blood in the rectum is found in only 25%.
- Peritonitis occurs with bowel infarction and is a late and ominous finding.

Two thirds of sufferers of mesenteric arterial occlusive disease are female. Risk factors include known coronary disorders that cause cardiac thrombus formation (atrial fibrillation, valvular disease, ventricular aneurysms, dilated cardiomyopathy). If there is a history of chronic symptoms of intestinal angina, consider atherosclerotic mesenteric occlusive disease.

Laboratory tests are (at best) only supportive of the diagnosis rather than conclusive. Leukocytosis is present in 90% of patients. Elevated serum lactate and acidosis are not a consistent finding and, when present, tend to reflect established infarction and therefore portend a poor prognosis.

<sup>&</sup>lt;sup>4</sup> Rhee RY+ : Mesenteric venous thrombosis: still a lethal disease in the 1990s. J Vasc Surg 1994; 20: 688-97.

<sup>&</sup>lt;sup>5</sup> Kumar S+: Mesenteric Venous Thrombosis. *N Engl J Med* 2001; 345: 1682-8.



#### IMAGING

- Plain abdominal films are usually non-specific but may show a pattern of ileus in approximately 60% of patients. Blunt, semiopaque indentations of the bowel lumen (*thumbprinting*) are indicative of mucosal oedema, whereas gas in the wall of the bowel (*pneumatosis intestinalis*) or in the portal vein is characteristic of bowel infarction (although these findings are uncommon).
- Computed Tomography (CT) is often the most useful, readily available means of diagnosing AMI but may not always provide definitive information. Findings on CT include a dilated, thickened bowel wall, engorgement of the mesenteric veins, gas in the bowel wall or portal vein, non-filling of the mesenteric arteries, calcification of the SMA or celiac plexus. In cases of venous thrombosis, a dense venous wall and the thrombus evident as a central opacity in the vein are the most specific findings. Suggestive findings on CT should prompt consideration of mesenteric angiography with a view to thrombolysis if the patient has not got frank peritonitis. The efficacy of MRI imaging is similar to CT.
- The most definitive examination is mesenteric angiography which defines anatomical lesions and may have therapeutic potential by facilitating selective cannulation and thrombolysis of the affected vessel. Angiography can usually distinguish acute thrombosis, embolism or non-occlusive mesenteric ischaemia. Venous phase films may be required to prove venous thrombosis.



Acute Mesenteric Ischaemia

#### MANAGEMENT

*Mesenteric arterial occlusion:* Prompt surgical intervention is the best approach as irreversible bowel ischaemia occurs 6-8 hours after mesenteric arterial occlusion.<sup>6,7</sup>

- Acute occlusive mesenteric ischaemia is an indication to operate in most instances for either an embolus or a thrombus.
- Prior to surgery the long saphenous vein should be assessed and marked for possible harvesting in the event of reconstruction being required.
- Embolectomy is the preferred treatment for mesenteric emboli.
- Thrombotic disease requires either endovascular revascularisation or open bypass grafting of the occluded vessels.
- Intestinal viability at operation may be assessed using clinical criteria (colour, contractions, and mesenteric pulsation) or Doppler probe assessment for arterial signals on the anti-mesenteric border of the intestine. Non-viable bowel should be resected. A previously equivocal bowel segment may improve following revascularisation and be preserved. However, it is often wise to exteriorise rather than to anastomose the remaining bowel ends.

Non-occlusive disease is treated with optimal supportive measures and removal of vasopressor agents. Anticoagulation and/or direct infusion of papaverin into the SMA following angiography for 24 hours may be useful. Laparotomy is however required for peritoneal signs indicating bowel infarction or perforation.

*Mesenteric venous thrombosis:* Patients require general supportive measures and immediate, full anti-coagulation with heparin with a low threshold to performing laparotomy if bowel gangrene is suspected to have supervened.<sup>8</sup>

<sup>&</sup>lt;sup>6</sup> RJ Fowl. Acute Mesenteric Ischaemia. In Mayo Clinic Gastrointestinal Surgery. First Edition. Edited by KA Kelly, MG Sarr, RA Hinder. Philadelphia, WB Saunders Company 2005, pp 441-6.

<sup>&</sup>lt;sup>7</sup> Char DJ+: Surgical intervention for acute intestinal ischaemia: experience in a community teaching hospital. J Vasc Endovascular Surg 2003; 37: 245-52.

<sup>&</sup>lt;sup>8</sup> P Gloviczki: Acute Mesenteric Venous Thrombosis. *In* Mayo Clinic Gastrointestinal Surgery. First Edition. Edited by KA Kelly, MG Sarr, RA Hinder. Philadelphia, WB Saunders Company 2005, pp 447-55.



- Blood samples to screen for hereditary or acquired thrombophilia states such as protein C and S deficiencies, factor V Leiden and other mutations, hyperhomocysteinemia, and paroxysmal nocturnal haemoglobinuria should be sent just prior to commencing anticoagulation.
- A few reports have noted successful thrombolytic therapy through a catheter in the superior mesenteric artery or in the portal or mesenteric veins through the liver although complications rates (mainly gastrointestinal bleeding) are high. This approach is contraindicated in patients with peritonitis or GI bleeding.
- Patients with peritonitis require emergency surgery. Anticoagulants should be initiated as soon as the diagnosis of mesenteric venous thrombosis is made or confirmed intra-operatively. Subsequent management is dictated by the surgical findings, which range from a segmental infarction of small bowel to necrosis of the entire bowel, with or without perforation. The aim of resection is to conserve as much bowel as possible. On rare occasions, thrombectomy can be accomplished successfully when the thrombus is recent and is restricted to the superior mesenteric vein.

*Second look laparotomy* – follow-up laparotomy at 24 hours is often proposed as a way of ascertaining the viability of the remaining bowel after either arterial or venous occlusive disease. Deliberately deciding on such a strategy during the first operation may limit the extent of bowel initially resected.



Acute Mesenteric Ischaemia

# Table 1: Comparison of acute mesenteric venous thrombosis and acute mesenteric arterial thromboembolism (modified from Kumar et al<sup>5</sup>)

Variable	Venous Thrombosis	Arterial Thromboembolism	
Risk factors	Prothrombotic states Inflammatory bowel disease Abdominal cancer	Atherosclerotic vascular disease Valvular heart disease Arrhythmias	
Abdominal pain	Insidious onset	Sudden onset with embolic disease	
Tests			
Plain films	Usually non-specific	Usually non-specific	
Computed tomography	Sensitivity of more than 90%	Sensitivity of approximately 60%	
Mesenteric angiography	Not usually required for diagnosis	Often helpful	
Involvement of inferior mesenteric vessels	Uncommon	Common	
Operative findings			
Mesenteric arterial pulsations	Preserved except late in disease	Absent	
Transition from ischaemic to normal bowel	Gradual	Abrupt	
Тһегару			
Thrombolysis	Rarely useful	Often useful	
Long-term anticoagulation	Indicated	Indicated	
Sequelae	Short bowel, varices	Short bowel	

# 11. Damage Control Surgery for Non-traumatic Conditions

#### Gavin O'Brien

#### INTRODUCTION

The term Damage Control Surgery (DCS) refers to an operative strategy in which priority is given to salvaging a deteriorating patient rather than attempting to definitively address all problems at a single operation. An initial abbreviated laparotomy with subsequent stabilization of the patient in the ICU is followed by a second more definitive operation once the patient's condition has been optimised. The concept initiated with the practice of peri-hepatic packing with planned re-operation for hepatic haemorrhage. DCS is now a well established practice in the high-volume trauma centers of the USA and South Africa where it has been shown to reduce mortality rates significantly when applied to injuries previously categorised as "not survivable".<sup>1,2</sup>

#### RATIONALE

The concurrence of acidosis, coagulopathy and hypothermia in a patient undergoing laparotomy portends a poor outcome and should be taken as a prompt to consider implementing DCS techniques (*see Table 1*). Hypothermia is associated with sympathetic  $\beta$ -adrenergic overdrive, peripheral vasoconstriction and end-organ hypoperfusion, resulting in conversion from aerobic to anaerobic metabolism and metabolic acidosis. Aggressive fluid resuscitation predisposes to impaired coagulation, and compounds hyperchloraemic acidosis. Whilst a physiological rationale for the adoption of DCS in the trauma setting is clear<sup>3</sup>, critically ill, general-surgical patients with intra-abdominal sepsis or haemorrhage are equally susceptible to the vicious cycle of acidosis, hypothermia and coagulopathy, and may therefore also benefit from early recourse to ICU for optimization of physiological parameters prior to further address of non-life-threatening problems.<sup>4</sup>

<sup>&</sup>lt;sup>1</sup> Moore EE+: Staged physiologic restoration and damage control surgery. *World J Surg* 1998; 22: 1184-191.

<sup>&</sup>lt;sup>2</sup> Loveland JA+: Damage control in the abdomen and beyond. *Br J Surg* 2004; 91: 1095-101.

<sup>&</sup>lt;sup>3</sup> Rotondo MF+: The damage control sequence and underlying logic. *Surg Clin North Am* 1997; 77: 761-77.

<sup>&</sup>lt;sup>4</sup> Finlay IG+: Damage control laparotomy. *Br J Surg* 2004; 91: 83-5.



## **CLINICAL APPLICABILITY IN NON-TRAUMATIC SETTINGS**

It is important to recognize that DCS is indicated in only highly selected situations (*see Table 2 for examples*). Although Level I evidence is lacking, the intuitive benefits of DCS are clear especially, perhaps, in patients who are preoperatively immunosuppressed or have with severe co-morbidity (particularly if the ominous pathophysiological triad is already present preoperatively). Potential benefiters of DCS in the non-trauma setting include those with:

- Severe intraperitoneal sepsis with gross faecal contamination in a patient with high level comorbidity.
- Extensive mesenteric infarction.
- Massive retroperitoneal bleeding.

#### COMPLICATIONS

The most common complication that results directly from a DCS technique is abdominal compartment syndrome (ACS), with an incidence approaching 15%. A fine line exists between sufficient packing to achieve tamponade and increasing the risk of ACS as a result. Renal failure occurs when intraabdominal pressure (measured intravesically by a transducer attached to a urinary catheter) exceeds 20 - 25 mmHg – therefore urgent decompression may be required in patients whose pressures approach or exceed this threshold. Closed loop obstruction after multiple bowel resections may also mandate an emergency 'relook' procedure. Timing of the second laparotomy is generally at the surgeon's discretion but should be as soon as possible after physiological variables have been restored.



Damage Control Surgery for Non-traumatic Conditions

#### Table 1: Some examples of Damage Control Surgical techniques

Techniques of Damage Control Surgery		
Haemorrhage	Bleeding is controlled as rapidly as possible (i.e. splenectomy rather than splenorrhaphy; nephrectomy rather than renorrhaphy) and takes precedence over examining for other injuries. Areas of oozing (e.g. liver, mesentery, pelvis, retroperitoneum) are packed.	
Intestinal injuries	Small holes are "whip stitched" without any attempt at definitive repair. Large defects/devitalized areas: Resect the affected area with GIA stapler. No attempt is made at re-anastomosis or stoma creation.	
Closure	Temporary closure techniques: towel clips, running nylon suture, 'Opsite sandwich'.	
Postoperative care	Monitor intra-abdominal pressure for compartment syndrome.	

# Table 2: Clinical scenarios where damage control surgery may be considered in a non-trauma setting

#### Clinical indications for considering Damage Control Surgery:

Inability to achieve haemostasis owing to coagulopathy.

Definitive operation is complex & time-consuming (usually >90 mins) in a critically ill patient.

Inaccessible major venous injury.

Associated life-threatening injury in a second anatomical location.

Planned reassessment of abdominal contents.

Inability to approximate abdominal layer of rectus sheath owing to visceral oedema or abdominal compartment syndrome.



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## 12. Priorities in the Management of Acute Limb Ischaemia

#### Joseph F Dowdall

Acute limb ischaemia (ALI) is defined by the TransAtlantic Inter-Society Consensus (TASC) document as 'any sudden decrease or worsening in limb perfusion causing a potential threat to extremity viability'.

#### INTRODUCTION

ALI is one of the most challenging problems encountered by general and vascular surgeons. The process can develop insidiously in the presence of established collateral channels (which may lead to worsening of claudication or new rest pain) or rapidly (such as in the case of embolisation or graft thrombosis) with severe symptoms developing precipitously.

There are 2 distinct aetiological categories of ALI: thrombosis and embolism. Distinction can sometimes be difficult but most series suggest that thrombosis outnumbers embolism by approximately 6:1. More important than the causation of the ischaemia, however, is determination of its severity.

Reporting standards for lower extremity ischaemia as recommended by the joint council of the Society for Vascular Surgery (SVS) and the North American Chapter of the International Society for Cardiovascular Surgery (ISCVS):

- I Viable: not immediately threatened. No sensory loss or muscle weakness. Arterial doppler signal audible.
- IIa Marginally threatened: salvageable if promptly treated. Minimal sensory loss, no muscle weakness. Arterial Doppler signals often inaudible.
- IIb Immediately threatened: salvageable with immediate revascularisation. Sensory loss associated with rest pain in more than the toes, mild to moderate muscle weakness. Arterial Doppler signals usually inaudible.
- III Irreversible: major tissue loss or permanent nerve damage inevitable. Profound limb anaesthesia and paralysis. Arterial and venous Doppler signals inaudible.



#### **DIAGNOSIS/INVESTIGATION**

#### Diagnosis

The diagnosis of acute limb ischaemia is primarily clinical. The six Ps of acute limb ischaemia may be simplistic but they help focus clinical examination. It is vital to compare the limb with its opposite extremity as this may give clues as to causation. It is important to note that the presence of doppler signals does not exclude acute limb ischaemia.

Duplex ultrasonography can be used to localise the site of an occlusion and may be particularly useful for accessing thrombosed grafts where the origin is obscure. Furthermore, where a prominent popliteal pulse is present on the opposite side, duplex scanning is useful to confirm or exclude a thrombosed popliteal aneurysm. While contrast arteriography (using the contralateral limb for access) is the gold standard, it may be forgone in patients with a classical presentation of femoral embolus or where the limb is acutely threatened. In appropriate circumstances, angiography may facilitate the insertion of a catheter for thrombolysis. It is important, however, that any imaging done does not involve a lengthy stay in the radiology department without any treatment. With increasing endovascular skills among vascular surgeons, angiography is often performed in the endovascular suite or theatre and followed immediately by therapeutic intervention.

In general patients with acutely ischaemic limbs should be transferred to a vascular surgical unit for investigation and treatment. Delaying transfer for investigation is pointless and only increases the risk of irreversible ischaemia. The situation is especially urgent when motor and/or sensory loss is present.

#### Treatment

Regardless of the planned treatment modality, the patient should undergo systemic anticoagulation with unfractionated heparin to prevent proximal and distal propagation of the clot (provided spinal or epidural anaesthesia is not planned). Physiological monitoring, correction of haemodynamic abnormalities and arrhythmias with replenishment of circulating volume and establishment of adequate urine output are essential.



As a priority, one must determine whether ischaemia is severe enough to warrant immediate operation without angiography – this is only rarely the case.

After reviewing the angiogram, the next decision is whether an operative or thrombolytic approach is appropriate. If thrombolysis is chosen and found successful, it must be followed by endovascular or open correction of defects unmasked by thrombolysis.

#### **Endovascular Options**

*Thrombolysis:* Thrombolytic therapy for acutely ischaemic limbs is given via the intra-arterial route through a variety of catheter systems. There is an appreciable minor and major complication rate (20% and 5% respectively) associated with this treatment and should therefore only be carried out in an environment where experienced medical and nursing staff are available.<sup>1</sup> There are 3 major randomised trials comparing thrombolysis and surgery for the treatment of acute limb ischaemia. In terms of their primary endpoints, one favoured surgery (STILE trial), one favoured thrombolysis (Rochester trial) and one showed no difference (TOPAS trial).

The study of surgery or thrombolysis for the ischaemic lower extremity (STILE trial<sup>2</sup>) evaluated t-PA and urokinase comparing them to primary operation for lower extremity ischaemic symptoms of less than 6 months duration. The trial was prematurely stopped because of poorer results in the thrombolysis group. A post-hoc analysis was performed to look at patients with recent symptom onset (<14 days). In this group there were fewer amputations in the thrombolysis arm. The thrombolysis or peripheral arterial surgery (TOPAS<sup>3</sup>) trial randomised patients with limb ischaemia of less than 14 days duration to thrombolysis or surgery – there were no differences in the primary endpoint of amputation free survival. The Rochester trial<sup>4</sup> showed similar limb salvage rates but a survival advantage for thrombolysis at 12 months.

<sup>&</sup>lt;sup>1</sup> Working party on thrombolysis in the management of limb ischaemia. Thrombolysis in the management of lower limb peripheral arterial occlusion. *JVIR* 2003; 7: S337-49.

<sup>&</sup>lt;sup>2</sup> The STILE Investigators. Results of a prospective randomised trial evaluation surgery versus thrombolysis for ischaemia of the lower extremity. Ann Surg 1994; 220: 251-68.

<sup>&</sup>lt;sup>3</sup> Ouriel K+: For the TOPAS Investigators. Thrombolysis or peripheral arterial surgery. J Vasc Surg 1996; 23: 64-73.

<sup>&</sup>lt;sup>4</sup> Ouriel K+: A comparison of thrombolytic therapy and operative revascularisation in the initial treatment of acute peripheral arterial ischaemia. J Vasc Surg 1994; 19: 1021-30.



Priorities in the Management of Acute Limb Ischaemi

*Percutaneous aspiration thrombectomy (PAT)* is a technique which uses thin walled, large catheters and suction with a 50 ml syringe to remove embolus or thrombus from native arteries, grafts or run-off vessels.

*Percutaneous mechanical thrombectomy (PMT)* – These devices operate on the basis of 'hydrodynamic recirculation' i.e. dissolution of the thrombus occurs within an area of continuous mixing referred to as the 'hydrodynamic vortex'. This selectively traps, dissolves and evacuates the thrombus. They generally work better for fresh than for old thrombus.

Trials comparing PAT, PMT, thrombolysis and surgery are necessary to determine their respective roles.

#### **Surgical Options**

TASC has recommended that surgery should be preferentially performed for patients with immediately limb threatening ischaemia (class IIb and early class III) or proximal (above the inguinal ligament) emboli.<sup>5</sup>

*Thromboembolectomy* – Catheter embolectomy is preferred for emboli in a non-atherosclerotic limb. When no further emboli can be retrieved, some form of completion imaging (usually angiography) should be performed. In up to one third of patients, there is residual thrombus in run-off vessels which may lead to failure.

Once thrombus is removed from an artery, an underlying lesion must be sought and surgically corrected.

*Intraoperative thrombolysis* – Distal clot that cannot be retrieved with an embolectomy catheter may be treated with thrombolytic agents delivered intra-arterially followed by irrigation or further passes of the catheter.

Fasciotomy should be performed if acute severe ischaemia has been prolonged or if signs of increased compartment pressures develop. There is no easy and accurate way to predict need for fasciotomy but in general the threshold for performing it should be low (*"If you think of it, do it!"*). Careful observation post-operatively is necessary to detect compartment syndrome in patients at risk.

<sup>&</sup>lt;sup>5</sup> TransAtlantic Inter-Society Consensus (TASC). Management of peripheral arterial disease (PAD). *Eur J Vasc Surg* 2000; 19: S115-43.



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Priorities in the Management of Acute Limb Ischaemia

*Amputation* – Primary amputation is preferred when the patient has sustained prolonged, irreversible limb ischaemia or, as judged by a senior clinician, attempting limb revascularisation would threaten the patient's life.



# 13. Perioperative Management of Diabetic Patient

#### Fiachra J Cooke

#### INTRODUCTION

Anaesthesia and surgery cause a stereotypical metabolic stress response that can overwhelm homeostatic mechanisms in patients with pre-existing abnormalities of glucose metabolism. The typical features of the metabolic stress response include release of the catabolic hormones (including adrenaline, noradrenaline, cortisol, glucagons, and growth hormone) and inhibition of insulin secretion and action.

An emphasis on prospective, rather than retrospective, insulin adjustment is now the goal of perioperative management of the patients with diabetes mellitus.

The treatment goals of the perioperative management of diabetic patients are the avoidance of:

- Hypoglycemia
- Excessive hyperglycemia
- Electrolyte disturbance
- Protein catabolism

while allowing for the nutritional support of patients appropriate to the circumstances.

Fasting causes particular problems in type 1 diabetes. Such patients need basal insulin to prevent ketosis and, therefore, tend to develop hypoglycaemia without additional carbohydrate intake.

Fasting is of little significance in type 2 diabetes, unless the patient has received oral hypoglycaemic agents.  $^{\rm 1}$ 

Eldridge AJ+: Peri-operative management of diabetic patients. Any changes for the better since 1985? *Anaesthesia* 1996; 51: 45-51.



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Target blood glucose during surgery should be 7–11 mmol/L (at normal levels, patients are too close to hypoglycaemia). At levels >11 mmol/L, urine output increases and dehydration may ensue.

Intravenous infusion of insulin, glucose, and potassium is now standard therapy and has replaced subcutaneous insulin therapy for the perioperative management of diabetes, especially in type 1 diabetic patients and patients with type 2 diabetes undergoing major procedures. Several reports have emphasized the advantages of the insulin infusion regimen over subcutaneous delivery.<sup>2:3,4</sup> Sliding scales however still remain the method of choice in many hospitals due to the unavailability of syringe drivers and lack of an agreed protocol with nursing staff.

#### MANAGEMENT

The perioperative management of diabetic patients begins with a preoperative assessment, including a complete history and physical examination. Even in patients who deny a history of diabetes mellitus, the history and physical examination should include an assessment for possible undiagnosed disease. The history should also include the type and treatment of diabetes or insulin resistance, known complications, and previous hospitalisations. Note the course and complications, if any, of prior surgeries. Questioning should also aim to elicit symptoms of ischaemic cardiac, renal, and/or peripheral vascular disease, if any.

Patients may then be commenced on either separate glucose and insulin infusions or on a combined Glucose (with potassium)/Insulin infusion (*see Table 1*). These recommendations may need adjusting depending on the age, weight and condition of the patient; generally, the more ill the patient, the more insulin needed. The suggested dextrose infusion rates are those required to control blood glucose. During prolonged fasts following major surgery, saline and other fluids may be required as agreed between the anaesthetic and surgical teams involved.

<sup>&</sup>lt;sup>2</sup> Christiansen CL+: Insulin treatment of the insulin-dependent diabetic patient undergoing minor surgery. Continuous intravenous infusion compared with subcutaneous administration. *Anaesthesia* 1988; 43: 533-37.

<sup>&</sup>lt;sup>3</sup> Alberti KG+: Insulin delivery during surgery in the diabetic patient. *Diabetes Care* 1982; 5 Suppl 1: 65-77.

<sup>&</sup>lt;sup>4</sup> Hirsch IB+: Perioperative management of surgical patients with diabetes mellitus. *Anesthesiology* 1991; 74: 346-59.
### Separate glucose + insulin infusions are suitable for:

- All emergency operations in patients with either type 1 or 2 diabetes.
- Moderate or major surgery in type 1 diabetes.
- Major surgery in type 2 diabetes.

The glucose infusion comprises 500mls of 10% dextrose with 10mmol potassium chloride given at a rate of 100 ml/hour. The infusion rate must be controlled using a pump (*see Table 2*). It is hazardous to attempt to control the rate by eye. The insulin infusion comprises soluble insulin 50 units in 50 ml 0.9% saline (at a concentration of 1 unit/ml), delivered in a syringe driver. Capillary blood must be measured accurately every hour and the insulin pump rate adjusted accordingly.

These patients should not be first on the theatre list – they should be placed towards the end or in the afternoon, to allow a few hours for the regimen to shift blood glucose levels into the desirable range (7–11 mmol/litre).

### A combined GKI infusion is suitable for:

- Moderate or major surgery in type 1 diabetes.
- All major surgery in type 2 diabetes.

The infusion of 500ml 10% dextrose with 10 mmol KCl and 10 units Actrapid insulin, is given at a rate of 100 ml/hour, using a rate controller. Capillary blood glucose is measured precisely every hour. If blood glucose falls to 4–7 mmol/litre in two successive hours, less insulin is needed (the solution should be changed to 5 units/500ml). If blood glucose rises to 17 mmol/L or more in two successive hours, more insulin is needed (the solution should be changed to 20 units of insulin/500ml). Plasma potassium must be checked before starting the regimen, immediately postoperatively, and at least daily during infusion.



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### **EMERGENCY SURGERY**

Up to 5% of diabetic patients require emergency surgery at some time, usually because of infection. Danger results from the loss of control of diabetes and the onset of ketoacidosis provoked by the surgical illness.

There may especially be confusion if diabetic ketoacidosis mimics an acute abdomen in its presentation. Glycaemic control and acid–base balance should be thoroughly evaluated by blood glucose, electrolytes, blood gases and urinary ketones. Intravenous fluids should be given, with potassium and insulin as indicated. In up to 60% of patients with diabetic ketoacidosis, abdominal symptoms resolve following control of the diabetes and fluid therapy, avoiding the need for laparotomy.

However, there is a further pitfall for the unwary – autonomic or sensory neuropathy may mask the symptoms and signs in a patient with diabetes with a true acute abdomen needing urgent laparotomy. The incidence of gallstone and gallbladder related complications in diabetics, is significantly increased in the diabetic population.<sup>5</sup>

Regimens for surgery in diabetes				
	Type of surgery			
Type of diabetes	Minor	Moderate	Major	Emergency
Type 1 (always treated with insulin – ketosis-prone)	Fast-and- check	Glucose– insulin or GKI	Glucose– insulin or GKI	Glucose– insulin
Type 2 (diet/tablets or insulin – not prone to ketosis)	Fast-and- check	Fast-and- check	Glucose– insulin or GKI	Glucose– insulin

#### Table 1: Preoperative regimens and protocols<sup>6</sup>

<sup>&</sup>lt;sup>5</sup> Cucchiaro G+: Deaths from gallstones. Incidence and associated clinical factors. *Ann Surg* 1989; 209: 149-51.

<sup>&</sup>lt;sup>6</sup> SR Heller+: Surgery in Patients with Diabetes. *Medicine* 2002; 30: 66-8.

## Perioperative Management of Diabetic Patient

### Table 2: Rate adjustment for pump-controlled glucose-insulin infusions

Infusion rate adjustments in the glucose-insulin regimen			
Blood glucose (mmol/litre)	Adjustment in pump rate (ml/hour)	Comments	
<4	Stop	Check glucose infusion is running, re-check in 30 minutes; if still low, call doctor	
4-6.9	+ 1	Two successive readings require a reduction in insulin infusion rate by 1 ml/hour	
7–10.9	+ 2	_	
11–16.9	+ 3	Two successive readings require an increase in insulin infusion rate by 1 ml/hour	
>17	+ 4	Re-check in 1 hour; if still high, check insulin is running, call doctor	



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# 14. Interruption of Long-term Anticoagulation

### Ronan A. Cahill

- Interruption of chronic antithrombotic therapy puts the patient at the risk of thromboembolism which pertains without such treatment (see Table 1).
- Therefore the risk and consequences of excessive bleeding if the therapy is continued perioperatively have to be balanced against the cardiovascular risks of interrupting the antithrombotic medication for a period of time.
- If held, anti-thrombotic agents should be re-administered as soon as possible after surgery.
- Only patients at very high risk of thromboembolism require treatment with heparin as late as possible before their operation with re-commencement as soon as possible afterwards.

### MANAGEMENT OF LONG-TERM WARFARIN (SEE TABLE 2)1,2

For patients taking warfarin for previous thromboembolism, the time since the event and the presence of other risk factors for venous thromboembolism (VTE) determines their overall preoperative risk. Surgery should be deferred for at least one month following a thromboembolic event. If this is not possible, preoperative heparin should be used. Patients with a VTE within the previous two weeks should be considered for insertion of a vena caval filter. Preoperative heparin should also be considered for those who have suffered a venous occlusion within the previous three months and if additional risk factors are present (e.g. immobility or malignancy). Additionally, adjuvant heparin is warranted during the interruption of warfarin in those with mechanical heart valves, especially when other risk factors for thromboembolism (such as mitral or combined valves or left ventricular dysfunction) are present.

While surgical intervention confers additional risks of VTE (due to immobility, venous stasis, intraoperative compression of vessels etc.), it does not contribute to the risk of arterial thromboembolism (ATE). This means that while only patients within one month of an ATE should be heparinised postoperatively, patients within three months of a VTE should also receive this treatment.

<sup>2</sup> Kearon C+: Management of anticoagulation before and after elective surgery. N Eng J Med 1997; 336: 1506-11.

<sup>&</sup>lt;sup>1</sup> Kearon C. Perioperative management of long-term anticoagulation. Semin Throm Hemost 1998; 24 (suppl 1): 77-83.



Interruption of Long-term Anticoagulation

Patients on warfarin for uncomplicated atrial fibrillation do not require heparinisation either before or after operation.

## MANAGEMENT OF LONG-TERM ANTI-PLATELET AGENTS

Experience with aspirin, the prototypical antiplatelet agent, may be used to guide the preoperative management of drugs that have an irreversible antiplatelet effect. Pre-operative aspirin usage has been linked to greater than usual perioperative haemorrhage in patients undergoing general surgical, orthopaedic, urological and anaesthetic procedures. However, not all clinical studies demonstrate a significant association with a clinically important increase in blood loss. Many clinicians do not operate electively on patients while they are taking aspirin, preferring, instead, to recommend withdrawal of therapy for a period of time prior to intervention to allow normal haemostatic capabilities recover. Advice about the length of time aspirin should be withheld varies but, probably, five days is sufficient.<sup>3</sup> However, as the overall anti-haemostatic effect of aspirin on a population is thought to be mild, although variable, recent aspirin usage is not a reason to delay essential or emergent surgery.

The duration of effect of clopidrogel (*Plavix*) is uncertain although as this medication also exerts an irreversible effect on platelet function, its duration of effect is likely to be similar to aspirin.

Although other nonsteroidal agents have a similar effect on cyclo-oxygenase, their inhibitory action is reversible and is therefore short-lived (usually about six hours).

<sup>&</sup>lt;sup>3</sup> Cahill RA+: Duration of increased bleeding tendency after cessation of aspirin therapy. J Am Coll Surg 2005; 200: 564-73.



# Table 1: Risk of thromboembolism by underlying predisposition plus benefit of anticoagulation

Risks of Thromboembolism with and without Chronic Warfarin Therapy			
Indication	Rate without and with Warfarin		Risk Reduction with Therapy
After venous thromboembolism			
First Month.	40%	8%	80%
Months 2 and 3	10%	2%	80%
Previous recurrent venous			
thromboembolism	15%	3%	80%
Nonvalvular atrial fibrillation	4.5%	1.5%	66%
Nonvalvular atrial fibrillation			
and previous embolism	12%	4%	6 %
After acute arterial embolism			
Annual risk of recurrence	8%	2%	75%
Within first month	15%		
Mechanical heart valve	15%	5%	66%

#### Interruption of Long-term Anticoagulation

# Table 2: Perioperative management strategies for patients on long-term anticoagulation

Type and Recency of Thromboembolism		Perioperative Management	
Arterial	Within the previous 1 month	Intravenous heparin pre- and postop	
	More than 1 month previous	No intravenous heparin required	
Venous	Within the previous 2 weeks	Delay surgery or insert caval filter if major op, iv heparin if minor	
	2-4 weeks previously	Intravenous heparin pre- and postop	
	1-3 months previously	Intravenous heparin postop only	
	>3 months previously	No intravenous heparin required	



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# 15. Optimising the Atherosclerotic Patient for Surgery

### **Steven M Kieran**

### INTRODUCTION

Myocardial infarction is a significant cause of perioperative morbidity and death with an incidence of 0.7% in males over 50 years of age who are undergoing major surgery and 3.1% in patients undergoing major vascular surgery. Although most research into reducing these risks has focused on patients undergoing vascular surgery, the principles remain the same for all patients. Preoperative assessment and identification of patients and procedures at risk, targeted investigations and certain pharmacologic pre-treatment (in particular perioperative cardioselective  $\beta$ -blockade) should permit safer surgery.

### CARDIAC RISK ASSESSMENT

Traditional cardiac risk factors (hypertension, hypercholesterolaemia, smoking, diabetes mellitus and family history) should be sought in the history and clinical examination and modified as early as possible prior to surgery. While certain operations have different operative risk of adverse cardiac events (*see Table 1*), patient specific variables are important for risk stratification (*see Table 2*). Lee et al identified six clinical conditions associated with increased risk of a significant adverse cardiovascular event after non-cardiac surgery (the risk of which is 0.5%, 1.3% 4% and 9.1% in patients with 0, 1, 2 or 3 of these conditions respectively).<sup>1</sup> Six risk factors predictive of adverse perioperative cardiac events after vascular surgery have also been elucidated using multivariate analysis by Eagle et al<sup>2</sup> (adverse cardiac events occur in 3.1%, 15.5% and 50% in patients with 0, 1, or 2, and 3 or more risk factors respectively). Ischaemia occurring in the perioperative period, however, is the single most important risk factor for myocardial infarction.

<sup>&</sup>lt;sup>1</sup> Lee TH+: Derivation and prospective validation of a simple index for the prediction of the cardiac risk of major noncardiac surgery. *Circulation* 1999; 100: 1043-9.

<sup>&</sup>lt;sup>2</sup> Eagle KA+: Combining clinical and thalium data optimizes preoperative assessment of cardiac risk before major vascular surgery. Ann Intern Med 1989; 110: 859-66.



### **CARDIAC INVESTIGATIONS**

All patients with risk factors and those undergoing intermediate or high risk procedures should have an electrocardiogram (ECG) in order to demonstrate arrhythmias and signs of previous ischaemia or left heart strain. A chest x-ray should also be routinely performed looking for evidence of congestive cardiac failure. Further additional non-invasive coronary artery disease testing is recommended in patients at high risk of coronary artery disease although the actual threshold varies between authors.

Exercise ECG testing can detect myocardial ischaemia with a sensitivity of 88% and specificity of 66%. However limitations exist - e.g. the patient must be ambulatory (sensitivity drops if the patient is unable to perform maximum exercise). Furthermore the specificity is poor if a resting ECG shows ST segment of T wave changes or if the patient is on digoxin. Dipyridamole Thalium stress testing (despite initial favourable data) in now felt to be of limited clinical value. In moderate risk patients with 2 or more reversible defects the sensitivity is only 11% and specificity 90%.<sup>3</sup> Dobutamine stress echocardiography is a promising investigation as the development of new wall motion abnormalities following the administration of dobutamine is indicative of significant CAD. However these reports are early and its use is not yet widespread. The indications for the use of coronary angiography in pre-operative assessment are controversial. Angiography should probably be performed in patients with overt symptoms, and in those patients with positive exercise stress tests and multiple risk factors. However the exact role of coronary angiography in asymptomatic patients is unclear and should be tailored to the individual patient.

Management strategies aiming to reduce perioperative cardiac morbidity and mortality utilise either the administration of medications or procedures aimed at revascularising the coronary arteries.

### **RISK REDUCTION – MEDICAL THERAPIES**

The effect of various medications including  $\alpha 2$  antagonists, nitrates and calcium channel blockers have been studied with little benefit identified overall. However, peri-operative cardioselective  $\beta$ -blockade has emerged as the

<sup>&</sup>lt;sup>3</sup> de Virgillo C+: Dipyridamole-thalium/sestamibi before vascular surgery: a prospective blinded study in moderate risk patients. *J Vasc Surg* 2000; 32: 77-89.



most effective intervention to date. Poldermans et al have demonstrated a 91% drop in the perioperative risk of myocardial infarction or death from cardiac causes in high risk patients undergoing vascular procedures.<sup>4</sup> These findings have been confirmed by other prospective randomised control trials and are now standard practice in vascular surgery units.  $\beta$ -blockade is commenced a number of days prior to surgery, allowing dose titration to achieve a resting heart rate of <60 beats per minute, and is likely to be of value in all patients with risk factors undergoing major surgery or vascular surgery. A combination of  $\beta$ -blockade and statins has also been shown to independently reduce myocardial infarction and mortality in high risk patients undergoing abdominal aortic aneurysm repair.<sup>5</sup> A recent meta-analysis concluded that the evidence overall for the use of  $\beta$ -blockade in noncardiac surgery (including non-vascular procedures) is encouraging but not yet definitive.<sup>6</sup>

### **RISK REDUCTION – REVASCULARISATION**

Coronary revascularisation can be performed by percutaneous angioplasty or traditional coronary artery-bypass grafting. Patients who undergo preoperative percutaenous transluminal coronary angioplasty (PTCA) have been shown to have fewer cardiac complications peri-operatively.<sup>7</sup> However, the greatest benefit appears to be in those patients who have had the procedure performed more than three months prior to their surgical intervention. Ultimately, PTCA has a role in coronary revascularisation in the pre-operative period, however its use should be tailored to the individual patient as specific indications are not yet clear. The use of pre-operative coronary artery bypass grafting (CABG) has been shown to decrease perioperative mortality by almost 50%.<sup>8</sup>

<sup>&</sup>lt;sup>4</sup> Poldermans D+: The effect of bisoprolol on perioperative mortality and myocardial infarction in high-risk patients undergoing vascular surgery. N Eng J Med 1999;34:1789-94.

<sup>&</sup>lt;sup>5</sup> Kertal MD+: A combination of statins and β-blockers is independently associated with a reduction in the incidence of Perioperative Mortality and Nonfatal Myocardial infarction in patients undergoing abdominal aortic aneurysm surgery. *Eur J Vasc Endovasc Surg* 2004; 28: 343-52.

 $<sup>^6</sup>$  Devereaux PJ+: How strong is the evidence for the use of perioperative  $\beta$ -blockers in non-cardiac surgery. BMJ 2005: 313-21.

<sup>&</sup>lt;sup>7</sup> Posner KL+: Adverse cardiac outcomes after noncardiac surgery in patients with prior percutaenous transluminal coronary angioplasty. *Anesth Analg* 1998; 89: 553-60.

<sup>&</sup>lt;sup>8</sup> Eagle KA+: Cardiac risk of noncardiac surgery: influence of coronary disease and type of surgery in 3368 operations. *Circulation* 1997; 96:1883-7.



**Optimising the Atherosclerotic Patient for Surger** 

### Table 1: Risk of cardiac complication by type of operative intervention

Cardiac risk classification of non-cardiac operations (adapted from Chassot et al <sup>9</sup> )			
Minor operations (Cardiac complication rate <1%)			
Endoscopic procedures Ambulatory surgery Plastic and reconstructive surgery	Breast and superficial procedures Eye Surgery		
Intermediate procedures (Cardiac complication rate 1-5%)			
Minor vascular surgery (including carotid endarterectomy) Abdominal and thoracic procedures Neurosurgery	ENT procedures Orthopaedic surgery Prostatectomy		
Major procedures (Cardiac complication rate >5%)			
Emergency intermediate procedures Aortic and major vascular surgery	Prolonged surgical procedures, large fluid or blood loss Unstable haemodynamic situations		

<sup>&</sup>lt;sup>9</sup> Chassot PG+: Preoperative evaluation of patients with, or at risk of, coronary artery disease undergoing noncardiac surgery. Br J Anaesth 2002; 89: 747-59.



	Non-cardiac Surgery (Lee et al)	Vascular Surgery (Eagle et al)
1	High risk type of surgery	Age >70 yrs
2	Ischaemic heart disease	History of angina
3	History of congestive cardiac failure	Previous MI or pathological Q waves on ECG
4	History of cerebrovascular accident	History of Congestive Cardiac failure
5	Diabetes Mellitus requiring insulin	History of ventricular ectopics requiring treatment
6	Preoperative serum creatinine >175 μmol/L	Diabetes Mellitus

### Table 2: Cardiac risk factors for patients undergoing major non-cardiac surgery



Care of Complex Surgical Patients Clinical Guideline

Notes



Care of Complex Surgical Patients Clinical Guidelines

Notes





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