COMPLICATIONS OF GASTROINTESTINAL ENDOSCOPY

Dr Jonathan Green

INTRODUCTION

Gastrointestinal (GI) endoscopy has now been part of conventional medical practice for over thirty years following the development of useable flexible fibreoptic endoscopes in the early 1970’s. Initially just used for diagnostic examination of the upper GI tract with biopsies, the technique was initially extended to the lower GI tract and then began the expansion of therapeutic techniques which continues to the present time.

Although using natural portals and not needing to cross tissue planes to gain access, this new technology was nevertheless invasive of the human body and so, like all invasive techniques, accompanied by attendant risks and complications. Sedation-related complications predominated in the early days but the expansion of therapeutic endoscopy dramatically widened the scope for complications. The potential benefits of therapeutic endoscopy need to be weighed against the potential to do harm. Two large audits of UK endoscopic practice (References 2 and 3 – Cardio-pulmonary and Sedation-related Complications) have shown a surprisingly high incidence of both morbidity and mortality following upper and lower GI diagnostic and therapeutic endoscopy. More recently, the National Confidential Enquiry into Post-operative deaths (NCEPOD) report into therapeutic GI endoscopy has also found further prima facie evidence of suboptimal endoscopic practice.

As these large audits show, many of the complications that occur are preventable. Some relatively simple precautions can be applied in most cases to bring about a dramatic reduction in the incidence of complications – as has been best illustrated with respect to reducing the complications associated with sedation (vide infra).

In order to apply risk reduction more widely, the Endoscopy Committee of the British Society of Gastroenterology in 2005 commissioned a group of experienced endoscopists under the chairmanship of Dr Jonathan Green to review the incidence of common endoscopy-related complications and to provide simple, practical and (where possible) evidence-based advice for the prevention, recognition and management of these complications and adverse events.

For ease of reference, complications are divided into five sections:-

1) Cardio-pulmonary and sedation-related complications
2) Complications specific to diagnostic and therapeutic upper gastro-intestinal (GI) endoscopy
3) Complications specific to diagnostic and therapeutic colonoscopy and flexible sigmoidoscopy.
4) Complications specific to endoscopic retrograde cholangiopancreatography (ERCP)
5) Complications of insertion of percutaneous endoscopic gastrostomies (PEG).

For each section, authors have structured their contributions to address the issues of which complications can occur and why, how to recognise them, the early management of the complications and sensible strategies to minimise them.

Although obvious, it must be clearly emphasized to every GI endoscopist that the best management of complications is to prevent their occurrence in the first place – and this must be the culture within which the service is delivered. The individual sections will clearly outline strategies for avoidance of complications for specific endoscopic techniques but there are some general principles applicable to all to prevent complications. These include:-

- Trained endoscopists performing within their level of competence and experience
- Adequate supervision of trainee endoscopists.
- Procedures performed in an appropriate setting – fully staffed with trained assistants, modern high quality equipment and access to other facilities (resuscitation, surgery or ITU) available in a timely manner
- A culture of team working and safety first throughout the Unit
- Clear and well publicised Unit policies for:-
  - Informed consent – together with a clear and prompt explanation to patients and relatives when a complication has occurred
  - Risk stratification in assessment of co-morbidities, clotting status etc
• Adherence to published national standards for safe sedation
• An agreed policy for antibiotic use
• Audit of complications with regular review meetings involving all endoscopists to give feedback on errors and, ideally, near misses.
• Regular reports to Clinical Governance committees with early warnings about foreseeable problems – e.g. failing equipment, inadequate staffing etc
• Institution of the Endoscopy Global Rating Scale (GRS) within the Unit
• Adherence to the BSG Quality and Safety Standards for GI Endoscopy

If all of the above can be part of the accepted culture within an endoscopy service, healthcare professionals at all levels will have already minimized the majority of risks associated with GI endoscopy.

Complications will always still occur despite the highest standards of practice. Risk is inherent in the procedures that we undertake. Our professional responsibility is therefore to completely prevent avoidable risks and to reduce unavoidable risks to an absolute minimum. The following sections are designed to help endoscopists achieve this.

EVIDENCE GRADING
Evidence, where available, is quoted in each section and is graded. The grading system adopted throughout is the clinically more appropriate ‘new’ grading scheme and hierarchy of evidence (Mason and Eccles, 2003; Fig 2) rather than the ‘old’ scheme (Eccles and Mason, 2001; Fig 1) which was specifically developed for evaluating therapeutic trials.
INTRODUCTION

Cardio-pulmonary complications account for about 50% of the potentially serious morbidity and approximately 50% of all the procedure-related deaths associated with GI Endoscopy. In many cases these complications are a direct or indirect consequence of elderly, frail or at-risk patients being given unnecessarily high doses of IV sedation [ref 1–3. Evidence Grade I].

Pre-Procedural Assessments

Before undertaking any GI endoscopic procedure, endoscopists should:-
• Obtain full and proper informed consent from the patient. The acronym EMBRACE can be used (see Fig 3) as an aide-mémoire [ref 4].
• Be familiar with the latest BSG Guidelines on Safety and Sedation [ref 5].
• Be aware of any relevant medical, surgical and drug history elicited in the pre-admission/admitting process.
• Consider whether formal anaesthetic help is needed or advisable. Certain patient’s endoscopic procedures are best carried out under a GA – see Fig 4. As yet, anaesthetists are not involved in setting sedation standards for endoscopy but this may change in future [ref 6].

Remember – Should complications occur, then the best policy is always to:-
• be frank and honest with the patient and his/her relatives and
• inform the consultant in charge of the case and, if necessary the GP [Grade II].

WHAT COMPLICATIONS CAN OCCUR?

These include:-
• Over-sedation resulting in either ‘deep sedation’ or even general anaesthesia
• Paradoxical excitement and or sexual fantasies (rare)
• Drug induced respiratory depression with hypoxia and CO₂ retention
• Aspiration pneumonia
• Cardiac arrhythmias
• Hypertension, hypotension and/or vaso-vagal fainting
• Angina and myocardial infarct
• Stroke
• Nausea and vomiting
• A local burning sensation at both the site of injection and up the arm
• Generalised flushing
• Side effects specific to anticholinergics

Fig 1
Evidence Grading – established system (‘old’)
-see Eccles and Mason, 2001

Evidence grades
Ia: Evidence from a meta-analysis of randomised controlled trials
Ib: Evidence from at least one randomised controlled trial
IIa: Evidence from at least one controlled study without randomisation
IIb: Evidence from at least one other type of quasi-experimental study
III: Evidence from observational studies
IV: Evidence from expert committee reports or experts

Recommendation grades
A directly based on category I evidence
B directly based on category II evidence or extrapolated from category I evidence
C directly based on category III evidence or extrapolated from category I or II evidence
D directly based on category IV evidence or extrapolated from category I, II or III evidence

Fig 2
Evidence Grading – experimental system (‘new’)- see Mason and Eccles, 2003

Evidence Grade
I: High
II: Intermediate
III: Low

Recommendation Grade Interpretation
A Recommendation - There is clear evidence to recommend a pattern of care
B Provisional Recommendation- On balance a pattern of care is recommended although there are uncertainties
C Consensus Opinion - The group recommends a pattern of care based on its shared understanding

Fig 3
A full Explanation for the recommended procedure
The Motivation or reasoning behind the medical recommendations
The Benefits from undergoing the examination
The possible Risks involved
What Alternatives are available – including doing nothing
What Complications may occur (and where possible their frequency)
Any possible side Effects – particularly of any sedation/analgesics to be given
The acronym ‘EMBRACE’ used as an aide-mémoire [ref 4] in the consent process
Patients requiring anaesthetic support in the Gastrointestinal Endoscopy Unit

Elective Cases – indications include:-
- Patients with severe learning difficulties
- Patients in whom sedation has previously failed (or is likely to fail) e.g. certain alcoholic or drug addicted patients who may prove difficult to sedate and/or have poor venous access
- Phobic or uncooperative patients who insist on being ‘put to sleep’
- Any patient being sedated with IV propofol
- Outflow obstruction and any serious form of cardiac or pulmonary compromise

Emergency Cases deemed at high risk of aspiration and therefore requiring an endotracheal tube and a GA include:-
- Patients undergoing Endoscopy for any large GI especially if :-
  a) Depressed levels of consciousness associated with encephalopathy and suspected bleeding varices
  b) Patients unlikely to cooperate during endoscopy

CAUSES, RECOGNITION AND MANAGEMENT OF COMMON CARDIOVASCULAR AND SEDATION-RELATED COMPLICATIONS

OVER-SEDATION RESULTING IN EITHER ‘DEEP SEDATION’ OR EVEN GENERAL ANAESTHESIA

Definitions
Conscious sedation is now more correctly called moderate sedation/analgesia.

It is defined as a “drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patient’s airway and spontaneous ventilation is adequate” [ref 4].

Recognition of Over-sedation
The greater the percentage benzodiazepine and/or opioid receptor occupancy in the CNS the greater is the degree of depression of consciousness.

There is a continuum from desirable anxiolysis and antegrade amnesia in an awake (or easily rousable) patient right through to a patient who is anaesthetised with an unprotected airway.

Hence the necessity to keep in regular and frequent verbal contact with the patient to repeatedly check the level of sedation [Evidence Grade I, Recommendation Grade A].

Management of Oversedation
If a patient is judged to be becoming too heavily sedated, then he/she should be stimulated both verbally and, if necessary, with light shaking to wake up, open the eyes as well as being encouraged to take in a number of deep breaths.

If they are not responding/ unrousable then IV antagonists such as flumazenil and or naloxone may be required – see below [Evidence Grade I Recommendation Grade A].

DRUG INDUCED RESPIRATORY DEPRESSION

Background
Intravenous benzodiazepines (midazolam and diazepam) can cause respiratory depression as a result of the drug occupying brainstem benzodiazepine receptor sites which in turn may reduce respiratory drive.

Intravenous opioids (pethidine and fentanyl) occupy opioid receptor sites within the brain and brainstem and can similarly cause respiratory depression with resulting falls in both tidal volume and respiratory rate.

The sedative effects of benzodiazepines and opioids are synergistic [ref 7] [Evidence Grade I] so particular caution is required when these drugs are used in combination [Recommendation Grade A].

Note – Patients who are morbidly obese, suffer from sleep apnoea syndrome or chronic respiratory failure are at particular risk from respiratory depression.

- Drug induced hypoventilation may cause both hypoxaemia and CO2 retention which in extreme cases may progress to apnoea and even respiratory arrest [Evidence Grade I].
- Clinical observation of ventilatory effort and central cyanosis are notoriously unreliable.
- Pulse oximetry is a very useful indicator of oxygenation but not ventilation. However when supplemental oxygen is used, the fall in SpO2 may be significantly delayed for between 30–90 seconds after the onset of severe drug-induced respiratory depression/apnoea. It is for this reason that continuous capnography is recommended in patients being sedated with propofol [ref 4, 5 Evidence Grade I Recommendation Grade A].

RECOGNITION AND MANAGEMENT OF RESPIRATORY DEPRESSION

- As for oversedation, loss of verbal contact due to reduced conscious level may be the first sign of impending respiratory depression.
- Reduction in SpO2 on pulse oximetry is a good indicator but it can be a late sign of respiratory depression (see above).
- Increased pACO2 (where capnography is available) is the most sensitive early warning of respiratory depression.
- Should respiratory depression be suspected or confirmed:-
  - The patient should be stimulated to wake up and take deep breaths. It is NOT sufficient to merely turn up the rate of oxygen delivery!
  - If the patient is not responding then the agonist sedative/ analgesics may need to be reversed with the IV antagonists flumazenil plus (if necessary naloxone).
  - It is recommended that the benzodiazepine is reversed BEFORE the opioid Evidence Grade I Recommendation Grade A
  - The airway may need to be protected with chin lift, jaw thrust, plus, if necessary, airway, laryngeal mask or ET tube insertion.
  - The patient may require ‘bagging’ with an Ambu bag attached to an oxygen supply while awaiting the antagonist drugs to effect drug reversal.
  - If there is ANY doubt the services of the ‘Crash Team’ sought earlier rather than later [Evidence Grade I Recommendation Grade A].

ASPIRATION PNEUMONIA

Aspiration of gastric contents into the lungs is common, causes pneumonia and may result in death [refs 1–3].

It is at particular risk of occurring:-
- In oversedated patients as a result of an unprotected airway.
- Where there is an increased propensity to vomit e.g. in patients with GI bleeding, gastric stasis, gastric outlet obstruction or those patients who have simply eaten or drunk fluid within the last 4 hours.
• When a local anaesthetic spray such as lignocaine is used in combination with IV sedation; there is some evidence of an increased risk of aspiration [ref 2 [Evidence Grade I]].
• In elderly patients where an increased tendency to aspirate may be further confounded by an already poor gag reflex.
• In obtunded patients (for whatever reason) e.g. those with hepatic encephalopathy.

Recognition
• Aspiration may be suspected when a patient starts coughing violently either during or soon after an endoscopic procedure.
• He/she may become cyanosed or develop dramatic oximeter evidence of oxygen desaturation.

Management
• Suction of fluids from oral cavity and throat
• Increasing the rate of supplemental oxygen
• Correction of the level of consciousness where this is depressed (as above)
• Encouraging the patient to cough;
• Arrange chest X-ray, admission and alert the resident staff to the problem so that antibiotics, physiotherapy and appropriate monitoring can be arranged.

Note – Anaesthetic consultation may be advisable to decide on the optimal location (e.g. HDU/ITU) and level of respiratory support required in more serious or higher risk cases.

CARDIAC ARRHYTHMIAS
Cardiac arrhythmias are frequently observed during GI endoscopic procedures [Evidence Grade I]. Fortunately, most are not clinically significant.

Sinus tachycardia may be:
• Caused by anxiety or related to pain
• A compensatory mechanism in patients who are hypotensive as a result of either dehydration or blood loss.
• Seen following IV anticholinergics such as buscopan.
• Sinus bradycardia is seen:-
• Most frequently in patients who are taking beta blockers either for hypertension or IHD.
• When induced by vagal stimulation – which occurs either at the time of intubation of the oesophagus or the stretching of the sigmoid mesentery during colonoscopy or flexible sigmoidoscopy.
• Other frequently observed cardiac arrhythmias include:-
• Atrial and ventricular ectopic beats
• Atrial fibrillation and supraventricular tachycardia.

Ventricular tachycardia and even cardiac arrest due to VF are well described but fortunately rare (ref 2).

Note – Continuous ECG monitoring is recommended in at risk patients with a relevant cardiac history and/or if agents such as propofol are to be used [Evidence Grade I Recommendation Grade A].

HYPERTENSION, HYPOTENSION AND VASO-VAGAL FAINTING

Causes of hypertension
• Background systemic hypertension.
• Anxiety or pain – both raise blood pressure
• Intubation of the oesophagus – this can cause a reflex pressor response.

Causes of hypotension
Blood pressure is a reflection of cardiac output and total peripheral resistance and a fall in either or both will lower the patient’s mean arterial pressure.

Sedation –
• Benzodiazepines (midazolam, diazepam) have a mild vasodilatory effect and usually produce only a slight fall in BP in normal sedative doses.
• May cause more profound falls in BP in a hypovolaemic patient e.g. due to blood loss or dehydration.
• Combined use of a benzodiazepine and opioid can profoundly drop blood pressure – as can IV propofol.
• Bradycardia – of any cause
• A fall in heart rate (e.g. as a result of vagal stimulation) and/or cardiac stroke volume will also lower BP.
• Septic shock with vasodilatation –
• This cause of hypotension must be borne in mind especially in a patient with obstructive jaundice and cholangitis.

Prevention of Complications
• Relevant medical and drug history must be taken pre-procedure.
• Particular detail required regarding current antihypertensive, anti-anginal and anti-arrythmic therapy and the use of systemic corticosteroids.
• Equally important is establishing the timing of the last dose.
• Blood pressure and pulse should be recorded before, during and after any endoscopic procedure [Evidence Grade I Recommendation Grade A].
• An endoscopic procedure may need to be deferred if:-
• the patient’s BP is deemed to be dangerously high – allow time for hypertensive agents to have an effect.
• the patient’s BP is too low. The hypertensive patient may require resuscitation with blood and/or IV fluids before it is safe to proceed [Evidence Grade I Recommendation Grade A].

ANGINA AND MYOCARDIAL INFARCT
Myocardial infarction occurs either during or in the few days after endoscopic procedures with or without sedation (ref 1–4). A proportion of these are undoubtedly causally related to the endoscopic procedure. [refs 1–4].

Cause of Angina/Myocardial Infarction
Increased Myocardial Oxygen Need –
• Often, both sedated and non-sedated endoscopic procedures cause an increase in the ‘rate/pressure product’ (mean arterial BP X heart rate) – an indirect measure of myocardial oxygen consumption.
• This can cause angina pectoris in patients with IHD – or occult symptomless myocardial ischaemia which is only apparent with sophisticated monitoring e.g. myocardial perfusion study (ref 4).
• Marked hypertension and/or tachycardia – which may be related to the endoscopist’s competence – increase myocardial oxygen consumption
• Reduced Myocardial Perfusion –
• Hypotension and/or bradycardia reduce myocardial muscle perfusion (see above).

Prevention or Minimisation of Myocardial Ischaemia during Endoscopy
• Pre-oxygenate at risk patients and give continuous supplemental oxygen [Evidence Grade I Recommendation Grade A].
• Leave patients on their normal antihypertensives and/or anti-anginal therapy right up to the time of the Endoscopy – but beware that this could lead to hypotension during the procedure.
• Angina developing during an Endoscopy is usually best managed by giving sublingual GTN and oxygen and discontinuing the examination.
• If angina or a frank MI is suspected during or following an Endoscopy, arrange an ECG. If the result is equivocal or the chest pain prolonged then admission to the CCU to exclude an MI may be warranted [Evidence Grade I Recommendation Grade A].

Cerebrovascular Attacks
• Both TIAs and fully completed strokes can and do occur both during and following endoscopic procedures [1–4 (Evidence Grade I)].
• Possible mechanisms include
  • periods of either hypo or hypertension
  • cardiac arrhythmias or
  • as a consequence of an MI.

Prevention
Avoid extreme rises or falls in BP (see above) in at risk patients [Recommendation Grade A]

NAUSEA AND VOMITING

Causes
• Vomiting and nausea are common side effects of IV opioids.
• Any tendency to vomit is worsened by stimulating the vestibular receptors e.g. when turning a patient undergoing ERCP or a colonoscopy.
• A frequent non-pharmacological cause of nausea is overdistension of colonic loops at colonoscopy – indicating that the examination is not proceeding smoothly.

Prevention/Management
• Minimise opioid dose used
• Reassure the patient and keep in recovery position
• Have a vomit bowl readily available
• If the vomiting and/or nausea is protracted then an IV or IM anti-emetic such as metoclopramide may be required.
• (At colonoscopy) – check technique and loop formation continuously.

ADVERSE REACTIONS TO SEDATIVE INJECTIONS

Local
• Burning sensation at injection site travelling up the arm This is common with IV propofol and may be improved by using a larger vein or by the prior injection of IV lignocaine [Evidence Grade I Recommendation Grade A].
• Transient red wheal along the course of the vein IV opioids such as pethidine can cause this due to local release of histamine from the vascular endothelial lining of the vein. The patient can be assured this is a transient phenomenon with no long-term sequelae.

Systemic
• Occasionally IV opioids cause a more dramatic generalised histamine release associated with facial flushing and even marked hypotension
• Treatment is with IV antihistamine and IV hydrocortisone [Evidence Grade II Recommendation Grade B].

SIDE EFFECTS SPECIFIC TO ANTICHOLINERGIC DRUGS

Anticholinergic drugs such as Buscopan can cause:-
• Dry mouth, mental confusion, palpitations, mydriasis, constipation and difficulty voiding urine.
• Buscopan should therefore be used cautiously at the lowest possible dose or avoided in:-
  • elderly male patients with prostatic symptoms
  • patients with a history of closed angle glaucoma [Evidence Grade I Recommendation Grade A].
• In such patients IV glucagon can be used if an antispasmodic is required.

It should be remembered that pethidine itself has anti-cholinergic properties and can produce tachycardia and mydriasis whereas most other opioids conversely produce bradycardia and miosis.

REFERENCES
4 Bell GD. Review – Premedication, Preparation, and Surveillance in “State of the Art in Gastroenterologic Endoscopy – A review of last year’s most significant publications” Endoscopy 2004;36:23–31
COMPLICATIONS OF UPPER GASTROINTESTINAL ENDOSCOPY

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A. Introduction

Upper GI endoscopy is a commonly performed procedure used to investigate a wide range of symptoms and treat a variety of complaints. Its relative safety has encouraged its use in elderly patients and those with significant co-morbidity. However, it is an invasive procedure which carries with it a range of complications and a small but well recognised mortality. The referring clinician and endoscopist should therefore take care not to consider such procedures as routine. All patients should be carefully assessed prior to endoscopy and should be made aware of the complications and consequences that may occur. Procedures should be performed with by fully-trained endoscopists, or trainee endoscopists under close supervision and since the complications that do occur may not be apparent during the procedure, nursing staff and patients need to be aware of the early manifestations of common complications. Patients should be given written instructions on how to report unexpected symptoms following discharge from the endoscopy unit.

B. COMPLICATIONS OF DIAGNOSTIC UPPER GI ENDOSCOPY

Diagnostic upper GI endoscopy is a remarkably safe procedure. Although there are no recent high quality prospective studies of complications following diagnostic upper GI endoscopy, one large US study estimated an overall complication rate (including mucosal biopsy) of 0.13% and an associated mortality of 0.004% (1).

Common ‘Minor’ Problems

Many patients experience minor throat and abdominal discomfort after upper GI endoscopy. Although these are often considered minor complaints, one prospective study found that approximately 2% of patients sought medical advice for these complaints and occasionally patients were hospitalised (2).

Cardio respiratory

Cardio respiratory complications related to sedation and analgesia are the commonest complication of diagnostic upper GI endoscopy. Complications range from minor changes in vital signs to arrhythmias, myocardial infarction, respiratory arrest, shock and death.

- Elderly patients and those with pre-existing cardiopulmonary disease are at increased risk.
- Hypoxia is particularly common when intravenous sedation is combined with intravenous analgesia.
- Careful assessment and monitoring are essential and reversal agents and resuscitation equipment should be readily available (C).
- The reader is referred to the BSG ‘Guidelines on Safety and Sedation for Endoscopic Procedures’ (3).

Infection

Transient bacteraemia is uncommon following diagnostic upper GI endoscopy and is rarely of clinical significance. However, those at higher risk of endocarditis (Table 1) should be identified and given appropriate antibiotic prophylaxis.

The reader is referred to the BSG guidelines on ‘Antibiotic Prophylaxis in Gastrointestinal Endoscopy’ (4, C).

Aspiration pneumonia may complicate diagnostic upper GI endoscopy. Patients with a depressed level of consciousness (including over-sedation) and those with gastric and oesophageal stasis are at increased risk. There is some evidence that the combined use of local anaesthetic spray and intravenous sedation increases the risk (6, 7, III).
Uncommonly retro-pharyngeal and retro-oesophageal abscesses have been reported following difficult intubations presumably as a result of occult perforation.

**Bleeding**

Significant bleeding is a very rare complication of diagnostic upper GI endoscopy and mucosal biopsy is rarely complicated by bleeding sufficient to require intervention, in the absence of coagulopathy, thrombocytopenia or portal hypertension (1).

- Biopsy site bleeding is more often gastric than oesophageal
- Diagnostic upper GI endoscopy appears safe in patients with platelet counts as low as 20,000, but biopsies should be performed with caution below this level. If biopsy is essential then platelet transfusion should be considered (8, C).
- The risk of procedure-related bleeding in patients on anticoagulants needs to be weighed against the risk of thromboembolism if these drugs are discontinued.
- There is no convincing evidence that anticoagulant levels within a therapeutic range need to be adjusted for diagnostic endoscopy with biopsy. However, elective procedures should be avoided when the level of anticoagulation is above the therapeutic range (C).
- The limited available data suggest that standard doses of aspirin and other NSAIDs do not increase the risk of significant bleeding during or after gastrointestinal endoscopic procedures, even those including biopsy (III).

The reader is referred to the ASGE document ‘Guideline on the Management of Anticoagulation and Antiplatelet Therapy for Endoscopic Procedures’ (9)

**Perforation**

Perforation related to diagnostic upper GI endoscopy is uncommon with an estimated frequency of 0.03 and mortality of 0.001% (1).

- Perforation may occur in the pharynx or oesophagus, usually at sites of pathology or when the endoscope is passed blindly.
- Patient-related predisposing factors include anterior cervical osteophytes, pharyngeal pouches, oesophageal strictures and malignant obstructions.
- Perforation may follow biopsy of oesophageal or gastric malignancy.
- Perforation is more likely when the examination is performed by an inexperienced endoscopist (6, II).
- The increasingly recognised condition of eosinophilic oesophagitis is thought to predispose to a higher incidence of oesophageal tears and perforations even at purely diagnostic examinations.

**Rare Complications**

A range of unusual and rare complications has been reported after diagnostic upper GI endoscopy.

- Anaphylactic reactions to topical anaesthesia is very rare but potentially fatal.
- Dental trauma and temporomandibular joint dislocation may occur.
- Rarely the endoscope may become impacted in the distal oesophagus or in a hiatus hernia.

**C. COMPLICATIONS OF THERAPEUTIC UPPER GI ENDOSCOPY**

There has been a remarkable increase in the volume and diversity of therapeutic endoscopy in recent years. Such procedures are particularly beneficial in the elderly and those with significant co-morbidity since these patients are most at risk from surgery. Unfortunately therapeutic endoscopy is not without risk and this is sometimes appreciable.

Patients undergoing therapeutic endoscopy are a risk of the same complications as those undergoing diagnostic endoscopy. The magnitude of risk, however, is at least ten times greater (I, III). In addition, patients are at risk of a number of procedure-specific complications.

**General Considerations**

Therapeutic upper gastrointestinal endoscopy usually takes longer than diagnostic endoscopy and is often more uncomfortable for the patient. Higher doses of intravenous sedation are therefore often used and sedation may be combined with intravenous analgesia putting the patient at greater risk of cardio respiratory complications.

- Careful patient selection and consent and the judicious use of conscious sedation with appropriate monitoring are an essential part of therapeutic endoscopy (3).

Transient bacteraemia is more common during therapeutic than diagnostic upper GI endoscopy. It is therefore important that patients at risk of endocarditis are identified and given appropriate antibiotic prophylaxis (4). Infection is also common after upper gastrointestinal bleeding in cirrhotic patients, and a major cause of morbidity and mortality. All patients presenting with an episode of variceal bleeding should be given antibiotic prophylaxis (10, A).

Electrocautery units are frequently used to facilitate therapeutic endoscopy. The endoscopist should be familiar with the techniques and equipment in order to avoid the well-documented complications that may occur.

**Managing Patients taking Anticoagulants and Antiplatelet Agents**

The risk of procedure-related bleeding in patients on anticoagulants needs to be weighed against the risk of thromboembolism if these drugs are discontinued.

- For patients with low-risk conditions undergoing high-risk procedures warfarin therapy should be discontinued 3 to 5 days before the scheduled procedure. A pre-procedure prothrombin time is advisable.
- For patients with high-risk conditions undergoing high risk procedures warfarin therapy should be discontinued 3 to 5 days before the procedure. Intravenous heparin should be started once the INR falls below the therapeutic level. Heparin should be discontinued 4 to 6 hours before the scheduled procedure and may be resumed 2 to 6 hours after the procedure. Warfarin therapy may generally be resumed the night of the procedure.
- Limited available data suggest that standard doses of aspirin and other NSAIDs do not increase the risk of significant bleeding.
- There are insufficient data to make recommendations regarding newer antiplatelet drugs, such as clopidogrel, but it is prudent to discontinue these medications seven to 10 days before a high-risk procedure.

The reader is referred to the ASGE document ‘Guideline on the Management of Anticoagulation and Antiplatelet Therapy for Endoscopic Procedures’ (9)

**COMPLICATIONS RELATED TO SPECIFIC UPPER GI THERAPEUTIC PROCEDURES**

1) Dilatation and Stent Insertion

**Oesophageal Dilatation**

The principal complications of oesophageal dilatation are perforation, pulmonary aspiration, and bleeding.

The overall perforation rate is 2–3% with a mortality of 1%. Perforation is less common following dilatation of benign strictures (1–2% with a mortality of 0.5%) than following...
dilatation and/or intubation of malignant strictures (4–6% with a mortality of 2–3%)(11). Caustic strictures may be at greater risk of perforation. The risk of perforation in achalasia is 0–7% (mostly 3–4%) with mortality of 1% (7,11). The perforation rate may be lower with a graded approach to balloon dilatation but most perforations occur during the first dilatation (12).

- The risks of perforation following oesophageal dilatation are greater when the endoscopist is inexperienced. Endoscopists who have performed less than 500 diagnostic procedures are four times more likely to cause perforation than their more experienced colleagues (6)
- The risks are also greater when strictures are angulated or complex, particularly when weighted bougies are passed blindly
- In most cases it is wise to use either wire guided or endoscopically controlled techniques. The addition of radiographic screening is recommended when the stricture is tortuous or complex or associated with large hiatus hernia or diverticulae and when difficulty is encountered passing the guidewire (11)(B)
- Elderly patients are more at risk but malignant stricture treated with radiation do not appear more susceptible to perforation
- Perforation usually occurs at the site of the stricture resulting in intra-abdominal or intra-thoracic perforation, the latter being more serious.
- Although perforation is often linked to the use of large dilators it may complicate the passage of a small dilator or be caused by the guide wire.
- The passage of a single large dilator appears safe in simple uncomplicated strictures but a cautious graded approach is recommended in patients with tight, tough, or complex strictures (13)(B).
- Comparative trials show there is no evidence that push dilators result in a higher perforation rate than balloon dilators when dilating benign strictures (14,15,16)(1)

**Oesophageal Stent Placement**

Several endoscopic modalities are available for the palliation of inoperable malignant dysphagia. The placement of an endoluminal stent is frequently used to relieve obstructive symptoms.

In recent years self-expanding metal stents (SEMS) have become more popular than rigid plastic stents since they are associated with fewer procedure-related complications. Despite high unit costs they appear cost-effective because of the shorter inpatient stay needed for treatment of procedure-related complications(17). Covered SEMS are the treatment of choice for patients with malignant tracheo – oesophageal fistula(18).

SEMS are effective in improving dysphagia in most patients but complications are frequent (20–40% with a mortality of 3%) • Pain is frequently reported after stent placement and may be severe
• Perforation is an often apparent during placement of the stent. It is more common following placement of rigid plastic stents than SEMS (approximately 5% versus 1–2%)
• SEMS may be inserted without dilation or with minimal dilation (delivery systems 7–11 mm). The risks may be increased when stent placement is performed in patients where other palliative therapies have failed (eg chemotherapy and/or radiotherapy) but this is not a consistent finding (19,20).
- Haemorrhage is usually a late complication and the precise source may be difficult to localize. It is often difficult to distinguish between disease progression and stent-related complication.
- Stent migration is common (5–15%). Initial concerns that covered stents may be more prone to migration have not been borne out. Migration is more common, however, when stents are placed across the gastro-oesophageal junction than when placed more proximally. Wider diameter stents appear less likely to migrate from this site but this may be offset by an increased risk of perforation and bleeding(21)(II)
- Tumour ingrowth is less with covered stents. Tumour overgrowth occurs in 10–20% of patients and affects both ends of the stent at a similar rate.
- Food bolus obstruction is reported in 5–15% of patients. Dietary advice can be helpful
- Gastro-oesophageal reflux develops frequently when the distal end of the stent straddles the gastro-oesophageal junction. Proton pump inhibitors and postural advice are frequently used. Recent experience with SEMS incorporating antireflux mechanisms has been encouraging but whether these will decrease the incidence of aspiration is not yet known(22)

**Gastroduodenal Dilatation and Stent Placement**

Several studies report successful endoscopic dilatation of pyloric stenosis. Perforation rates of 0–6.7% are reported. Balloon diameters greater than 15 mm seem associated with increased risk (23)(III).

Enteral SEMS have been used increasingly for the palliation of inoperable upper abdominal malignancy. Through the scope and wire guided systems are available. They appear to be associated with similar complications to oesophageal stents(24).

2) **Haemostatic Techniques**

**a) Non Variceal Haemorrhage**

A range of endoscopic treatments is available for patients who have actively bleeding lesions and those at high risk of rebleeding. Injection, thermal and mechanical methods are used, often in combination (25).

- Injection with adrenaline (1 in 10,000) is widely employed for ulcer haemostasis. It is easy to use and has few clinically significant complications.
- The use or addition of sclerosants (STD, polidocanol, and ethanolamine) or absolute alcohol should be avoided since they do not aid primary haemostasis or reduce the rate of rebleeding and may cause life threatening tissue necrosis (26)(C)
- Multipolar and bipolar coagulation and the heater probe are the most widely used techniques to induce coaptive coagulation. Perforation rates of 0–2% have been reported but may increase with repeat treatment(27). Traditional monopolar electrocoagulation and laser treatment should be avoided because of the increased perforation risk(C)
- Induction of bleeding occurs in 5% and can usually be dealt with endoscopically
- Clips are used increasingly in acute non variceal bleeding. The technique appears safe and the principal complication is failure to accurately position the clip leading to treatment failure

**b) Variceal Haemorrhage**

**Oesophageal Varices**

Both variceal sclerotherapy and band ligation are useful in the treatment of oesophageal varices. Variceal band ligation is the method of first choice but if banding is difficult or not available, sclerotherapy should be performed(A).

Sclerotherapy has been used for many years. A wide variety of sclerosants has been employed for both intra- and para-variceal injection. Coagulation necrosis ensues and variceal thrombosis and inflammation lead to scarring and
variceal obliteration. Sclerotherapy is an effective technique but complications are frequent (28,29).

- Superficial ulceration occurs in 90% of patients the day after sclerotherapy and in 70% at one week.
- Deep ulceration is less common but may lead to rebleeding (approximately 5%) and oesophageal stricture formation (2–20%). Ulceration occurs more often when sclerotherapy is performed frequently.
- Transmural inflammation may lead to mediastinitis, pleural effusion and perforation (2–5%).
- Minor complications such as chest pain, fever and mild dysphagia are common.
- Serious procedure-related complications occur in 1–20% of patients, with an overall mortality of 2–5%.
- Portal vein thrombosis may occur.
- Aspiration pneumonia may occur in up to 5%. Endotracheal intubation should be used early in patients with impaired consciousness or significant ongoing haematemesis.
- Oesophageal dysmotility is common after sclerotherapy but does not appear to be of clinical significance.

Endoscopic Band Ligation relies on physical strangulation of the varix by a rubber band to induce thrombosis and necrosis. The treatment is as effective as sclerotherapy but since the local and systemic inflammatory response is less, complications are fewer (30,31)(1).

- Superficial ulceration is frequent (5–15%).
- Perforation has been reported in 0.7% but in patients where an overtube was used.
- Oesophageal structuring is rare.
- Overall mortality is approximately 1%.

Gastric Varices

The intravariceal injection of tissue adhesives appears more effective than band ligation in the treatment of gastric varices. On contact with blood, the adhesive rapidly polymerises forming a hard cast that plugs the varix. The treatment is as effective as sclerotherapy but since the local and systemic inflammatory response is less, complications are fewer (30,31)(1).

- Embolisation of glue occurs in 2–5%. The lung, spleen, portal vein, renal vein, inferior vena cava or brain may be affected.
- Rarely fistulae may occur.
- Extraction of the glue cast may be associated with massive rebleeding.
- The injector needle may become stuck on the varix.
- The accessory channel of the endoscope may get blocked.

3) Polypectomy and EMR

Upper gastrointestinal polyps are usually found incidentally during diagnostic upper GI endoscopy. Endoscopic appearances do not reliably distinguish histological type. Since simple biopsy may be subject to sampling error it is suggested that gastric epithelial polyps over 5 mm should be removed. Furthermore, in patients with multiple polyps it would seem wise to remove the larger polyps and those with atypical endoscopic features with multiple simple biopsy of the smaller polyps. When a submucosal lesion is suspected EUS should be undertaken (C).

- Haemorrhage following gastric polypectomy occurs in approximately 2%. Adrenaline injection prior to polypectomy and the application of loops and clips have been used when polyps are large.
- Perforation may occur.

Endoscopic Mucosal Resection (EMR). Note – this technique should only be undertaken by experienced endoscopists who have undergone appropriate training. It involves lifting a mucosal lesion away from the deep muscle layer by the injection of fluid into the submucosal layer. The lesion is then removed by snare, sometimes combined by suction into a cap device. The technique is useful for the removal of flat/depressed and sessile lesions. The availability of the resected sample for histological examination gives it a clear advantage over ablative techniques (33).

- Bleeding is the most frequent complication (up to 17% depending on definition) and is usually apparent at the time of resection. Fortunately, bleeding during EMR usually stops spontaneously. Some studies suggest resections >1–2 cm are more prone to bleeding. No significant associations have been found with EMR method, type of lesion, amount of saline injected, type of current or location of the lesion (34).

- Perforation occurs either when the deeper muscle layer is included in the resection specimen or when overly aggressive cautery results in a transmural burn and delayed perforation. Gastric EMR perforation rates are high (1–5%) and seem more common with the insulated knife technique (35).

- To reduce the risk of perforation it is important to: inject an adequate volume of fluid to get safe separation between the resection line and muscle layer; exercise caution or avoid when previous EMR or interventions have occurred. Since scarring may prevent proper lifting of the lesion from the muscle layer; stop if the patient develops pain since this may suggest full thickness resection.

- The management of EMR associated perforation should be undertaken in collaboration with a senior surgeon. If the perforation is early, small and asymptomatic the defect may be clipped. Conservative management should be combined with regular surgical review (see later).

- Luminal stenosis has been reported as a late complication of oesophageal EMR and tends to occur after extensive, near circumferential, resection (36). Balloon dilatation and stent placement have been used with good effect.

4) Ablative Techniques

Laser, Photodynamic Therapy and Argon Plasma Coagulation are ablative techniques used for treatment of pre-malignant and early malignant upper GI lesions and the palliation of advanced disease. Chemical injection to induce ablation has occasionally been used as a palliative technique.

Laser light is able to coagulate, cut and vapourise tissue depending on the wavelength and power settings employed. Laser is thus a powerful tool but the need for ocular protection, exhaust ventilation and specialist training limits its value. Furthermore complications are frequent (37).

- Major bleeding has been reported in up to 12.5% of patients treated for gastric tumours.
- Perforation occurs in 1–9%.
- Occasionally fistula develop.
- Procedure-related mortality of 1%.
- Stricture are a frequent late complication.

Photodynamic therapy utilises laser light excitation of a photosensitiser which releases reactive oxygen causing tissue necrosis. A number of photosensitisers are available. The technique is used for the treatment of Barrett’s oesophagus and the palliation of oesophageal and gastric malignancies (38,39). Complications include:

- Prolonged skin photosensitivity.
- Chest pain, dysphagia, odynophagia, nausea and fever are common.
- Perforation and fistula.
- Strictures in up to 30%.

Argon plasma coagulation employs high frequency current and ionised argon gas to deliver superficial thermal energy. It appears the easiest and safest ablative technique but
when used for palliation of bulky tumours multiple sessions may be required (40)

- Perforation is rare due to the superficial nature of the injury and the thickness of the upper gut wall. Higher power settings are associated with greater risk
- Bleeding is also uncommon and indeed APC is employed as a haemostatic technique.
- Abdominal distension is common but can be minimised if the endoscopist remembers to aspirate the insufflated gas
- Stricture may develop as a late complication

Chemical injection with small volumes of alcohol or polidocanol is an inexpensive alternative palliative technique in patients with malignant dysphagia (41)

- Exophytic tumours are best suited to this treatment
- Fistula, perforation and mediastinitis occur but are rare

5) Removal of Foreign Bodies

Foreign body removal and food bolus disimpaction comprise a small but important aspect of therapeutic endoscopy. Complications have been reported in up to 8% (42)

- The blind passage of dilators and the use of papain are associated with a significant risk of perforation and should be avoided (C)
- The risk of aspiration and mucosal damage from sharp objects may be reduced by the use of an overtube but the use of an overtube may increase the risk of haemorrhage and perforation (C)
- Failure to retrieve the foreign body occurs in 5%
- Repeat endoscopic examination after retrieval is useful to look for the presence of underlying pathology

D. RECOGNISING COMMON COMPLICATIONS

Perforation

In most cases a combination of technical difficulties and usually an interventional procedure should lead to a high index of suspicion. The history and physical signs may be useful pointers to the site of perforation

- Cervical perforation of the oesophagus: this can result in pain localised to the neck, hoarseness, pharyngeal dysphagia, painful neck movement, sternomastoid spasm and subcutaneous emphysema.
- Intra-thoracic and intra-abdominal perforations: these are more common and the endoscopist should consider any of the following as indicators of perforation:

  Immediate  
  - Chest pain
  - Haemodynamic instability
  - O2 desaturation
  - Visual evidence

  Early  
  - Any of the above
  - Respiratory difficulties
  - Increasing abdominal pain
  - Subcutaneous emphysema/pneumothorax/hydro pneumothorax
  - Peritonitis

  Late  
  - Any of the above
  - Unexplained pyrexia
  - Systemic sepsis/abscess formation
  - Fistula development
  - Rising inflammatory markers
  - Unexplained radiological appearances
  - Insidious deterioration

Prompt and thorough investigation is the key to management

- Careful endoscopic assessment at the end of any procedure, often combined with a chest X-ray, will identify many cases of perforation immediately.
- If not recognised immediately, then early and late suspected perforations should be assessed by a water-soluble contrast swallow.
- If this is negative, a small leak may not have been identified and a dilute barium swallow should be considered. CT scan can be used to replace a contrast swallow or as an adjunct to accurately delineate specific fluid collections (C).

Haemorrhage

- This may be evident during the procedure
- Early signs of haemodynamic instability, haematemesis, melena and the onset of anaemia should all prompt the need for appropriate resuscitation and investigation.
- Endoscopic re-examination is the procedure of choice once the patient is haemodynamically stable (C)

E. MANAGING COMPLICATIONS

Perforation

Cervical oesophagus

- Small instrumental perforations can nearly always be managed conservatively.
- The development of a local abscess is an indication for cervical drainage. This prevents the extension of sepsis into the mediastinum. The ensuing salivary fistula will close in the absence of any distal obstruction (43).

Thoraco-abdominal oesophagus

Conservative management should be undertaken in conjunction with a senior surgeon. It is successful and appropriate if the perforation is relatively small and occurs in the fasted, empty oesophagus – as long as the following criteria apply (44,45,46) (III,III,III) (C)

- Perforation detected early and prior to oral alimentation
- Absence of shock or systemic sepsis
- At worst, mild to moderate fever or leucocytosis
- Pain readily controlled with opiates
- Absence of crepitus, diffuse mediastinal gas, hydropneumothorax or pneumoperitoneum
- Mediastinal containment of the perforation
- Absence of widespread extravasation of contrast material
- No evidence of ongoing luminal obstruction or retained foreign body
- Patients demonstrating tolerance following diagnostic delay
- Patients who remain clinically stable with conservative management

These same principles can be applied to more distal perforations in the stomach and duodenum but the proportion of patients with perforation at these sites that can be managed conservatively is much lower.

The principles of non-interventional management are:

- Hyperalimentation preferably by an enteral route if feasible
- Naso-gastric suction
- Broad spectrum intra-venous antibiotics

Deterioration in the patient’s clinical condition should prompt further investigation and re-evaluation. Persistence with non-operative intervention may be appropriate but only if satisfactory drainage of any collections can be achieved by CT or ultrasound guidance.
Surgical management is required in the following circumstances:

- Clinically unstable patients with sepsis or shock
- Heavily contaminated mediastinum, pleural space or peritoneum
- Widespread intra-pleural or intra-peritoneal extravasation of contrast material
- Ongoing luminal obstruction or retained foreign body
- Failed medical therapy

Perforations in Malignancy and/or Frail Patients

Patients who sustain an instrumental tear related to malignancy represent a unique group. Those with disseminated disease or who are considered unfit for a surgical resection are best treated by placement of a covered self-expanding stent if this can be achieved (C). The use of covered stents in perforations in benign disease may be appropriate in unfit patients or where co-morbid disease is likely to lead to a short life expectancy.

Bleeding

The need for endoscopic intervention may be evident during the procedure and repeat endoscopy to localise the site of bleeding and facilitate endoscopic haemostasis is nearly always appropriate. Prompt resuscitation and the correction of coagulopathy are mandatory, although these steps should not unduly delay definitive attempts to arrest haemorrhage. Standard methods of endoscopic haemostasis should be employed. Many would recommend combination therapy (C).

- Adrenaline injections are easy to use and frequently the best first option
- Mechanical haemostasis with clips, snares and bands are technically more demanding but widely used for control of haemorrhage from polypectomy and EMR sites. Clips are technically easier to apply when the bleeding point can be approached en face.
- Thermal methods should be applied cautiously if the bleeding has resulted from thermal treatment of a mucosal lesion in order to minimise the risk of transmural burn and perforation

 Patients should be closely observed for signs of rebleeding following apparently successful endoscopic treatment. A low threshold for a further check endoscopy is sensible. Failed endoscopic haemostasis should lead to consideration of radiological or surgical intervention determined on the basis of the patient’s overall condition and the nature of the source of bleeding (C).

F. PREVENTING COMPLICATIONS

Given the serious nature of many of the complications of upper GI endoscopy it is desirable to consider strategies to minimise complications and aid early detection (C).

Appropriate Patient Selection

The use of an interventional technique in particular, merits careful consideration in patients with specific co-morbidities. The balance between risk and benefit should be considered in relation to the endoscopic procedure versus other forms of investigation or treatment.

Appropriate Patient Preparation

Attempts should be made to improve pre-existing medical problems, particularly coagulopathy.

In patients with obstructing lesions, efforts should be made to reduce contamination should perforation occur, by the use of liquidised diets, appropriate fasting and occasionally lavage via a wide bore naso-gastric tube. The latter may be particularly appropriate in the context of pneumatic dilatation for achalasia.

Orthodox Techniques

- Familiarity with recognised techniques and equipment is essential.
- The endoscopist should follow established protocols
- Procedures should be performed with by fully-trained endoscopists, or trainee endoscopists under direct supervision

Prompt Recognition and Acceptance that a Complication has Occurred

- Monitoring during the recovery period is an essential part of endoscopy
- Nursing staff and patients need to be aware of the early manifestations of common complications. Patients should be given written instructions on how to report unexpected symptoms following discharge from the endoscopy unit.
- A high index of suspicion and early and thorough investigation provides the best opportunity to institute correct management and minimise the risk of further complications or mortality.

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COMPLICATIONS OF COLONOSCOPY

Dr Owen Epstein

INTRODUCTION

Colonoscopy is a highly efficient imaging modality. By its nature, it is invasive and thus carries with it an intrinsic potential for a range of adverse events ranging from quite mild right up to and including death. Every colonoscopy should therefore be performed by a fully trained operator – or by a trainee with close support.

When performing the procedure, the colonoscopist should be constantly aware of this potential for harm. It should always be performed with care and compassion. Both endoscopy staff and patients need to be alert to the early manifestations of complications and patients should be given clear instructions on how to respond to unusual symptoms following discharge from the endoscopy unit.

INCIDENCE OF COMPLICATIONS OF DIAGNOSTIC AND THERAPEUTIC COLONOSCOPY

Reports on the frequency of complications at or after colonoscopy vary widely in the literature. Reasons include:-

- Most studies are retrospective and based on case note audits.
- Few prospective studies have been designed with the sole purpose of monitoring complications.
- Case-mix, single or multi-centre analysis, level of skill and training of the colonoscopist and the availability of facilities for detection and management of adverse events are uncontrolled variables which confound interpretation of published reports.
- Measurement bias, failure to standardise definitions of complications, incomplete reporting and lack of independent validation also prevent comparability of different studies.

Mortality complicating Colonoscopy

- Death is a fortunately rare but nevertheless significant consequence of colonoscopy in a very small number of patients.
- There is no one single cause – it can be the final result of many different complications outlined below in more detail.

Incidence of death

This is difficult to estimate accurately as:-

- Most of the other reports did not specify 30 day mortality rates

Causes of death

- In the UK study, most deaths occurred in symptomatic patients who were elderly with co-morbidity and at risk from any interventional procedure.
- In this group, colonoscopy related mortality was due to stroke, myocardial infarction, bronchopneumonia and sepsis.

CLASSIFICATION OF COMPLICATIONS OF COLONOSCOPY

Adverse events associated with colonoscopy may be:

- Complications caused by the pre-colonoscopy bowel preparation period
- Complications due to the procedure itself – due to:-
  - The sedation
  - The insertion of the instrument
  - Diagnostic or therapeutic techniques employed during the procedure

Complications may be:

- Either immediately apparent when they occur
- Or apparent only after a variable delay – complications have reported for up to 30 days after the investigation.

COMPLICATIONS OCCURRING BEFORE COLONOSCOPY

Complications of bowel preparation

Bowel cleansing regimens are all uncomfortable for patients but significant complications are rare.

- Most complications occur in elderly patients – up to 25% of whom experience at least one episode of incontinence during colonoscopic cleansing.
- Particular care is required to avoid excessive fluid and electrolyte shifts in this age group
- Higher incidence of complications in those with significant co-morbidity such as cardiac failure, hypertension and renal failure

The most frequently used cleansing regimens are polyethylene glycol (PEG) and sodium phosphate-based preparations:

- Patients taking PEG are required to drink 3 or 4 litres of fluid. Many may experience satiety, volume intolerance, abdominal bloating and nausea and even vomiting.
- Sodium phosphate is well tolerated and employs smaller volumes, but:
  - This compound commonly causes subclinical intravascular volume depletion and hyperphosphataemia with occasional deaths reported due to this
  - The incidence of this complication appears to be related to age as well as renal function (Grade III evidence).
• The preparation should therefore be used with great care in elderly patients and those with significant co-morbidity, especially renal or cardiac failure (Grade C recommendation).

**COMPLICATIONS OCCURRING DURING COLONOSCOPY**

**Complications related to sedation**
See section on Complications of Sedation.

Remember:
- Patients undergoing colonoscopy are at extra risk because of the more usual combined use of a benzodiazepine and an opioid (usually pethidine and midazolam).
- Sedation training is an essential prerequisite for all colonoscopists including recognition and management of the first signs of over-sedation before hypoxia manifests.
- Try to avoid “top-up” medication; good colonoscopic technique should allow comfortable colonoscopy to be performed in most patients without the need for deeper sedation with its associated greater risks.

**Cardiovascular complications**

Serious cardiac events such as cardiac arrest and myocardial infarction are rare during or after colonoscopy. However:-
- Routine colonoscopy is associated with increase in sympathetic tone which may result in hypertension, arrhythmias and ST segment changes (grade III evidence).
- No direct comparative studies are available but tachycardia, dysrhythmia, myocardial infarction and cerebrovascular accidents appear to occur more frequently in colonoscopy than upper GI endoscopy – probably caused by stretching of the viscus and its mesentery triggering the autonomic responses.

Prevention:-
- Good technique, with avoidance and control of looping
- Pulse rate monitoring throughout the procedure
- Discontinue the procedure if significant and persistent tachy- or bradycardia occurs.
- Continuous ECG monitoring should be considered if there is a history of heart disease (grade C recommendation).

**Pain reaction during colonoscopy**

Pain is the most frequent undesired effect of colonoscopy

- Whilst some discomfort is expected during insertion of the colonoscope, pain should act as a “red flag”
- Most commonly caused by:
  - Loop formation – especially in the sigmoid colon
  - Excessive air insufflation.

Pain can indicate:
- Unacceptable stretching of the viscus and its mesentery
- That the procedure is being performed near the limits of the organ’s capacity to accommodate the instrument.

Nausea (and vomiting) accompanying pain is particularly worrying; it can indicate loop overdistension and if the patient complains of this symptom, the operator should consider stopping the procedure.

**Prevention of pain:**
- Avoidance of looping
- Good technique
- Early recognition of loop formation – loss of synchronous “1:1” advancement of the tip.
- Withdraw and straighten the shaft whenever tip-control is lost and looping becomes prominent

• Liberal use of hand pressure, “torque-steer” and change of position to prevent excessive loop formation
• Avoidance of over inflation

Note – the assessment of the significance of pain during colonoscopy requires the highest degree of clinical judgment by the endoscopist and his/her assistants since so many variables contribute to the interpretation. Absolute rules are impossible to frame but the wise endoscopist takes full account at all times of the views and opinions of his/her experienced assistants.

**PAIN AFTER COLONOSCOPY**

Whilst abdominal discomfort may occur after any colonoscopy, post-procedure pain should be carefully observed and managed.

Commonest cause is gaseous distension:-
- In these patients, pulse, blood pressure and temperature are within acceptable limits,
- The patient does not look unwell; abdominal examination does not suggest peritoneal inflammation.
- The symptom eases with the passage of flatus.
- Symptoms resolve within a few hours.

Gaseous distension:
- May be more common after the use of anticholinergics such as buscopan
- May resolve faster by lying patient prone rather than supine.

Other important causes of pain include perforation and the post-polypectomy syndrome.

**COLONIC PERFORATION**

**Background**

Perforation occurs by three different mechanisms: pneumatic perforation, mechanical perforation and perforation associated with therapeutic colonoscopy.

Pneumatic perforation occurs when the intraluminal pressure is sufficient to rupture the colon wall.

- This is more likely if transmural inflammation or ulceration weakens the colon wall.
- The caecum is most susceptible to pneumatic rupture, followed by the transverse colon, sigmoid colon, and rectum. This is because:
  - The wall of the right colon is thinner than the left colon.
  - Colonic wall tension is highest in the caecum. The Law of Laplace states that wall tension is directly related to the radius of a cylinder. The radius is greatest in the caecum and hence the mural pressure greatest for any given intraluminal colonic pressure.

Mechanical perforation is most commonly due to forceful insertion of the colonoscope.

- The generation of high stretch and shear pressures transmitted to either the shaft or the tip of the instrument may cause laceration of the colon wall and rupture.
- Weakened areas of colonic tissue (eg diverticulitis, inflammatory bowel disease, ischaemic colitis or recent surgical anastomosis) need less intense transmitted pressure and thus predispose to perforation.

Therapeutic colonoscopy may cause perforation in several ways.

- Uncontrolled, forceful, or blind passage of instruments (forceps, brush, or unopened snares) may go directly through the colon wall.

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• Thermal injury may occur during hot biopsy or polypectomy.
  • The potential for thermal tissue injury is related to the intensity, duration, and diameter of application of the current, which in turn is inversely proportional to the diameter of the polyp stalk.
  • It is important to remember that, as the polypectomy snare tightens, the stalk diameter decreases causing a sharp increase in current density and thus propensity to cause transmural damage.
  • A transmural burn with perforation may also occur when the head of the polyp is in contact with the opposite wall allowing current flow through and injury to that wall.

Frequency of perforation:
The reported frequency of perforation varies and is probably related to both case-mix and experience of colonoscopists (Grade III evidence).

• In a prospective audit of 9223 colonoscopies in 68 units in England, perforation was reported in 12 patients (7 males and 5 females, age range 30–93). Overall perforation rate was 1.769 (0.13%). Perforation occurred in 1:923 (0.11%) patients undergoing diagnostic colonoscopy and 1:460 (0.21%) following therapeutic intervention (2 with snare polypectomy, 1 with hot biopsy and 1 with hot biopsy and snare polypectomy).
  • Bowles has summarised perforation rates in 10 other studies with rates ranging from 0–0.19% (1:520).
  • A report of 183 perforations indicated that the sigmoid is most commonly affected (72%), followed by the ascending and descending colon (both 8.6%), rectum 6.9%, transverse colon 3.4%. Two patients had small bowel perforation and no site was defined in two others.

Clinical features:
All colonoscopists need to be mindful of the possibility that diagnostic and therapeutic colonoscopy can cause perforation.

Perforation may be:-
Immediately apparent – recognised during colonoscopy.
  • This occurs in 42% of patients (Garbay et al).
  • Perforation may be recognised by the appearance of peritoneal structures such as mesenteric vessels, fatty tissue or the external surface of surrounding bowel. However, if perforation has been caused by colonic overdistension by the shaft of the instrument, there will be no abnormal view – except perhaps for a sudden inability to maintain adequate intraluminal distension.
  • The sudden onset of pain, difficulty maintaining insufflation or a change in the patient’s clinical status (pulse, BP or O2 saturation) may all be clues that perforation has occurred.
  • Approximately 50% of perforations are less than 2cm in size – so may not be readily visible

Diagnosed after a delay – ranging from 1 hour to 42 days
  • This occurs in the majority (Garbay et al).
  • Should be suspected if pain persists after the procedure and worsens or becomes generalised over a period of a few hours.
  • The most common symptom associated with perforation is increasing abdominal pain which may be accompanied by abdominal distension.
  • Failure to pass flatus despite obvious distension also suggests the diagnosis of perforation.

• Tachycardia, fever, localised or generalised abdominal tenderness, guarding and leucocytosis should raise a strong suspicion of perforation.

The clinical features of perforation are variable. They depend on:-
  • the site and size of the perforation
  • the amount of faecal spillage into the peritoneum
  • the premorbid condition of the patient.

Recognition:
Usually not difficult to diagnose perforation accompanied by generalised peritonitis.

Often more difficult to diagnose lesser degrees of abnormality e.g. localised pain, which does not progress to peritonitis,
  • This may be due to a small perforation which remains localised, or the “postpolypectomy syndrome”.
  • In patients with a small and localised bowel perforation, the pain and tenderness remains localised but peritoneal gas is confirmed on abdominal radiology. These perforations are (incorrectly) termed “miniperforations”.

Post-polypectomy syndrome.
After polypectomy (either by snare or hot biopsy), a transmural burn without perforation may cause symptoms resembling localised perforation.

  • The clinical features of the burn injury include localised pain, tenderness, guarding and rigidity, fever, leucocytosis and tachycardia.
  • The clinical picture is indistinguishable from localised perforation with peritonitis, but can be distinguished by the absence of free air on the abdominal radiology.
  • In most cases, the injury settles without progressing to full perforation

Investigation:
Any patient suspected of perforation should immediately undergo a plain erect chest and abdominal X-ray.

  • The presence of air under the diaphragm and pneumoperitoneum is diagnostic of perforation. Pneumoperitoneum or retroperitoneal air can be detected on plain abdominal radiology in 88% of patients (grade III evidence).
  • If there is no air on plain radiology, an abdominal CT scan should be performed. This investigation is more sensitive for the detection of pneumoperitoneum (grade C recommendation).
  • If there is evidence of perforation, a contrast enema using water-soluble contrast may help differentiate between a free perforation and a localised sealed off breach.
  • In patients who have undergone a polypectomy, failure to demonstrate an air leak suggests a transmural burn and the postpolypectomy syndrome.

Management of colonic perforation:
The most important first step is that an experienced gastrointestinal surgeon should assess the patient as soon as a perforation is suspected.

Conservative management
  • Some post-polypectomy perforations may be managed with non-operative care.
  • Conservative management may be considered in reasonably well patients with a localised, contained perforation or postpolypectomy syndrome (grade C recommendation).

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• Non-surgical treatment includes careful monitoring, nasogastric suction, intravenous hydration and antibiotics and bowel rest.
• Gram-negative aerobes and anaerobes are the main pathogens; the aerobes are primarily responsible for early fatal peritonitis, and the anaerobes (especially Bacteroides fragilis) are responsible for late intra-abdominal abscess formation. The microbiologists should guide choice of antibiotic and it is usual for patients to be treated with metronidazole and a 3rd generation cephalosporin.

Surgical management
• Almost all traumatic perforations need surgical repair as they are often large and irregular(grade III evidence).
• Immediate laparotomy is indicated if there is peritonitis, abdominal pain with pneumoperitoneum on X-ray or CT scan or if a trial of antibiotics for localised disease fails to elicit a response(grade C recommendation).
• If at surgery there is limited bowel injury, no pre-existing colonic pathology, and minimal faecal contamination of the peritoneum, a simple primary repair can be performed.
• If the patient has colonic injury, and limited or moderate faecal contamination, resection of the damaged colon with primary anastomosis can be considered. If there is extensive peritoneal soiling, a colectomy with colostomy should be performed.

Minimising the risk of perforation:

Patient selection –
Be aware that the following groups of patients are at higher risk of perforation:
• Elderly frail patients (especially women).
• Patients with severe diverticulosis
• A history of abdominal or pelvic malignancy
• A history of radiation therapy for abdominal or pelvic cancer
• Patients with a history of pelvic or major abdominal surgery.
• Extensive adhesions from prior abdominal surgery
• Fulminant colitis, severe active colitis and toxic megacolon (grade C recommendation). In most circumstances, colonoscopy should be avoided or delayed in these patients

Alternative imaging modalities such as barium enema, CT or CT colography can be offered as the initial imaging examination for some of these patients and colonoscopy only undertaken as a second line procedure if information is still incomplete or inadequate – or therapy is required.

Good technique –
Is likely to reduce the incidence of perforation.
• Avoid excessive colonic insufflation by judicious use of the air button, regular deflation after inspecting a segment and possibly using CO2 which, unlike air, is rapidly resorbed from the colon.
• Avoid excessive looping and the use of excessive shaft pressure.
• Particular care needs to be taken when colonoscopying patients with weakened walls (eg active colitis, toxic megacolon, ischaemic colitis and diverticulosis).

Safe practice –
Therapeutic perforation can be minimised by:-
• Thorough understanding of heat generation by electrocautery and
• the dangers of transmitting current to the contralateral wall
• avoiding/minimising use of ‘hot biopsy’ in the thinner right colon; cold snaring of small polyps in the right colon is now near universal practice.
• Increasing use of submucosal ‘saline lift’ for transverse and right colonic lesions (Grade C)

Haemorrhage
Significant bleeding is an unusual complication of diagnostic colonoscopy. It most frequently complicates snare polypectomy or hot biopsy and only rarely complicates cold biopsy.

Frequency:
In 11 studies summarised by Bowles, bleeding was reported in 0.001–0.24% of colonoscopies.

Risk factors:

Factors relating to blood coagulation
There is controversy about the risks of aspirin and non-steroidals.
• No prospective randomised trials to assess this risk; however, Shiffman et al(10) could find no evidence that these drugs, used in conventional doses, increased the risk of a significant bleed.
• In a prospective study of 694 patients(11) 46% of patients who had taken either aspirin or NSAID within a week of undergoing an endoscopic procedure. No patient undergoing pinch biopsy suffered a major bleed and 4/154 patients who underwent snare polypectomy suffered major bleeding. These were equally distributed between the NSAID and the control groups(13).
• Despite lack of evidence to implicate anti-platelet agents as a risk factor for bleeding including after polypectomy, some clinicians prefer to recommend discontinuation of these drugs 7–10 days prior to colonoscopy.

Warfarin is an independent risk factor. The reader is referred to the ASGE document ‘Guideline on the Management of Anti-coagulation and Antiplatelet Therapy for Endoscopic Procedures’ (Reference 9 –Section on Upper GI Endoscopy)

Factors related to the procedure
• Pinch biopsy – No evidence of increased risk of haemorrhage, even in patients on warfarin in the therapeutic range
• Polypectomy – Increased risk of bleeding after polypectomy. Highest risk with:-
  • Large, thick stalked and sessile polyps,
  • Poor technique causing premature mechanical transection prior to application of effective electrocoagulation

Clinical features:
• Bleeding is usually observed immediately after the polyp is excised, but delayed haemorrhage also occurs.
• An immediate haemorrhage usually presents as a slow ooze but can manifest as a brisk arterial spurt.
• Secondary haemorrhage usually occurs 1–14 days after polypectomy although secondary haemorrhage has been reported up to 30 days after polypectomy.
• Immediate haemorrhage appears to be more common when a blending or cutting current is employed whilst secondary haemorrhage is more commonly associated with the use of a coagulation current.

Treatment:
Most post-polypectomy bleeding is minor and both primary and secondary bleeding almost always stops spontaneously.
Primary Haemorrhage
If bleeding is recognised at the time of polypectomy, various haemostatic techniques have been described:

• A persistent oozing can be treated by repeat local electrocoagulation, local injection of 1:10,000 adrenaline or argon plasma photoagulation (grade C recommendation).
• If there is intense haemorrhage, try to gain haemostatic control of the haemorrhage before blood obscures vision.
  • Control is often quickly obtained by re-snaring the residual stalk and maintaining enough pressure to stop the bleed. Simple strangulation, maintaining snare closure for 10–15 minutes, is often effective without the need for further electrocoagulation.
  • If the stalk has been re-snared and bleeding recurs on releasing the snare, further electrocoagulation or adrenaline injection can be used to achieve haemostasis.
  • Clips or endoloops may also be used to gain haemostatic control.
  • If re-snaring is difficult, the injection of to 5–10 ml of 1:10,000 adrenaline solution into the submucosa adjacent to the stalk remnant can be highly effective.
  • If blood obscures the view of the bleeding point, infuse large volumes of water containing dilute topical adrenaline (5 ml of 1:10 000 adrenaline per 50 ml water) and consider repositioning the patient to gain a view of the bleeding site.
  • If significant bleeding persists, whilst deploying the above measures, make back up arrangements which include:
    • Alerting an experienced gastrointestinal surgeon
    • Contacting the interventional radiology team. If (rarely) arterial bleeding persists despite all other measures, selective arterial catheterisation and embolisation should be considered. This technique can however cause ischaemic colonic necrosis requiring surgery once haemorrhage has arrested.

Secondary haemorrhage
Patients presenting with a secondary haemorrhage should be admitted to hospital.

• Following resuscitation, most patients settle spontaneously. Intervention is required only rarely (grade III evidence).
• Treatment of persistent haemorrhage includes repeat colonoscopy in an attempt to inject or cauterise the bleeding point. Clips and endoloops may also be used in this setting.
• If bleeding is torrential or a direct approach is not possible, angiography may be helpful in defining the bleeding site. Whilst selective mesenteric vessel embolisation can be livesaving, it is important to remember that this procedure confers a high risk of bowel infarction.

Prevention of haemorrhage
Pre-procedure clotting studies before therapeutic colonoscopy. Good technique to reduce the potential for haemorrhage from large polyp stalks includes some or all of:-

• judicious use of maximum electrocoagulation
• the use of pre-polypectomy adrenaline injection
• nylon-loop or clip strangulation

VERY RARE COMPLICATIONS OF COLONOSCOPY
Case reports of rare complications include:

• glutaraldehyde-induced colitis
• colonoscope incarceration in an hernia
• splenic rupture and/or haematoma
• other extracolonic haemorrhages (liver, mesentery)
• volvulus of caecum, sigmoid or transverse colon
• pneumatic ileal perforation
• pneumatosis coli
• pneumomediastinum, pneumothorax

Prevention of Complications
Whom to Colonoscopy
Since colonoscopy carries the potential for significant harm to patients, the endoscopist must be aware of, recognise and take account of the following:-

• A constant awareness of the potential for harm.
• The extra risk associated with the sedation because of the usual combined use of a benzodiazepine and an opioid (usually pethidine and midazolam).
• The higher risk of death in:
  • Elderly patients with significant comorbidity
  • Patients with significant cardiovascular, respiratory, renal or widespread malignant disease.
• The higher than average risk of complications is where there is:
  • fulminant colitis
  • a history of radiation therapy for abdominal or pelvic cancer
  • a history of abdominal or pelvic malignancy
  • extensive adhesions from prior abdominal surgery
  • a bleeding disorder
  • anticoagulant therapy
  • a history of complications with intravenous conscious sedation
  • a known history of diverticulosis/diverticulitis
  • decompensated cardiorespiratory status
  • an uncooperative patient

In all of these groups of patients, alternative imaging such as double contrast barium enema, CT and CT colography should at least be considered as possible alternatives to colonoscopy as the first line investigation.

Safe Practice

• Full informed consent must be obtained before the procedure
• Sedation training is essential for all colonoscopists including recognition and management of the first signs of over-sedation before hypoxia manifests.
• Every colonoscopy should be performed by a fully trained operator – or by a trainee with close support.
• Colonoscopy should always be performed with care and compassion.
• Both endoscopy staff and patients need to be alert to the early manifestations of complications.
• Patients should be given clear instructions on how to respond to unusual symptoms following discharge from the endoscopy unit.

REFERENCES
6. Bowles CJA, Leicester R, Romaya C, Svarbrick E, Williams CB, Epstein O. A prospective study of colonoscopy practice in the UK: today are we
adequately prepared for national colorectal cancer screening for
12 Shiffman ML, Farrel MT, Yee YS. Risk of bleeding after endoscopic biopsy or polypectomy in patients taking aspirin or other NSAIDs. Gastrointest Endosc 1994; 40: 458–62
COMPLICATIONS OF ERCP

Dr Roger W Chapman

INTRODUCTION

Endoscopic retrograde cholangiopancreatography (ERCP) is the most complicated endoscopic procedure performed by gastroenterologists. Whilst it is a very rewarding therapeutic procedure endoscopically it is also the most hazardous. It requires specialist equipment and needs a long learning curve in order to develop competence. The undoubted therapeutic benefits of ERCP in the minimally invasive management of biliary and pancreatic disorders have to be weighed against a high rate of serious complications, when compared with other forms of therapeutic endoscopy. It should be remembered that:

- People who need ERCP the least are the most likely to develop complications
- Avoidance of marginally indicated ERCP is the best away to avoid serious complications (Ref 1)

COMPLICATIONS

What complications & why?

ERCP is associated with the same risks associated with all types of GI endoscopic procedures including adverse drug reactions, respiratory and cardiopulmonary complications. These generic risks of GI endoscopy are dealt with elsewhere. This section will be solely concerned with the specific complications associated with diagnostic and therapeutic ERCP.

The most common and important complications of ERCP and sphincterotomy are:

- Pancreatitis
- Bleeding
- Cholangitis / septicaemia
- Perforation
- Basket impaction

The overall incidence of complications depends on many factors. An overall complication rate after sphincterotomy is around 5%; 60% of these are mild, 20% moderate and 20% severe. The procedure-related death rate associated with sphincterotomy, whilst initially reported as being around 1% is now around 0.2% in the latest series.(ref 1,2) Evidence Grade I.

The commonest cause of post procedure death is cardiopulmonary, emphasising the need for attention to safety careful monitoring of sedation and analgesia during ERCP.

POST ERCP PANCREATITIS

How to recognise it after ERCP

- Elevated serum amylase occurs in up to 75% of patients after ERCP. However, only 3–10% of these patients have actual clinical acute pancreatitis defined as a clinical syndrome of abdominal pain and hyperamylasaemia requiring hospitalisation. It is important to realise that the amylase can rise to up to 600 IU/L, after a normal ERCP without the development of pain or other evidence of pancreatitis.
- The diagnosis of clinical pancreatitis is based on clinical and laboratory features, namely abdominal pain and elevation of serum amylase and/or lipase. Clinical symptoms usually develop within 24 hrs.
- Two large series have shown that pancreatitis accounts for >50% of the complications of endoscopic sphincterotomy (refs 3,4) Evidence Grade I. The overall incidence of post ERCP pancreatitis was 5.4%. The majority of cases were mild or moderate and only nine patients (0.4%) developed severe pancreatitis, one of whom died. Three patients required surgical treatment and one needed percutaneous drainage of a pseudocyst (ref 3) Similar results were found in another prospective post-sphincterotomy study in which acute pancreatitis developed in 4.7% of patients, severely in 0.6%. (Ref 4)

Why does it happen?

There are a number of factors which, either acting together or independently, may cause pancreatitis after ERCP:

- Traumatic injury from instrumentation of the Ampulla of Vater
- Hydrostatic injury from over injection of contrast medium
- Chemical or allergic injury from contrast medium
- Thermal injury from electrocautery current, causing oedema of the pancreatic orifice
- Enzymatic injury from reflux of intestinal contents.
- Infection - whilst bacterial injury is a possibility it is unlikely to play a major role in the pathogenesis

Grading & Incidence (Table 1)

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Abdominal pain with serum amylase at least three times normal at more than 24hrs after the procedure; requiring admission or postponement of planned discharge by two to three days</td>
<td>3–10%</td>
</tr>
<tr>
<td>Moderate</td>
<td>Abdominal pain with elevated amylase and hospitalisation of four to ten days</td>
<td>1–5%</td>
</tr>
<tr>
<td>Severe</td>
<td>Severe abdominal pain, elevated amylase and hospitalisation of more than ten days or a complication such as haemorrhagic pancreatitis, pseudocyst or peripancreatic fluid collection</td>
<td>0.1–1%</td>
</tr>
</tbody>
</table>

Risk Factors

The risk of pancreatitis is increased after therapeutic ERCP compared with diagnostic ERCP. Multi-variant analysis has shown that there are a number of parameters which are independent predictors of post ERCP pancreatitis:

- Operator related factors – low volume
- Patient related factors – higher risk of pancreatitis seen with:
  - Younger age,
  - Normal size bile duct
  - Normal bilirubin
  - Pre-existing pancreatitis
Technique related factors – higher risk of pancreatitis seen with:
• Difficult cannulations (prolonged, repeated cannulation attempts)
• Overfilling of the pancreatic duct causing opacification of parenchyma
• Repeated pancreatic contrast injections,
• Sphincter of Oddi manometry,
• Pre-cut sphincterotomy,
• Pancreatic sphincterotomy
• Biliary sphincteroplasty (ref 6) Evidence Grade I

Note – Independent predictors are additive (ref 2–5). For example a young non-jaundiced woman with suspected Sphincter of Oddi dysfunction with a normal size bile duct and a difficult cannulation without gallstones involves a risk of pancreatitis caused by the procedure of over 40%.

Management
• Fortunately, most episodes of ERCP pancreatitis are mild and only require a short hospital stay.
• The minority of patients who develop severe pancreatitis may require prolonged hospitalisation in an intensive care unit setting.
• The management of severe post ERCP pancreatitis is the same as that of severe pancreatitis from other causes.

Sensible strategies for prevention:
Technique: Scrupulous basic ERCP technique is very important to reduce the risk of post ERCP pancreatitis:
• Adequate training and competence of both endoscopist and endoscopy assistants
• Adequate case volume to acquire experience and maintain competence
• Avoidance of diagnostic ERCP when alternative and less invasive methods are available such as endoscopic ultrasound and MRCP
• Avoidance of cannulation of the pancreatic duct when not indicated
• Limiting cannulation time to avoid trauma to papilla
• Limiting injection number and volume of contrast to avoid pancreatic overfilling causing acinarization
• Selective use of electrocautery current with a sphincterotomy

Evidence Grade B

PHARMACOLOGICAL PROPHYLAXIS
Although a number of therapeutic agents have been assessed experimentally and clinical trials, none have gained universal acceptance. The agents which have been tried to reduce post – ERCP induced pancreatitis are shown below:
• Somatostatin
• Octreotide
• Gebaxate mesilate
• Interleukin 10
• Diclofenac
• Glyceryl trinitrate
• Antibiotics
• Calcitonin
• Glucagon
• Nifedipine
• Allopurinol
• Infusion of C1 inhibitor
• Secretin
• Low dose anticoagulation
• Intravenous cortico-steroids
• Afrinoton

Somatostatin
• Three controlled studies have shown a reduced incidence of post ERCP pancreatitis after the use of somatostatin.
• In one study, a single iv bolus of somatostatin given immediately after the diagnostic imaging but before therapy reduced the incidence of pancreatitis after therapeutic ERCP (ref 7).
• Further studies are needed to confirm the role, if any, for somatostatin in prophylaxis of post ERCP pancreatitis.

Use of a stent in the pancreatic duct for sphincter of Oddi manometry or pancreatic sphincterotomy

Table 1 Consensus Grading System for the major complications of ERCP and endoscopic sphincterotomy

<table>
<thead>
<tr>
<th>Complication</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bleeding</td>
<td>Clinical (ie not just endoscopic) evidence of bleeding, haemoglobin drop &lt;3g, and no need for transfusion</td>
<td>Transfusion (4 units or less) no angiographic intervention or intervention or surgery</td>
<td>Transfusion 5 units or more, (angiographic or surgical)</td>
</tr>
<tr>
<td>Perforation</td>
<td>Possible, or only very slight leak of fluid or contrast, treatable by fluids and suction for 3 days or less</td>
<td>A definite perforation treated medically for 4–10 days</td>
<td>Medical treatment for more than 10 days or intervention (percutaneous or surgical)</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Clinical pancreatitis, amylase at least three times normal at more than 24hr after the procedure requiring admission or prolongation of planned admission to 2–3 days</td>
<td>Pancreatitis requiring hospitalisation of 4–10 days</td>
<td>Hospitalisation for more than 10 days, or haemorrhagic pancreatitis, phlegmon, or psuedocyst, or intervention (percutaneous drainage or surgery)</td>
</tr>
<tr>
<td>Infection</td>
<td>&gt; 38oC for 24–48hrs</td>
<td>Febrile or septic illness requiring more than 3 days of hospital treatment or endoscopic or percutaneous intervention</td>
<td>Septic shock or surgery</td>
</tr>
<tr>
<td>Basket Impaction</td>
<td>Basket released spontaneously or by repeat endoscopy</td>
<td>Percutaneous intervention</td>
<td>Surgery</td>
</tr>
</tbody>
</table>

Table 2 prospective study of the complication rate for endoscopic sphincterotomy for bile duct stone (ref 12)

<table>
<thead>
<tr>
<th>Complication</th>
<th>All Patients (%)</th>
<th>Patients &lt; 60 years with ducts &lt; 9mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no.</td>
<td>1921</td>
<td>238</td>
</tr>
<tr>
<td>Patients</td>
<td>(%)</td>
<td>(%)</td>
</tr>
<tr>
<td>Complications</td>
<td>112 (5.8)</td>
<td>10 (4.2)</td>
</tr>
<tr>
<td>Mild</td>
<td>70 (3.6)</td>
<td>7 (2.9)</td>
</tr>
<tr>
<td>Moderate</td>
<td>26 (1.3)</td>
<td>2 (0.8)</td>
</tr>
<tr>
<td>Severe</td>
<td>12 (0.6)</td>
<td>1 (0.4)</td>
</tr>
<tr>
<td>Fatal</td>
<td>4 (0.2)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

Table 1

Table 2

November 2006

BSG Guidelines in Gastroenterology

• Previous post ERCP pancreatitis
• Sphincter of Oddi dysfunction

Evidence Grade I

Evidence Grade B
Octreotide
• This long acting somatostatin analogue does reduce the serum amylase after ERCP but has not been shown to reduce the incidence of clinical pancreatitis.
• The discrepancy between the results with somatostatin and octreotide, if real, may be due to the direct effect of octreotide which increases sphincter of Oddi basal pressure.

Gebaxate Mesilate
• Early controlled trials of this protease inhibitor suggested its prophylactic use before ERCP reduced the rate of clinical pancreatitis from 8% to 2%.
• As further studies did not confirm this, it remains unclear whether this is a useful prophylactic agent in ERCP (ref 8).

Interleukin 10 (IL-10)
• One small controlled study has shown that a single iv dose of this anti-inflammatory cytokine given half an hour prior to ERCP reduced the rate of pancreatitis (ref 9)

Diclofenac
• A single trial in 220 patients reported that a single rectal dose of 100mg of the nonsteroidal anti-inflammatory drug (NSAID) diclofenac, given after the procedure, reduced the rate of pancreatitis compared with placebo (ref 10)

In conclusion:
• There is no agent which has gained universal acceptance for use in prophylaxis of post ERCP pancreatitis.
• The reported promising results with some of the agents described all need confirmation.
• Whilst some pharmacological intervention may be reasonable in certain patients at high risk of pancreatitis (e.g. somatostatin in young patients with sphincter of Oddi dysfunction), cost effectiveness studies have not yet been performed to justify this practice.
• The main stay of prevention for post ERCP pancreatitis remains a properly trained and experienced endoscopist using good technique in the setting of an experienced unit (ref 11). Recommendation Grade C

POST ERCP BLEEDING

How to recognise it
• Diagnostic ERCP, when performed, has virtually no incidence of significant bleeding.
• Sphincterotomy accounts for virtually all significant post ERCP bleeding:
  • Advances in sphincterotomy techniques and experience have reduced the incidence of bleeding
  • Approximately 50% of bleeding complications occur and are recognised immediately after sphincterotomy – although there may be a delay of up to ten days before a significant bleed occurs.
  • The most recent studies have shown the incidence of significant bleeding post endoscopic sphincterotomy to be around 2% with a mortality rate of 0.1%. (ref 3)

Post ERCP bleeding can be graded as mild or moderate severe based upon a consensus definition:
• Mild – where there is clinical evidence of bleeding (ie not just endoscopic) associated with a haemoglobin drop of <3gms
• Moderate – need for transfusion of four units or less and no angiographic intervention or surgery
• Severe – transfusion of five units of blood or more, or requiring intervention either angiographic or surgical

Why does it happen?
A number of risk factors for post ERCP bleeding have been identified from retrospective and prospective studies (Ref 1–3, 5–7).

Patient factors – include:-
• Coagulopathy
• Anti-coagulation within three days of the procedure
• Child’s Grade C cirrhosis
• Renal failure and ongoing haemodialysis
• Bleeding during the procedure
• Bleeding as an indication for the procedure (i.e attempted haemostasis)
• Preceding cholangitis

Anatomical features – include:-
• Bilroth II partial gastrectomy
• Peri-ampullary diverticulum
• Stenosis of the ampulla of Vater
• Impacted common bile duct stone

Technical factors – include:-
• Length of the sphincterotomy made
• Extension of a previous sphincterotomy
• Uncontrolled sphincterotomy e.g. a rapid zipper cut
• Needle knife sphincterotomy
• Low case volume of the endoscopist and endoscopy unit.

Management of Post ERCP bleeding
Post sphincterotomy bleeding:
• Usually stops spontaneously
• Is rarely life threatening – except in patients with a bleeding diathesis.

Angiography or surgery is reserved for patients with refractory bleeding but the majority of patients can be managed with medical treatment and endoscopic therapy.

Endoscopic therapy
A number of endoscopic techniques can be used:
• Injection Therapies
  • Adrenalin – spray the area with adrenalin followed by an adrenalin injection if bleeding persists using diluted adrenalin 1/10,000 through a sclerotherapy needle (ref 12). Sclerosants – should be avoided in this situation.
  • Injection therapy with fibrant sealants – there is less experience with these agents
  • Haemoclip and electrocautery – have been used successfully in the treatment of post-sphincterotomy bleeding.
  • Complete the sphincterotomy despite bleeding if it is required for stone removal. This may also have a beneficial effect on allowing full retraction of a partially severed vessel.
  • Argon beam coagulation and balloon tamponade have also been used, although clinical evidence of efficacy is unclear. Recommendation Grade C.

Other therapies
• In refractory cases, angiographic treatment with embolisation can be effective. Surgery is reserved for patients in whom all other methods fail.
• Surgery is still sometimes required although the rate of surgery has declined considerably over the last decade

SENSIBLE STRATEGIES FOR PREVENTION OF ERCP BLEEDING

Risk factors
Identifying patients at risk:
**Complications of ERCP**

- All patients undergoing any form of ERCP should have a platelet count and INR measured prior to the procedure – preferably within the preceding 24hrs. As above, there is little risk of bleeding with diagnostic ERCP.
- It is considered that a platelet count exceeding 50,000 and a normalised ratio and INR of <1.2 is safe for sphincterotomy.
- There is no data to indicate that patients undergoing stent deployment or exchange alone (without any form of sphincterotomy) are at excess risk of bleeding even when coagulopathy is present.

**Sensible precautions before sphincterotomy:**
- Avoid Sphincterotomy in patients with severe coagulation disorders – correct these where possible.
- Take preventative measures, where possible, in patients with known platelet dysfunction e.g a prophylactic infusion of DDAVP in patients with renal failure on haemodialysis.

**Sphincterotomy and drugs that affect blood clotting:**
- Aspirin and NSAIDs – Sphincterotomy appears to be safe in patients taking these within three days pre or post sphincterotomy. However, some authorities recommend their discontinuation where possible.
- Low molecular weight or subcutaneous heparin or the newer anti-platelet drugs such as Clopidogrel. There is no data regarding patients taking drugs. As a general rule they should probably be stopped prior to the procedure.

**Warfarin:**
- Should be discontinued three to five days before planned sphincterotomy.
- A prothrombin time (INR) should be checked two hours prior to the procedure; if necessary fresh frozen plasma can be used to reverse the anticoagulation.
- Vitamin K should be avoided if at all possible. This is because of the delayed onset of action and because of the time it takes to re-establish therapeutic anticoagulation following the procedure.
- There is no evidence to guide the timing of re-starting warfarin therapy. Many endoscopists re-start it the same evening or within 48 hours whilst others wait for three days. If more urgent reinstitution of anticoagulation is needed (e.g. for metallic cardiac valve prostheses) intravenous heparin may be required to cover the interval. This is started some 6–12 hours after the sphincterotomy, a balance of risks.

**Recommendation Grade C**

**Technique**
Higher risk of bleeding after sphincterotomy is associated with:
- Low volume units and inexperienced operators.
- Electrocautery current –
  - It would appear that pure cut current may cause less risk of haemorrhage than a blended current at the lower power settings (30 Watts or less) but not at higher power settings.
  - The use of pure cut current is said to lower the pancreatitis rate but it may be associated with an increase of localised bleeding.
  - Some endoscopists therefore combine pure cut and blended current in sequence starting with pure cut and completing with blend (ref 13).

**Recommendation Grade C**

**POST ERCP SEPSIS**

**Why does it happen?**
Potential septic complications of ERCP include: ascending cholangitis, acute cholecystitis, infected pancreatic pseudocyst, liver abscess, peritonitis with bacterial infection following perforation of a viscus and less commonly endocarditis.

**Ascending cholangitis –**
- The most common infective complication of ERCP, ascending cholangitis:
  - Usually occurs after incomplete drainage of an infected, obstructed biliary system; 87% of patients with incomplete biliary drainage develop sepsis due to cholangitis.
  - Is the most frequent and early indication of an occluded biliary stent.
  - Usually develops within twenty-four to seventy-two hours after ERCP in at risk patients.
- It is presumed that the mechanism is raised biliary pressure causing a biliary/venous reflux and hence an access for bacteria to the bloodstream. Retrograde cannulation without drainage is another potential entry mode for pathogenic organisms.

For clinical purposes, post ERCP cholangitis is classified as either:
- Mild – temperature greater than 38°C for twenty-four to forty-eight hours
- Moderate – febrile or septic illness requiring more than three days hospital treatment or endoscopic or percutaneous treatment
- Severe – septic shock or requirement of surgery

**Acute cholecystitis –**
- The incidence of acute cholecystitis is probably less than 0.5% of patients and may be related to the introduction of non-sterile contrast media through a poorly emptying gall bladder.
- Patients at higher risk may be those with gall bladder stones and a patent cystic duct after common bile duct stone clearance.
- Treatment includes hospitalisation with bed rest and antibiotics and consideration of cholecystostomy and cholecystectomy.
- Prevention may be achieved by post ERCP prophylactic antibiotics in high risk patients.

**Pancreatic sepsis –**
This is uncommon following ERCP; it usually occurs in patients with a pseudocyst which becomes infected following injection of the pancreatic duct.

**PREVENTION & TREATMENT OF SEPTIC COMPLICATIONS**

**Technique**
Techniques to avoid septic complications following ERCP include:
- Proper cleansing and disinfection of endoscopes prior to the procedure.
- Sterile radiographic contrast media.
- Prompt endoscopic decompression when biliary obstruction is demonstrated.
- If definitive drainage cannot be obtained, then temporary drainage with a nasobiliary tube or stent is mandatory so that definitive can be performed.
• If endoscopic drainage cannot be achieved, then percutaneous or surgical manoeuvres should be considered and undertaken without delay
  Recommendation Grade B

PREVENTION WITH ANTIBIOTIC PROPHYLAXIS

• Antibiotic prophylaxis is widely considered to be indicated in selected (as opposed to all) patients (ref 14–16).
• Antibiotic prophylaxis should be used in:
  • Patients at risk of infective endocarditis
  • Patients with a known or strongly suspected pancreatic pseudocyst
  • Patients with large duct biliary obstruction, especially if jaundice is present – treatment should be continued if adequate biliary drainage has not been established
• Using the above guidelines, a number of studies have shown that prophylactic antibiotics given before ERCP have demonstrated efficacy (ref 14–15)
• Standard treatment in patients who are fit to take oral therapy is considered to be oral Ciprofloxacin 750mgs two hours prior to the procedure and patients who cannot take oral therapy intravenous Cefuroxime 750mgs is the treatment of choice in the United Kingdom.
  Evidence Grade I ;Recommendation Grade A.

POST ERCP PERFORATION

Why does it happen?

Three types of perforation complicating ERCP are recognised:
• Retroperitoneal duodenal perforation – this is the most common and usually occurs after a sphincterotomy that has been extended beyond the intramural portion of the duct.
• Perforation of the bile ducts – this usually occurs following dilatation of biliary strictures, inadvertent portal cannulation and the insertion of guide wires; it is commoner in malignancy.
• Free abdominal perforation of the duodenum or jejunum – this is rare, usually occurring in the setting of abnormal anatomy such as a Bilroth II gastrectomy or a duodenal stricture. Perforation of peri-ampullary diverticula either by the scope or at sphincterotomy has been described but it remains a rare complication. Gastric and oesophageal perforation and pneumomediastinum without perforation have also been described after ERCP and sphincterotomy.

Risk factors

Patient related risk factors:
• Sphincter of Oddi dysfunction
• Dilated common bile duct
• Peri-ampullary diverticula (possibly)

Procedure related risk factors:
• Performance of sphincterotomy
• Long duration of procedure
• Biliary stricture dilatation
• The risk of retroperitoneal perforation is increased with pre-cut sphincterotomy and larger sphincterotomies

Grading

Post ERCP retroperitoneal perforation can be graded as mild, moderate or severe:
• Mild – associated with a slight leak of contrast, treated by fluids and suction for three days or less.
• Moderate – any definite perforation treated medically for four to ten days.
• Severe – medical treatment for more than ten days or requiring intervention, either percutaneous or surgical.

How to recognise post ERCP perforation

Retroperitoneal perforation:-
• Clinically significant perforation has been reported in 0.5%-5.2% of sphincterotomies (ref 17–18). However, retroperitoneal air has been observed in up to 30% of patients after sphincterotomy who are asymptomatic (ref 17). This (often chance) finding in asymptomatic patients does not normally require intervention; patients can be observed and treated conservatively with intravenous fluids, nil by mouth and antibiotics.
• It is usually diagnosed by radiological evidence of air or contrast in the retroperitoneal space – it cannot be easily determined endoscopically. The amount of air present does not indicate the size of the perforation or correlate with the severity of the complication – it merely reflects the amount of air inserted after the perforation had occurred.
• Abdominal CT scan is the most sensitive way of detecting perforation.

Free abdominal perforation:-
• Free peritoneal perforation occurred in 0.1% of patients undergoing diagnostic ERCP and free abdominal perforations occurred in about 0.09% (ref 17).
• Is associated with classical signs of abdominal pain and peritonitis.

Management

• Oesophageal, free abdominal gastric, jejunal or duodenal perforation usually require surgery.
• Retroperitoneal perforation, in contrast, is usually successfully managed by a conservative approach and patients rarely require surgical intervention (ref 18)
  • They should be kept nil by mouth on intravenous fluids and treated with nasogastric and nasoduodenal suction (Ref 18)
  • Patients who develop retroperitoneal collections can be treated with percutaneous drainage.
• The prognosis of patients with perforation depends on the rapidity with which it is recognised and patient comorbidities.
  Evidence: Grade II

Sensible strategies to prevent perforation at sphincterotomy

• Ensure proper orientation at the papilla, cutting the sphincter between eleven and one o’clock
• Progress the sphincterotomy step by step using small short bursts of current to avoid the ‘zipper’ (uncontrolled, long) cut. Some diathermy current generators have specific settings to prevent this. Beware that previous inflammation (causing fibrosis at the ampulla) may make the cut difficult to initiate.
• Tailor the length of the sphincterotomy to the size of papilla, bile duct and stone seen on cholangiography
• Be very wary performing sphincterotomy when the papilla is small, flat and flush against the duodenal wall (i.e has no or minimal intramural bile duct above the papilla). A very limited sphincterotomy is all that can be performed.
  Recommendation: Grade C.

IMPACTION OF RETRIEVAL BASKETS

Prior to the introduction of mechanical lithotripsy impaction of a wire basket around a large calculus was a major complication. This has been reduced by mechanical lithotripsy although the complication can still occur (ref 19–20)

Why does it happen.

The basket usually becomes trapped at the ampulla within the intra duodenal part of the common bile duct, although
Impaction can occur at any site within the biliary system e.g. above a more proximal biliary stricture.

**Risk factors**
- Large stones; irregular shape; hard (non-crumbly) stones
- Small sphincterotomy
- Distal narrowing of common bile duct “duct disproportion”
- Segmental dilatation of intrahepatic ducts e.g. Caroli’s syndrome

**Management of basket impaction**
If the ‘high disimpaction manoeuvre’ fails, the endoscopist will need to use one or more of the following:-
- Mechanical lithotripsy, using “rescue lithotripter”
- To extend the sphincterotomy
- Intra or extra corporeal lithotripsy
- Surgery – as the final option

**Sensible Strategies for Prevention**
- Careful consideration of options with large stone or narrowed distal bile duct
- Tailoring the sphincterotomy size to fit the stone size
- Avoid fully closing the basket around a large stone
- Consider using balloon if possible

**Recommendation Grade C**

**REFERENCES**
19. Freeman ML. Adverse outcomes of ERCP. Gastrointest Endoscopy 2002; 56:5273
COMPLICATIONS ASSOCIATED WITH THE PLACEMENT OF PERCUTANEOUS ENDOSCOPIC GASTROSTOMY

Dr Paul O’Toole

INTRODUCTION

The insertion of a percutaneous endoscopic gastrostomy (PEG) is a routine procedure in nearly all GI endoscopy units in the UK. However, there is a high incidence of serious complications following this procedure.

This guideline is confined to the description of complications that occur when the decision has been taken to insert a PEG. It does not attempt to cover the critical question of patient selection for the procedure. There is an increasing view that selection of all patients should be a function of a multi-disciplinary Nutrition Team.

It cannot be emphasised too strongly that correct selection of appropriate patients for PEG insertion is the single most effective strategy for preventing serious complications of this therapeutic procedure.

The insertion of enteral tubes under endoscopic guidance is a rapidly developing area of therapeutic endoscopy with techniques such as percutaneous jejunostomies and even colostomies. This guideline is however confined to the majority procedure of PEG which is performed on a frequent basis by the majority of endoscoping gastroenterologists.

INCIDENCE OF COMPLICATIONS

Minor complications are underestimated in many series because of limited follow-up but probably occur in about 20%.

• Major complications arise in about 3%, the exact incidence depending on the predominant patient population. PEGs performed for malignant disease generally result in more complications [4].
• Direct procedure-related mortality rates vary between 0.7–2%.
• Thirty-day mortality rates of 10–13% are reported in some series [1][2], and up to 28% in others.
• Complication rates are similar irrespective of the PEG technique used whether this is the common Pull (Ponsky) method, Push (Sachs-Vine) method or the direct introducer (Russell) method.

Since mortality following PEG insertion is closely related to the severity of the patient’s underlying illness, such high death rates indicate inappropriate patient selection. The involvement of a specialist nutrition team in decision making can reduce the number of PEG’s placed inappropriately [3].

PEG complications can be divided into immediate (those arising from the procedure itself), early (those occurring within the first 4 weeks) and late (those arising after the first few weeks, when the gastro-cutaneous fistula is fully established).

IMMEDIATE COMPLICATIONS

RESPIRATORY

Patients undergoing PEG insertion are at particular risk of respiratory complications:

• Endoscopy in the supine position carries an increased risk of aspiration, especially when the gag reflex is impaired by neurological disease.
• Neurological impairment further increases the risk of respiratory depression due to sedation.
• Patients with oropharyngeal cancer may be at risk of airway obstruction during intubation.

Prevention

• Careful pre-procedure assessment, avoidance of excessive sedation and attention to oropharyngeal suction will reduce the risk of aspiration and respiratory suppression. (II)

If the airway is felt to be compromised:

• Consider a non-endoscopic gastrostomy technique – or
• Delay the procedure until after a tracheostomy is established.

BLEEDING

• Significant bleeding from the abdominal wall or stomach is rare.
• More serious bleeding can result if another intra-abdominal structure is punctured inadvertently during needle passage.
• Minor oesophageal bleeding is occasionally seen as the internal retention disc is dragged through when using the pull method.

Prevention

• Ensure that the stomach is well localised before needle passage. This will reduce the risk of damage to another structure (text box 1)[5] (I)
• Coagulopathy should always be corrected prior to PEG placement. A platelet count of 100,000 or more and an INR < 1.4 are recommended. (III)

1 Localising the stomach

Taking time and care in selecting a site for needle puncture avoids many PEG complications. Ideally all three of the following conditions should be met:

• Jabbing with a finger at the correct puncture site should produce a clear and well-localised gastric wall indentation seen on endoscopic view.
• Positioning the endoscope tip at the point of indentation and boosting the light should produce a clear transillumination.
• While passing the 21G needle with local anaesthetic, draw back on the syringe. Air bubbles should appear in the syringe at the same moment as the needle tip appears in the stomach. If air is aspirated but the needle tip cannot be seen, another viscus may have been punctured. Take the needle out completely and select an alternative puncture site.
PERITONITIS
Peritonitis immediately after the procedure usually indicates damage to another viscus:

- Structures most likely to be damaged are small bowel and transverse colon. The risk is probably increased if there has been previous upper abdominal surgery resulting in adhesions.
- If the trocar needle completely traverses colon or small bowel on its way to the stomach, there may be no indication of a problem initially but complications will arise later (see below)[6].

If the patient moves or retches violently, the trocar needle can become dislodged from the stomach before the string or guidewire has been passed through it:

- This creates the potential for gastric contents to leak into the peritoneal cavity.
- If this occurs, complete the procedure with a second needle pass; this generally averts peritonitis because the initial puncture site usually lies within the region of stomach that is pulled up against the anterior abdominal wall.

Management
- Severe peritonitis occurring in the first few hours after PEG placement (before feeding has commenced) usually requires an exploratory laparotomy.
- If the patient is unfit for surgery or the signs are less severe, conservative therapy may be attempted (see below). (III)

Prevention
- Ensure the stomach is well localised prior to needle passage (text box 1) (I)
- Secure the trocar needle as soon as possible after it enters the stomach using a snare passed through the endoscope. (III)

INFECTION
- Peristomal infection is a frequent complication of PEG placement. It appears to be more common in patients with diabetes mellitus.
- In severe cases an abdominal wall abscess may develop
- Necrotising fasciitis is a rare complication caused by a rapidly spreading infectious process involving the fascia and subcutaneous tissues [7]. It is recognised by oedema and marked erythema around the PEG site, usually with surgical emphysema (crepitus), accompanied by fever and systemic upset.

Management
- Less severe infections will often respond to regular wound cleaning and local antisepsis.
- Severe peristomal infections require systemic antibiotics, guided by the results of swabs sent for microbiological culture.
- Necrotising fasciitis requires aggressive treatment with urgent surgical debridement and broad-spectrum antibiotics. (II)

Prevention
Prophylactic antibiotics – shown to reduce the risk of early infectious complications (for example, 1.2g co-amoxiclav given iv 30 minutes before gastrostomy insertion)[8][9][1]. This remains the advice despite increasing problems with nasopharyngeal carriage of MRSA – see below.

- Patients with oral carriage of MRSA will inevitably become colonised when PEGs placed by the “pull” or “push” methods.
- This may result in MRSA peristomal infection, an increasing problem not prevented by prophylactic antibiotics.
- Attempts to clear the organism from the mouth prior to PEG placement may be worthwhile – but this has not been demonstrated in clinical trials. (III)

Other Measures
- Avoid excessive tightening of the external fixator – may cause local ischaemia and encourage infection[10]. (II)
- Avoid proton pump inhibitors wherever possible on the basis that gastric acidity will inhibit micro-organisms. (III)

EARLY DISPLACEMENT
- The gastro-cutaneous fistulous tract becomes established within two weeks of PEG placement but may take up to 4 weeks to mature.
- If the internal retention disc (“bumper”) becomes displaced within this period, the tract may break down and the stomach falls away from the anterior abdominal wall. Gastric contents can then leak into the peritoneal cavity.
- Displacement may be complete (i.e. the tube comes out completely) or partial (i.e. the “bumper” displaces from the stomach but remains within the peritoneal cavity).
- Partial displacement is more dangerous because it may not be recognised and feeding continued, leading to gross contamination of the peritoneal cavity.

Management (See text box 2)
- If gross peritoneal contamination has occurred, laparotomy is usually indicated.

2 Management of Early Post-Procedure Abdominal Pain
- Generalised peritonitis in the first few hours after the procedure indicates damage to another organ or viscus. Laparotomy is usually indicated.
- Localised pain and guarding around the PEG site is common in the first few days. In severe cases, suspend feeding and give broad-spectrum intravenous antibiotics as a precaution until things settle. Inspect site for peristomal infection and take swabs as necessary.
- Perform a PEGogram if tube position uncertain.

Air under the diaphragm does not indicate that a complication has arisen. Benign pneumatoperitoneum is common and may persist for several days.
- Severe pain during feeding suggests partial displacement. Suspend feeding and perform a PEGogram. If displacement is confirmed, deciding between conservative or surgical treatment depends on the patient’s fitness and the degree of peritoneal contamination suspected. CT scan may be helpful in determining this (see text).
- If severe pain or peritonitis is present, but the PEGogram does not demonstrate displacement, a CT scan should be performed to exclude peritoneal leakage without displacement.
Complete displacement without peritonitis. The PEG should be replaced as quickly as possible. This is probably best done under fluoroscopic guidance. Endoscopic replacement is reported to be feasible but efforts should be made to keep air insufflation to a minimum.

Complete displacement with peritonitis but no obvious gross peritoneal contamination.
- A CT scan may be helpful to confirm that there is not a large amount of free intraperitoneal fluid.
- If little/no free fluid, conservative treatment may be attempted, especially if the patient is a poor candidate for surgery[11].
- Replace the tube, give broad-spectrum antibiotics and keep the stomach empty. Nutritional support, preferably by NJ tube, may be required. (II)

Prevention
- Avoid traction on the tube during the first 4 weeks after placement.
- Confused patients, who are likely to pull at their tube, are best fitted with a non-traction removable PEG. (III)

PERITONEAL LEAKAGE WITHOUT DISPLACEMENT
- The defect created in the gastric wall during PEG placement will usually seal snugly around the tube during the process of gastro-cutaneous fistula formation.
- Rarely, the fibrous fistula will fail to develop properly and the gastric puncture enlarges such that there is room for stomach contents to leak around the tube into the peritoneal cavity.
- This is a rare cause for peritonitis during the first two weeks.
- This complication appears more likely if there are patient factors resulting in impaired wound healing e.g. extreme old age, use of long-term corticosteroids and very severe malnutrition.
- Mechanical ventilation has been suggested as another risk factor
- A PEGogram will show that the “bumper” remains in the stomach and may not show any leak (see text box 2).

Management
- Laparotomy with repair of the gastric wall defect.

Prevention
- In severe malnutrition, a period of naso-gastric feeding prior to PEG placement may help.
- Avoid endoscopy during the week after PEG placement as this may interrupt track formation. If unavoidable, use minimal air insufflation [4]. (III)

ASPIRATION PNEUMONIA
- PEG feeding does not abolish the risk of aspiration pneumonia in patients with an unsafe swallow.
- The true incidence of this is unknown, but aspiration pneumonia is a common cause of mortality following PEG placement.
- Source of Aspiration
  - Patients may continue to aspirate oral secretions
  - Feed may reflux up from the stomach to the mouth (gastro-oesophago-tracheal aspiration).

Recognition
- The presence of feed in the mouth can be confirmed by testing with Glucostix.
- Adding colouring to feed is not recommended because it risks introducing infection.

Prevention
- Patients should be sitting upright or in a semi-recumbent position (propped up at 30O or more) during feeds and should maintain this position for 30 minutes after feeding. (II)
- Avoid large boluses and use continuous or intermittent pump feeding (III)
- Prokinetics may help – but are untested in clinical trials.
- If reflux continues despite these manoeuvres, consider post-pyloric feeding by adding a jejunal extension to the PEG or performing a direct percutaneous jejunostomy[12]. (II)

LATE COMPLICATIONS

LATE DISPLACEMENT
- If displacement occurs after the tract has matured, peritoneal leakage will not result.
- The track will close very quickly however (within 24 hours) so prompt efforts should be made to preserve it.

Management
- Either – Preserve the track by replacing the gastrostomy tube as soon as possible and securing it with tape.
- Or – Pass a Foley urinary catheter through the stoma. This should be replaced by a proper gastrostomy tube as soon as possible because a Foley catheter balloon will not survive long in the presence of gastric acid.

Note – Carers should not attempt to pass anything through the stoma unless it is certain that the tract is properly mature (see below). (I)

Prevention
- Avoid traction-removable devices in patients who are confused and likely to pull at their tube. (III)

LEAKAGE
- Leakage of gastric contents around the gastrostomy tube results in chemical burns to the surrounding skin.
- This occurs when repeated circular movement of the tube enlarges the stoma.
- It is common in patients who are severely unwell from other causes or in the terminal stages of illness.

Management
- Protect the skin with a barrier cream (such as Cavilon,) and leave open to the air as much as possible.
- Reduce acidity of gastric juice with a proton pump inhibitor; prokinetics are advocated by some.
- Temporary tightening of the external fixator may help; long term, this is likely to cause pressure necrosis of the gastric wall and may exacerbate the problem.
- Replacing the tube with one of greater diameter rarely helps because the stoma eventually becomes even larger. Replacing the gastrostomy with a snugly fitting low profile device (“button”) sometimes helps. (III)

Prevention
- Rotate tubes gently periodically to ensure maintenance of a healthy fistula.
- Avoid excessive rotation or movement of the tube (III)

HYPERGRANULATION
- Excessive granulation around the stoma is uncomfortable, may bleed and makes cleaning more difficult.
- Factors leading to its formation are poorly understood.
- It frequently recurs after treatment.
Management
• Steroid ointments such as Sofradex, are commonly used although this is an unlicensed indication.
• Silver nitrate cautery may help and cautery with the argon plasma coagulator has also been described. (III)

TUBE DYSFUNCTION
• PEG tubes may become blocked or split. Tube blockage is more commonly due to medication than feed.
• Polyurethane tubes generally outlive silicon tubes but they can split if repeatedly kinked or indented at the same place.

Management
Blocked PEG tubes:
• Can usually be cleared by flushing with warm water using a small volume (2ml) syringe.
• If this fails, an alkaline solution of pancreatic enzymes can be used.
• Passing guide-wires down the tube to clear blockages is not recommended unless performed under fluoroscopy.

Split PEG Tubes:
• If PEG tube is split far enough away from the skin, it can be cut below the split and the feeding port reassembled on the end of the shortened tube.
• If the split is too close to the skin a replacement tube is necessary.

Prevention
• Always use specially formulated liquid feed.
• Avoid very sticky or particulate medication.
• Flush the tube with at least 50ml of cooled boiled water after every feed and all medication.
• Acidic fruit juice (e.g. pineapple juice) or fizzy soda water help clear adherent fatty deposits but may cause tube degradation.
• Move the C-clamp up and down the tube so that it does not leave its indentation at the same place every time.
• Leave the C-clamp open once the spigot is in place.
• Avoid kinking the tube.

BURIED BUMPER
• Buried bumper is a particular problem with PEG tubes that have a silicon internal retention disc.
• The disc embeds within gastric mucosa which then overgrows the bumper and eventually obstructs the passage of feed.
• Buried bumper is suspected if the PEG cannot be pushed in easily.

Management
• A deeply buried bumper may require surgical removal.
• Other approaches have been described:
  • Using a needle knife papillotome to cut away the overgrowing granulation tissue
  • Stiffening the tube with a dilatation balloon or Savary guidewire and then pushing it back into the stomach.

Prevention
• Slacken off the external fixation device a little once the track is mature.
• Make further adjustments if the patient puts on weight and the thickness of the anterior abdominal adipose tissue layer increases.
• Rotate the tube and push in gently once a week.

OBSTRUCTION
Obstruction may occur in two situations:
• Detachment of internal bumper (spontaneous or deliberately cut) – this will drop into the stomach and will usually pass through the gastro-intestinal tract without a problem. Rarely it can become impacted resulting in obstruction.
• Detachment of external fixation device – if this occurs, it is possible for an intact bumper to be carried down into the duodenum and cause obstruction – from where it can be surprisingly difficult to pull back.

Prevention
• Avoid the “cut and drop” method of PEG removal in patients with intestinal strictures or in those who would not be fit for laparotomy in the event of obstruction[13].(III)
• Keep the external fixator in place.

TRACK DISRUPTION
Occurs most frequently at tube replacement:
• If excessive force is used during tube replacement, even a mature gastro-cutaneous fistula will break down.
• Disruption reported most often during placement of “mushroom-retained” buttons. (These devices are now used less often as a consequence.)

Prevention
• Do not remove a PEG or pass a replacement tube through a gastrostomy stoma unless you are confident the fistulous tract is well established (2–4 weeks after placement).
• Avoid excessive force when passing replacement devices.
• Check that the any replacement device is correctly positioned by aspirating gastric contents and checking the pH with litmus paper.
• Do not inflate a gastrostomy balloon until you are sure the tube is well within the gastric lumen.
• If any doubt, perform a PEGogram before starting feeding.

COLO-CUTANEOUS FISTULA
• If a bowel loop (usually colon) overlies the stomach, the trocar needle may inadvertently pass through it during PEG placement.
• If this is not recognised, the PEG tube will end up traversing the colon and a gastro-colo-cutaneous fistula is created.
• This may present early with symptoms of colonic perforation or obstruction.
• More often it remains unrecognised until the gastrostomy tube is changed and the replacement tube becomes positioned in the interposed colon or small bowel.

Management
• The colo-cutaneous fistula will usually close spontaneously once the tube is removed.

Prevention
• Always localise the stomach as clearly as possible before PEG placement.
• Use a “safe track” technique (see Text box 1) (I)

TUMOUR IMPLANTATION
• PEG’s are commonly placed pre-operatively for patients with oropharyngeal cancer.
• If the pull or push method is used, the PEG bumper may come into contact with the tumour on its way through the mouth.
• Cancer cells can be picked up and implanted into the PEG tract presenting as metastatic tumour mass at the PEG site between 3 and 16 months after the procedure[14].
Prevention

• Use a direct introducer or radiological Seldinger technique for pre-operative PEG placement in oro-pharyngeal cancer.

REFERENCES


7 Cave DR, Robinson WR, Bratschi EA. Necrotising fasciitis following percutaneous endoscopic gastrostomy. Gastrointest Endosc 1986;32:294–296


These guidelines have been prepared by the British Society of Gastroenterology. They represent a consensus of best practice based on the available evidence at the time of preparation. They may not apply in all situations and should be interpreted in the light of specific clinical situations and resource availability.