Upper GI (Oesophago-gastric) Pathway Board (NSSG) Guidelines

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Agreement

Co-director of NSSG -

Name: Prof Muntzer Mughal
Organisation: London Cancer
Date agreed: 27 November 2013

Name: Mr David Khoo
Organisation: London Cancer
Date agreed: 27 November 2013

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Contents
1. Epidemiology, Aetiology, Prevention ................................................................. 5
  1.1 Oesophageal cancer ......................................................................................... 5
  1.2 Gastric cancer ................................................................................................ 5
2. Diagnosis ............................................................................................................. 6
  2.1 Symptoms ......................................................................................................... 6
  2.2 Endoscopy ........................................................................................................ 6
3. Staging .................................................................................................................. 7
  3.1 Staging methods ............................................................................................... 7
  3.2 Preoperative TNM Staging .............................................................................. 8
  3.3 Restaging after neoadjuvant therapy .............................................................. 8
4. Pathology ............................................................................................................. 9
  4.1 Oesophageal cancer ......................................................................................... 9
  4.2 Gastric cancer .................................................................................................. 9
5. Treatment ........................................................................................................... 10
  5.1 Aims of treatment ........................................................................................... 10
  5.2 Treatment Process .......................................................................................... 10
6. Preoperative Assessment .................................................................................... 10
  Fitness for surgery ............................................................................................... 10
7. Oesophageal Resection ....................................................................................... 12
  7.1 Selection for surgery for oesophageal cancer .............................................. 12
  7.2 Surgical approach to oesophageal cancer .................................................. 12
  7.3 Radicality of resection ................................................................................... 13
  7.4 Anastomosis ................................................................................................... 14
  7.5 Routine postoperative care ............................................................................ 14
  7.6 Postoperative complications ......................................................................... 14
  7.7 Mortality .......................................................................................................... 15
8. Gastric Resection ................................................................................................ 15
  8.1 Curative resection ........................................................................................... 15
  8.2 Longitudinal extent of gastric resection ..................................................... 15
  8.3 Oesophago-gastric junction (OGJ, junctional) carcinomas ....................... 16
  8.4 Radical endoscopic resection ....................................................................... 17
  8.5 Palliative surgery for gastric cancer ............................................................. 17
  8.6 Morbidity and mortality .................................................................................. 17
  8.7 Rare gastric neoplasia .................................................................................... 17
9. Chemotherapy and Radiotherapy ........................................................................................................... 18
  9.1 Oesophageal cancer .............................................................................................................................. 18
  9.2 Gastric and oesophago-gastric junction cancer ..................................................................................... 18
10. Palliative Treatment ................................................................................................................................... 18
  10.1 Access to palliative care consultant .................................................................................................... 18
  10.2 Oesophageal cancer ........................................................................................................................... 18
  10.3 Gastric cancer and oesophago-gastric junction cancer .......................................................................... 19
  10.4 Endoscopic palliative treatment for advanced disease ........................................................................ 19
  10.5 Physical, psychological and spiritual care .......................................................................................... 20
11. Follow Up .................................................................................................................................................. 20
12. Data Collection and Audit ....................................................................................................................... 21
13. Appendices ............................................................................................................................................... 22
  13.1 Referral Guidelines – for Primary Care ............................................................................................... 22
  13.2 Referral Guidelines for Specialist Centre ............................................................................................. 22
  13.3 Classification of oesophago-gastric tumours .................................................................................... 23
  13.4 TNM Staging and stage grouping (6th Edition) .................................................................................. 25
  13.5 TNM-7 (December 2009) .................................................................................................................. 27
  13.6 Clinical Information Required on Specimen Request Form .............................................................. 28
Introduction

These guidelines are based on the widely accepted guidelines published by Association of Upper Gastrointestinal Surgeons of Great Britain and Ireland, the British Society of Gastroenterology and the British Association of Surgical Oncology (2002). Similarity to these guidelines in structure and content is therefore deliberate but replication of detail has been avoided. They have been adapted for use in the North Central / East Thames Cancer Network with specific local implementation. It is intended that these local guidelines are not in contradiction to the nationally promulgated version. These guidelines are intended to be more didactic and succinct than the national guidelines, which are appended for reference. Operational detail is not the remit of these clinical guidelines.

1. Epidemiology, Aetiology, Prevention

1.1 Oesophageal cancer
1.1.1 The incidence of adenocarcinoma of the lower oesophagus and cardia is increasing. Treatment of obesity and gastro-oesophageal reflux may reduce the incidence of this condition and these treatments should be available. A diet rich in fruit and vegetables is to be encouraged in public health programmes.

The cause of this increase in incidence is unknown, but may be related to increasing population obesity and reflux disease. Prolonged gastro-oesophageal reflux leads to Barrett’s metaplasia and dysplasia and cancer.

1.1.2 The incidence of squamous cell carcinoma of the oesophagus has been shown to relate to smoking and excessive alcohol intake. Public health programmes should emphasise this.

1.2 Gastric cancer
1.2.1 Distal gastric adenocarcinoma is becoming less common, but cardia carcinoma is rapidly increasing in incidence. Helicobacter pylori eradication cannot be recommended as a preventive measure as it may decrease the incidence of distal cancer but may increase the incidence of cardia cancer. Diets rich in fruit and vegetables and low in preserved and salt rich foods are to be encouraged in public health programmes.

Reduction in duration and severity of gastro-oesophageal reflux by medical or surgical intervention may be effective in preventing cardia as well as lower oesophageal cancer. Eradication of Helicobacter is warranted for treatment of dyspepsia and peptic ulceration.
2. Diagnosis

2.1 Symptoms

Oesophago-gastric cancer symptoms have a large overlap with those of benign or functional disease.

2.1.1 The cardinal presenting symptom of oesophageal cancer is dysphagia. Older patients with symptoms of prolonged gastro-oesophageal reflux are at high risk and may harbour an early cancer. Whilst the former clearly should have investigation, the latter should have investigation prior to prolonged treatment.

2.1.2 Dyspepsia with alarm symptoms may indicate gastric cancer and warrants investigation (Appendix 13.1).

There are few community-based studies indicating the combination of factors that may help to predict pathology. The word “dyspepsia” is ambiguous, and the concept of a “symptom provoked by eating” or more specific concepts i.e. “feeling full after small meals” may be better.

2.1.3 Emergency admission is a common mode of presentation to hospital.

A significant number of new diagnoses present as emergencies to acute medical or surgical admission with haematemesis, anaemia, difficulty with eating or cancer cachexia.

2.1.4 Barrett’s oesophagus surveillance may pick up early oesophageal cancer.

Patients with newly diagnosed Barrett’s oesophagus and who are fit to withstand potential surgery should be offered surveillance. Diagnosis requires the visual endoscopic appearance or the demonstration of specialised (intestinal) columnar epithelium. Four-quadrant biopsy at 2 cm intervals in the Barrett’s segment should be undertaken at 2 yearly intervals or more depending on the degree of dysplasia. The demonstration of adenocarcinoma indicates surgery. High-grade dysplasia requires the agreement of 2 independent specialist pathologists and is treated either endoscopically or surgically.

2.2 Endoscopy

2.2.1 Access to endoscopy should be made easy. Government guidelines on referral criteria will be adopted (Appendix 13.1).

Awareness of individuals’ risk factors is necessary to facilitate urgent referral. Screening of all referrals will result in exclusion of patients without alarm symptoms or who are not in a high-risk group from urgent investigation. This is necessary when the waiting time for investigation is so high, and urgent procedures need to be prioritised. Urgent 2-week wait endoscopy referrals are thus separated from “open access” referrals. The age of 55 has been set for patients at high risk i.e. with new or continuous symptoms since onset.

2.2.2 Endoscopic tumour description should be exact with regard to the site, measured as a distance from incisors and also in respect of anatomical landmarks in particular the Z line and oesophago-gastric junction. The length, width and morphological appearance should be recorded.

Recording of endoscopies should conform to high standards to prevent multiple procedures. Descriptive terms such as published by the Japanese Endoscopy Society should be applied (Appendix 15.2). The Siewert classification of junctional tumour site is vital to the definition of whether there is an oesophageal or gastric tumour. This has important consequences for staging of M status. Tumours of Siewert Type 1 should be considered as oesophageal tumours and the remainder as gastric tumours. (Chromoendoscopy should be explored for low grade dysphagia for second endoscopy.)
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2.2.3 Endoscopic biopsy should include at least 8 biopsies of any lesion and should include brush cytology of stricturing oesophageal lesions or any lesion that is difficult to biopsy. Greater sampling of suspicious lesions reduces the probability of false negative histology.

2.2.4 Barium contrast versus endoscopy

Direct GP referral for radiology should be prevented. Early lesions are likely to be missed. This technique of imaging is not a preferred diagnostic method as endoscopic biopsy is required in every case.

3. Staging

3.1 Staging methods

All patients should have accurate staging of local and distant disease to allow well-informed management decisions. Except in those who refuse treatment or who have obvious co-morbidity precluding surgical intervention, these modalities are mandatory.

3.1.1 Endoscopy will have to be repeated as a staging procedure where the original diagnostic endoscopy does not contain adequate information as in 0 It would be preferable to have this staging undertaken at the original endoscopy to save a repetition for the patient.

3.1.2 Spiral computerised tomography (CT) Scanning is required in all cases as the fundamental staging modality.

Spiral contrast enhanced prone CT of thorax, abdomen and pelvis with 5 mm collimation should ideally be used. Cardia lesions should be scanned with the stomach distended with 400 ml water. Distal stomach tumours should be scanned with the patient prone.

3.1.3 PET/CT (positron emission tomography/CT) should be considered to stage all patients being considered for radical oesophageal or upper gastric resection or for radical loco-regional radiotherapy after all other staging tests are favourable.

There is superior detection of occult distant and nodal metastases compared with CT alone. SUV measurements of (18)FDG uptake may be predictive of response to chemotherapy and may have prognostic value. It is limited by false positive findings and sufficiency of state-of-the-art CT, EUS and laparoscopy.

3.1.4 Endoscopic ultrasound (EUS) is required for the local disease staging of oesophageal cancers including junctional tumours where there is no evidence of distant metastases.

Either radial or linear scanning should be used. Only 70% of oesophageal tumours can be fully staged with a conventional endoscopic ultrasound probe and this can be circumvented by the use of a slim probe

3.1.5 Laparoscopy is required in patients being considered for surgery other than mid or upper oesophageal tumours or distal gastric tumours in which a palliative surgical procedure is contemplated.

Liver and peritoneal disease may be missed by all other modalities.

3.1.6 Indeterminate liver lesions should undergo MRI screening.

Other imaging techniques, including abdominal ultrasound, magnetic resonance imaging, and bronchoscopy should be considered as adjuncts.
3.2 Preoperative TNM Staging

The measured staging of oesophago-gastric tumours is critical to the management decision for surgery. The process aims to determine whether a tumour can be resected with curative intent, and there is a prognostic value which should inform decision-making. Feedback of the pathological staging of resected specimens is required to improve the value of these tests.

3.2.1 T staging of oesophageal tumours with CT is unreliable other than for mediastinal invasion. T staging of oesophageal tumours requires endoscopic ultrasound that can identify the component layers of the oesophageal wall.

CT can suggest invasion of local structures, but this information is insufficient to determine resectability. Overall CT accuracy is 50% whereas EUS is 85-90% accurate. Understaging is more common.

3.2.2 T Staging of stomach tumours can be with CT and gastric water contrast.

Adequate gastric distension with water can help distinguish T1/2 and T3 and T4. Endoscopic ultrasound will distinguish T1 from T2.

3.2.3 N Staging of oesophagus and stomach tumours with CT is important in respect of distant nodes not amenable to imaging by endoscopic ultrasound scanning and outside the limits of radical resection. Endoscopic ultrasound detects local nodes and can define coeliac node status. Needle biopsy should be required to confirm malignant infiltration when a decision for surgery is contingent on the diagnosis. PET/CT is the optimum modality for detecting nodal involvement.

Size is the only feature on CT suggesting malignant infiltration of a node. Hypoechoic, greater than 1cm nodes, rounded and with well-defined margins on endoscopic ultrasound are indicative of involvement.

3.2.4 M Staging combines the use of CT, PET/CT and endoscopic ultrasound, but should be supported by other modalities (13.4.1)

The importance of the definition of tumours at the junction as either gastric or oesophageal is highly important (13.3.1). What may be regional nodes in gastric are considered metastatic for oesophageal and vice versa. The definition of M1a includes regional nodal spread, crossing the divide between N staging and M staging. M1a includes the presence of cervical nodes and coeliac nodes in oesophageal cancer.

3.3 Restaging after neoadjuvant therapy

Progression of disease after neoadjuvant chemotherapy suggests a poor prognosis and may preclude surgery. Similarly, a complete response may lead to a decision to refuse surgery by a patient. The limitations of imaging should be recognised particularly where there is endoscopic and clinical evidence of regression.

3.3.1 A CT scan of thorax, abdomen and pelvis should be undertaken to detect the presence of new distant metastases and local disease progression.

3.3.2 Endoscopic ultrasound cannot distinguish residual tumour from fibrotic changes, but if the question is resectability (i.e. less than T4), EUS can be of value.

3.3.3 Laparoscopy has proved its worth locally in the detection of new disease and progression or regression of local disease and may be repeated in those patients in whom it was originally indicated

3.3.4 (18)FDG PET scanning should be used to detect interval metastases.
4. Pathology

4.1 Oesophageal cancer

4.1.1 Precursor lesions should be assessed against a background of repeat biopsy

Oesophageal squamous dysplasia indicates malignant potential and high-grade dysplasia suggests malignant transformation has already occurred.

Barrett’s oesophagus with intestinal metaplasia may lead to malignant transformation. Low-grade dysplasia may lead to high-grade dysplasia but may also regress. High-grade dysplasia may lead to malignancy. Regenerative atypia in healing areas may be confused with high-grade dysplasia. Disagreement occurs among pathologists over the appearance of low-grade dysplasia.

4.1.2 Biopsy and cytology reporting of oesophageal cancer should be undertaken by an experienced pathologist

4.1.3 Surgical specimen reporting for oesophageal cancer should be reported according to the Royal College of Pathologists minimum dataset. Both TNM 7 (13.5) and its predecessor, TNM 6 (13.4.1), should be used to preserve historical compatibility.

The features included in this dataset have recognised prognostic significance. In addition, node groups should be reported according to the Japanese Cancer Society Guidelines (13.3.2). It should be noted that subdiaphragmatic nodes are as important as mediastinal nodes.

4.2 Gastric cancer

4.2.1 Precursor lesions should be assessed against a background of repeat biopsy amongst multiple pathologists (at least 2).

Type 3 gastritis and incomplete intestinal metaplasia are associated with progression to dysplasia and follow up should be advised to clinicians and MDT. High-grade dysplasia may lead to malignancy. Regenerative atypia in healing areas may be confused with high-grade dysplasia. Disagreement occurs among pathologists over the appearance of high-grade dysplasia, which in repeat biopsies is likely to indicate adenocarcinoma.

4.2.2 Surgical specimen reporting should be according to the Royal College of Pathologists minimum dataset. Both TNM 7 (13.5) and its predecessor, TNM 6 (13.4.2), should be used to preserve historical compatibility.

**In addition, node groups should be reported according to the Japanese Cancer Society Guidelines (13.3.2).**

4.2.3 Oesophago-gastric junction cancer should be carefully reported as to site according to the Siewert classification (13.3.1)

Type I lesions may spread caudally to coeliac nodes and cephalad to mediastinal nodes as well whereas Type II and III lesions metastasise almost exclusively in a caudal direction.
5. Treatment

5.1 Aims of treatment

Treatment of oesophago-gastric cancer is for the purpose of relieving suffering and possibly achieving cure and should be at all times caring and compassionate.

5.2 Treatment Process

5.2.1 A patient-centred approach is imperative, so that an ideal treatment choice should not override the wishes of the patient.

5.2.2 Multidisciplinary team meeting discussions are required for all new diagnoses of cancer and for all treatment decisions.

Expert review is required of all clinical data, including clinical findings, endoscopy, imaging and pathology findings.

5.2.3 Responsibility for patient care should nevertheless remain with the consultant in charge of a particular treatment episode.

In the case of emergency admission or incidental diagnosis by a non-team consultant, an appropriate consultant of the upper gastrointestinal cancer team should assume patient care.

6. Preoperative Assessment

Fitness for surgery

There are no reliable indices for the assessment of whether a patient is fit to survive a complex major oesophago-gastric resection. Most indices and scoring systems are only robust for comparing outcomes of groups of patients. Therefore, responsibility for the assessment of fitness is assumed by the anaesthetist, surgeon and physician in close collaboration.

6.1.1 Assessment scores

- An ECOG score of 2 or less should be considered for surgery
- American Society of Anesthesiology (ASA) scores of 3 or less should be considered for surgery

6.1.2 Past medical history including ischaemic heart disease, pulmonary dysfunction, cirrhosis and diabetes predispose to poor outcome

Medication should be optimised prior to surgery

6.1.3 Social habits such as smoking should be ascertained at the time of diagnosis and patients should be encouraged to give up smoking and offered support to do so prior to any anaesthetic.

Operative risk is reduced significantly if patients stop smoking at least 3 months prior to surgery.

6.1.4 Preoperative investigations should be directed to where there is a clinical indication.

Respiratory function should be assessed in those with pre-existing lung disease with baseline blood gases on air, pulmonary function tests and chest X ray. A FEV1 of more than 1.3 is required for one lung anaesthesia.

Cardiovascular function may be assessed by a simple exercise stress test, i.e. climbing stairs, but as a minimum should include a resting ECG and may require a stress ECG, echocardiography and thallium scanning.

Nutritional deficiency is very common and a BMI of less than 18.5 or less than 90% predicted or a low serum albumin predispose to increased risk.
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6.1.5 Age per se is not an indication to deny curative surgery, but this is an independent risk factor in surgery.

6.1.6 Cardiopulmonary exercise stress (CPX) testing should be considered for all patients but more so in whom co morbidity might otherwise preclude surgery.

This investigation also gives lung function studies. The performance of the patient assists in the level of postoperative care.

Preoperative preparation

6.1.7 Nutritional support and advice should be provided for all patients particularly when there is difficulty with eating.

Hyperalimentation is not indicated. However oral supplements should be encouraged during the staging period.

6.1.8 In patients with dysphagia to liquids, laser treatment to the tumour bulk when appropriate can alleviate the symptom. Tube feeding is indicated in advance of surgery if laser fails. Failure of nasogastric feeding necessitates a surgical jejunostomy tube.

6.1.9 Psychological status. Psychological assessment tool should be offered.

Everyone involved in the care is responsible for identifying the need and providing psychological support to the patients at any stage. Depending on the stage of illness this may need to be provided by Upper GI nurse, clinicians, community Macmillan nurses etc. There should be provision to formal counselling and a named member of MDT to arrange this.

6.1.10 Informed consent should be obtained by the surgeon carrying out the procedure or by somebody capable of doing so, ideally in outpatients in advance of surgery.

This should include detailed and clear description of the procedure and what to expect during postoperative recovery. There should be clear explanation of the risk of death and morbidity including pulmonary complications and anastomotic leakage. Patients and relations should be provided with written information well in advance of the procedure and should be actively encouraged to assimilate and reflect on the information provided and ask questions.

6.1.11 Thromboembolic prophylaxis should be given to all patients because of the increased risk in a patient with cancer undergoing major and lengthy surgery and should be continued for 21 days. An IVC filter should be placed in advance of surgery in those who have had DVT or PE during neoadjuvant chemotherapy or in the preceding 6 months.

This should including low molecular weight heparin at increased dosage and calf compression stockings. Additionally, pneumatic compression may be used intra-operatively. Low molecular weight heparin preparations should be avoided preoperatively within 12 hours when epidural analgesia is considered.

6.1.12 Antibiotic prophylaxis should be given to all patients

Prophylaxis of wound and deep sepsis requires an antibiotic such as Cefuroxime and Metronidazole intravenously at induction and after four hours if surgery is continuing.

6.1.13 Blood cross matching should be undertaken for all resections.

Two units of blood should be cross-matched for oesophageal resection and two units for total gastric resection. Transfusion should be avoided wherever possible.
7. Oesophageal Resection

7.1 Selection for surgery for oesophageal cancer

7.1.1 Surgery for oesophageal cancer should only be undertaken exclusively with curative intent. The resection rate is expected to be about 20%.

The principle is that if all loco-regional neoplastic tissue can be removed a worthwhile period of survival can be achieved and possibly cure with a good quality of life. Minimal access palliative procedures are investigative modalities that may have a role to play in the future. A resection rate of about 20% appears to be a reasonable expectation by British standards.

7.1.2 Tumour stages earlier then T4 are suitable for consideration of surgery either as single or multimodality treatment.

Results from surgery alone are excellent for mucosal and T1N0 tumours, T1a (localised to mucosa) and T1b (limited to lamina propria) and high-grade dysplasia in Barrett’s metaplasia. In view of the MRC OEOII trial results any tumours more advanced than T1N0 should be considered for neoadjuvant therapy (9.1.1). T4 disease is usually inoperable, but if the involved structure is dispensable eg diaphragmatic crura, the patients are operable and should be considered for resection after neoadjuvant chemotheraphy. They may, alternatively, be suitable for radical chemoradiotherapy. Any metastasis diagnosed preoperatively precludes operative intervention.

7.1.3 Histology: Both adenocarcinoma and squamous cell carcinoma are suitable for surgical resection.

However, there is good evidence that all squamous cell carcinoma may be treated by chemoradiotherapy but selected cases with lower third lesions or where the target is more than 10cm in length (therefore not amenable to radical radiation) should have surgery.

7.1.4 Premalignant lesions such as high grade dysplasia should be referred for specialist endoscopic treatment.

7.2 Surgical approach to oesophageal cancer

This should take into consideration the site of the tumour, the fitness of the patient and surgical skills.

7.2.1 Transthoracic 2-field oesophago-gastrectomy should be performed for mid and lower third and Type I (and possibly II) junctional tumours

The standard approach for resectable oesophageal carcinoma in the UK is a two stage Ivor-Lewis oesophago-gastrectomy. This involves a laparotomy, preparation of gastric tube, gastric lymphadenectomy and right thoracotomy for oesophageal resection. A further neck incision may be indicated to achieve adequate proximal clearance for more proximal lesions. These patients may have mediastinal lymph node metastases, so this approach has the advantage of easier access for a mediastinal lymphadenectomy.

7.2.2 Transhiatal Oesophagectomy is appropriate as an alternative surgical option.

This technique has been associated with a lower morbidity from surgery and may be appropriate in patients with carcinoma in situ or T1 stage cancer. Compared to the Ivor-Lewis approach a mediastinal lymphadenectomy is more difficult to achieve. However, such lymphadenectomy has not shown clear survival advantage and some surgeons may achieve dissection of lower mediastinal lymph nodes with this approach. The majority of surgeons today carry out this procedure under direct vision. Because of the nature of the approach good longitudinal clearance is nearly always achieved. No clear advantage has been demonstrated consistently with transhiatal oesophagectomy but it has a place in early lower oesophageal tumours and Type1 junctional tumours and high-grade dysplasia in Barrett’s oesophagus.
This technique is not suitable for middle third tumours because the resection may be hazardous. Nor is it suitable for squamous cell carcinoma, which should have a complete lymphadenectomy. (7.3.3)

7.2.3 Left thoracoabdominal oesophago-gastrectomy is an alternative to Ivor-Lewis oesophagectomy and provides an alternative approach for junctional cancers (Type II) when an extended total gastrectomy may be required.

7.2.4 Laparoscopic and thoracoscopic mobilisation may be considered in conjunction with 7.2.1.

These modalities of surgical access allowing more rapid recovery and improved quality of life in the convalescent period and reduce chest complications.

7.3 Radicality of resection

The objective of surgical resection is to remove all neoplastic tissue (R0 resection) as this has been shown to improve survival. Surgical resection is the only modality that has been consistently shown to achieve cure especially for adenocarcinoma.

Any resection less than R0 i.e. R1 or R2 is likely to lead to locoregional recurrence.

7.3.1 Because there is a possibility of longitudinal submucosal spread, margins should be long within the limits of the conduit length and the proximal extent of the tumour. Although surgery is planned in view of preoperative staging information, occasionally the plan may need to be changed on the basis of operative findings. This may involve thoracotomy or extended transhiatal dissection following hiatal division for type2 or 3 junctional carcinoma, or the addition of a cervical incision for mid oesophageal carcinoma.

In such cases patients should be informed of these possibilities while obtaining consent.

7.3.2 The radial resection margin should be complete at all stages in the dissection.

Usually this not a major issue as radial margin can be cleared in standard Ivor Lewis type procedure. However especially for type 2 junctional tumours special attention may need to be paid to incorporate the crura in the resection. Special attention will be needed for T3 lesions.

7.3.3 Lymphadenectomy for oesophageal cancer is undertaken to improve the chance of complete resection and to improve staging to benchmark survival

The survival advantage separate from avoidance of stage migration is unclear. Two-field lymphadenectomy is the standard followed in the UK. This can be performed without increasing operative morbidity or mortality. This dissection involves the upper abdominal nodes i.e. the right and left cardiac, lesser curvature, left gastric, hepatic and splenic nodes and also the thoracic nodes i.e. the Para aortic along the thoracic duct, Para oesophageal, right and left pulmonary hilar, and along the tracheal bifurcation. At present para tracheal nodes are not dissected by most Western surgeons.

7.3.4 The choice of conduit for oesophageal resection is the stomach.

This achieves best functional results and has less early complication rates. In the absence of stomach colon may be used. Prevertebral placement of the stomach tube is preferable to retrosternal or subcutaneous route.
7.4 Anastomosis

7.4.1 The technique of anastomosis should avoid ischaemia of the ends without tension and with accurate apposition of the epithelial edges.

There is no clear evidence that one method of anastomosis is superior to the other with respect to anastomotic leakage. Both manual and stapled anastomosis may be carried out depending on convenience.

7.4.2 The site of anastomosis is dictated by the resection required.

No clear advantage of thoracic or cervical anastomosis has been established in terms of mortality. Cervical anastomosis is considered safer by some while others feel it is possible to have mediastinitis even with cervical anastomosis and recommend bilateral chest drains after transhiatal resection.

7.5 Routine postoperative care

7.5.1 The immediate post operative location of care for the first 24 hours should be in an environment which has a trained nursing ratio of at least 1:2 and with facilities for oxygen saturation, blood gas, central venous pressure, arterial pressure, non invasive blood pressure, electrocardiograph, urine output and fluid infusion monitoring. It should have facilities for active resuscitation including wall oxygen, suction, intubation, and cardiopulmonary resuscitation. If the patient is independent on all organ systems, step down may proceed.

7.5.2 The step-down location of care for the subsequent inpatient stay should be in a designated surgical ward.

This area requires a trained nursing ratio of at least 1:5. Wall oxygen and suction is required and oxygen saturation monitoring should be available.

7.5.3 Ventilatory cardiovascular or renal support should be available (in Intensive Care) if required in the event of a complication.

7.5.4 Enteral nutrition should be introduced within 24 hours of surgery by jejunostomy.

Nutrition should be introduced by mouth from day 3-5 post surgery. Liquid supplements should be followed by solids. There is evidence of improved respiratory function and reduced postoperative infections associated with a shorter hospital stay from this intervention.

7.5.5 Drains should be removed as soon as possible only after instructions from the surgical team. After the first 24 hours a drain volume of less than 100ml is sufficient to remove a chest drain.

7.5.6 Mobilisation of patients should occur within 48 hours of surgery.

7.5.7 Analgesia involves a combination of thoracic epidural opiate analgesia, paravertebral block and patient controlled opiate analgesia supplemented with intravenous paracetamol and non-steroidal anti-inflammatory drugs.

7.6 Postoperative complications

7.6.1 Pulmonary complications may be avoided by early extubation with adequate analgesia with chest physiotherapy and mobilisation. Treatment will be with broad-spectrum antibiotics but then with agents directed at the sensitivities of organisms cultured from the sputum. Pleural collections should be drained if large and causing symptoms.

Microbiological consultant opinion will be sought.
Chest infection is a major cause of morbidity and mortality. Atelectasis and subsequent pneumonia are caused by painful abdominal and thoracic incisions. Impaired lymphatic drainage and diaphragmatic incisions are contributory factors.

7.6.2 Anastomotic leakage may be avoided by meticulous technique. Postoperative management should be directed to promote good tissue perfusion. If a leak is suspected a water-soluble contrast study or CT with luminal contrast is indicated urgently. A high index of clinical suspicion should occur in the first 10 days after surgery and a gastroscopy may still be required to exclude a leak in the absence of contrast leak.

The clinical anastomotic leak rate should be less than 10%. Water-soluble contrast radiology can be used to confirm the presence and degree of clinical leakage. A false negative study must be considered where clinical suspicion is high.

Early disruption (2-3 days) is uncommon and may require re-exploration.

Late disruption (5-9 days) is the most common and can be treated successfully by aggressive conservative measures with antibiotics, jejunal feeding and pulmonary drainage.

Disruption of stomach suture line or gastric necrosis presents dramatically and needs urgent re-exploration. Gastric necrosis may require exteriorisation of the oesophagus, debridement and late colon interposition.

7.6.3 Chylothorax, if significant, should be treated by early re-exploration and ligation of the thoracic duct. This complication may be prevented by routine ligation of the thoracic duct at the diaphragm during surgery.

This manifests as drainage of turbid fluid through the chest drain. Initial conservative management is justifiable but drainage of greater than 10ml/kg/day beyond the 5th post-operative day should prompt re-exploration.

7.6.4 Recurrent laryngeal nerve palsy should be avoided.

This should be rare, but is more common after upper oesophageal dissections. Pulmonary complications may ensue and tracheostomy may be required.

7.6.5 Benign anastomotic stricture may develop after a few weeks but responds to multiple gradual dilatation

7.7 Mortality

The in hospital operative mortality rate for oesophageal resection averages less than 5%.

8. Gastric Resection

8.1 Curative resection

8.1.1 The resection rate of gastric cancer is about 20%

The resection rate in the UK is 20% for gastric cancer. The principle of surgery is that gastric cancer presents as a locoregional disease with late distant metastases. Therefore, locoregional surgery is the treatment of choice and the results are dependent on resectability and stage at presentation. Even with extensive staging investigations only 30-50% of resections are truly “curative.

8.2 Longitudinal extent of gastric resection

The site of the tumour and stage dictate the margin and type of resection that is possible.
8.2.1 Total gastrectomy should be performed for all potentially curable cancers of middle or proximal gastric body. This procedure facilitates lymphadenectomy.

8.2.2 “Extended” total gastrectomy may be offered to patients with Type II or III junctional tumours. A significant amount of oesophagus can be involved in these tumours. The majority of these patients will have lymph node metastases below the diaphragm, so an adequate lymphadenectomy in the gastric territory is appropriate but with an adequate (5-10 cm) length of oesophagus. These patients should be considered to have had a partial oesophagectomy in view of the extensive length of oesophagus taken.

8.2.3 Subtotal gastrectomy is appropriate for pyloric and antral tumours. An 80% distal gastrectomy with removal of the lesser curve should be performed, with the aim of obtaining a 10 cm proximal margin.

8.2.4 Proximal gastrectomy may lead to severe bile reflux, but has a place in some Type II lesions and Type I lesions. It is a rarely performed procedure as an alternative to a total gastrectomy. A jejunal interposition reduces the risk of symptomatic bile reflux.

8.2.5 Reconstruction by Roux en Y jejunal reconstruction is ideal. However, a side-to-side gastrojejunostomy is suitable for subtotal gastrectomy.

8.2.6 Radial margins of resection of gastric cancer should be clear. Adjacent organ resection to reach a situation of complete tumour clearance (R0) is worthwhile where involvement (T4) is suspected if the patient is fit enough to withstand radical resection.

8.2.7 Lymphadenectomy may cure patients with lymph node metastases and should be performed. Systematic modified D2 lymphadenectomy should be attempted where the patient is fit enough to withstand radical surgery. Notwithstanding the lack of evidence of benefit from randomised clinical trials of D2 versus D1 lymphadenectomy, the International Gastric Cancer Association Consensus view (1997) and the prevailing UK mood is that every patient with curable gastric cancer should have modified D2 gastrectomy. This involves en-bloc removal of N1 and N2 nodes. The greatest advantage seems to be to the patients with stage II and IIIA disease. D2 gastrectomy allows more accurate staging and stage migration is avoided and some patients will have had a curative resection which would otherwise not have been undertaken. For distal cancers there is little rationale for splenic hilar dissection (Station 10) or splenic artery lymph nodes (station 11) as they are very infrequently involved. Removal of the lesser sac peritoneum may potentially prevent gastric bed recurrence for serosa positive (T3) carcinoma but this does not prevent recurrence in the general peritoneal cavity.

8.2.8 Resection of spleen and distal pancreas is unlikely to benefit the patient and may result in increased (usually pulmonary) morbidity. These organs are removed routinely in the radical D2 lymphadenectomy, but the associated morbidity appears prohibitive without much evidence of improved nodal clearance. The modified D2 gastrectomy does not involve these resections.

8.3 Oesophago-gastric junction (OGJ, junctional) carcinomas

In reality, it may not be always possible to distinguish between type II and Type III junctional cancers preoperatively. There should be no hesitation to resect the distal 5cm of oesophagus. Accurate endoscopic staging, sometimes during surgery is vital.

8.3.1 Type I lower oesophageal adenocarcinoma, often arising in Barrett’s metaplasia, should be treated by oesophagectomy with two-field lymphadenectomy.
8.3.2 Type II junctional carcinomas may be treated as oesophageal or gastric tumours. They are treated with an extended total gastrectomy or an oesophagectomy.

8.3.4 Type III junctional cancers are treated as proximal gastric cancers. They commonly involve proximal stomach and total gastrectomy with lymphadenectomy is indicated.

8.4 **Radical endoscopic resection**

For unfit patients endoscopic mucosal resection may be considered for the treatment of early T1 (<5mm) tumours and for high-grade dysplasia.

8.5 **Palliative surgery for gastric cancer**

8.5.1 Palliative resection may be appropriate for some patients with gastric outlet obstruction or intractable bleeding from the tumour bed, and each case should be considered for quality of life benefit and prognosis.

There is evidence of quality of life benefit from gastric resection for palliation, particularly where the tumour burden is low and disease performance status is good. Minimal access procedures have a particular benefit, as return to normal activity can be rapid. Prevention of local complications and anaemia are particular benefits of resection above bypass. In medically fit patients, even total gastrectomy can be contemplated.

8.5.2 Gastric bypass is appropriate for distal obstructing tumours

This lends itself to a minimal access procedure and can give excellent quality of life.

8.6 **Morbidity and mortality**

The in-hospital operative mortality should be less than 5% for total and subtotal gastrectomy.

8.7 **Rare gastric neoplasia**

The biology of these tumours requires a very different approach to that for adenocarcinoma.

8.7.1 Type 1 and 2 Carcinoid tumours of the stomach rarely metastasise and may be observed. Localised Type 3 tumours should be managed as for adenocarcinoma. Metastatic tumours should be assessed in a unit specialising in neuroendocrine tumours.

8.7.2 Gastrointestinal stromal tumours of the stomach can be treated by local resection with adequate margins.

These tumours can be distinguished from leiomyomas by the expression of a growth factor receptor with tyrosine kinase activity, termed KIT which can be detected by immunohistochemistry. They metastasise by blood borne and transcoelomic spread. Radical gastric excision is not indicated and segmental laparoscopic excision is appropriate. Advanced disease is effectively controlled by the KIT inhibitor imatinib. Mutational analysis should be considered to assess potential response to KIT inhibitors.

8.7.3 Lymphoma of the stomach should not be managed by resection as chemotherapy is first line therapy.
9. Chemotherapy and Radiotherapy

9.1 Oesophageal cancer

9.1.1 Neoadjuvant chemotherapy should be considered for all patients with operable carcinoma of the oesophagus and junctional tumours with T stage more than 2 or with nodal disease. (7.1.2)

There is clear evidence from MRC OEO2 trial of significant survival benefit for neoadjuvant chemotherapy without increased mortality or morbidity. In that trial, Cisplatin/5FU was given on day 1 and 22 with surgery on day 50. Similar results are shown in the MAGIC study, which considered gastric cancers along with lower oesophageal and junctional tumours.

9.1.2 Neoadjuvant radiotherapy has no current role in operable patients with oesophageal carcinoma in the UK

9.1.3 Neoadjuvant chemoradiotherapy may be offered in highly selected cases.

9.1.4 Radical chemoradiotherapy should be offered for patients with localised squamous cell carcinoma and some inoperable adenocarcinomas.

9.1.5 Adjuvant chemoradiotherapy may be offered to patients with positive pathological resection margins.

9.2 Gastric and oesophago-gastric junction cancer

9.2.1 Neoadjuvant chemotherapy should be offered to all patients with gastric cancer with a T stage of more than 2 or with nodal disease.

The recent MAGIC study indicates increased disease-free and overall survival with neoadjuvant chemotherapy using ECF/ECX 3 cycles preoperatively and 3 cycles postoperatively.

9.2.2 Neoadjuvant chemoradiotherapy should not be offered outside clinical trials.

Some early benefit in terms of disease free and overall survival has been reported. Further investigation is required to ascertain whether this early benefit is durable.

9.2.3 Adjuvant chemoradiotherapy may be used in postoperative patients with adverse prognostic factors

Patients considered for this would not have received MAGIC protocol and would have a heavy nodal burden and other adverse factors in the resection specimen.

10. Palliative Treatment

10.1 Access to palliative care consultant

In view of the fact that the majority of such patients have advanced disease, access to a palliative care team is very important. Patients with inoperable disease and patients with operable disease unsuitable for radical intervention should be treated using similar principles. Individual need has to be the guiding principle.

These patients should have the same multidisciplinary approach to decision making as those with being treated with curative intent. Early liaison with primary care and Macmillan team has to take place.

10.2 Oesophageal cancer.

Quick and durable restoration of swallowing is the aim of therapy
10.2.1 Palliative chemotherapy and radiotherapy in locally advanced disease may be offered provided the patient is fit to withstand it. With such an approach, the response rate is high and there is a survival benefit.

10.2.2 Palliative chemotherapy may be offered with ECF/ECX. Patients with advanced adenocarcinoma or squamous cell carcinoma should be considered for palliative chemotherapy. This may be used as an adjunct to endoscopic palliation.

Epirubicin (60 mg/m^2) day 1, Cisplatin (60 mg/m^2) day 1 and 5FU (ECF) (200 mg/m^2) days 1-21 repeated every 3 weeks for 6 cycles. This regime provides reduction in the number of endoscopic sessions for these patients. The use of Paclitaxel should remain in the setting of clinical trials. Alternatively, Capecitabine (Xeloda) may be used.

10.2.3 Palliative chemotherapy or radiotherapy as standalone treatment is not standard treatment in dysphagic patients.

Chemotherapy and radiotherapy and endoscopic treatment should be used in conjunction.

10.3 Gastric cancer and oesophago-gastric junction cancer

10.3.1 Palliative chemotherapy should be offered to every patient, fit to withstand it, with advanced carcinoma of stomach or junctional cancer. There is clear evidence of significant quality of life and survival benefit with chemotherapy.

10.3.2 First line. ECF or ECX (Epirubicin, Cisplatin, infusion of 5FU or Capecitabine) over 6 cycles is the standard first line palliative chemotherapy regime.

10.3.3 Second line. Docetaxel or Irinotecan/de Gramont or Irinotecan/Mitomycin C may be used where ECF has not produced the desired response.

10.3.4 Downstaging chemotherapy prior to surgery. Fit patients with advanced disease (stage IIIa, IIIb) may be offered chemotherapy bearing in mind that complete tumour resection rate using such an approach is low.

ECF may be used. Survival is related to complete clinical or pathological response and complete tumour resection.

10.3.5 In patients with advanced intestinal type adenocarcinoma, the tumour HER2 status should be determined as these patients may be suitable for treatment with Herceptin.

10.4 Endoscopic palliative treatment for advanced disease

10.4.1 Endoscopic palliation is the preferred strategy in dysphagic patients. A combination of techniques may need to be used to achieve rapid and durable restoration of swallowing.

10.4.2 Laser tumour obliteration may be used to palliate dysphagia. There is a 90% success rate with two treatment sessions in terms of initial relief of dysphagia. The complication and mortality rates are low. However, laser is contraindicated with fistulating disease and for lesions crossing the cardia.

10.4.3 Laser therapy should be followed by radiotherapy. Addition of radiotherapy following initial laser treatment prolongs dysphagia free interval.

10.4.4 Injection should not be used except for bleeding tumours or where the tumour is unsuitable for intubation (situated too close to the cricopharyngeus muscle).

There is a significant complication rate and recurrent dysphagia is common.
10.4.5 Intubation (stent) should be the preferred initial modality where complete relief of dysphagia with one treatment session is desired.

It can be used as an adjunct in recurrent dysphagia following initial laser therapy. Stenting is unsuitable for very proximal tumours, but ideal in the presence of a fistula. Self-expanding metal stents are safer, more effective and associated with reduced hospital stays. Tumour ingrowth results in recurrent dysphagia and may need further stents or laser therapy. Metal stents should not be used where radical radiotherapy is contemplated or has taken place.

10.4.6 Argon plasma coagulation (APC) and photodynamic therapy (PDT) are not recommended for advanced disease palliation.

There are insufficient data to recommend either APC or PDT for oesophago-gastric cancer palliation at the present.

10.4.7 Endoscopic palliation with a stent may be used for advanced distal cancers.

10.5 Physical, psychological and spiritual care

10.5.1 Physical care. This is primarily the responsibility of clinical staff (doctors and nurses). However, advice should be sought from specialist members of the multidisciplinary team (e.g., pain nurse, dietician etc.) whenever appropriate.

Clear documentation is important for continuation of physical care following discharge and also during movement of patients from one hospital area to another.

10.5.2 Psychological care is everyone’s responsibility during the whole clinical care pathway. However, to ensure this is done adequately the person primarily informing the MDT has to identify the need for formal counselling to the patient and family.

There should be one named member of MDT to arrange formal counselling should it be necessary. All patients and their relatives should good access to the team through the upper GI nurse specialist during diagnosis, treatment, diagnosis of recurrence, terminal care, death and bereavement.

10.5.3 Spiritual care

All patients and their relatives should have access to spiritual support appropriate for their religion and culture.

11. Follow Up

Every patient is followed up in the hospital at present, unless it is against the patient’s explicit wish.

There is no evidence in favour of improved outcome with intensive follow up. However, such practice is important for various reasons detailed in the following sections. Duplication of follow up and investigations should be avoided. An open access to the upper GI nurse has to exist as the majority of patients need this link in between appointments.

11.1.1 Nutritional support. Patients to be assessed at least once for their nutritional need during hospital stay. There has to be one named member of the MDT responsible for ensuring this and coordinating with community nutritional teams.

Patients should have an adequate supply of oral nutritional supplements during staging investigations and afterwards if necessary.

11.1.2 Psychological support (10.5.2)
11.1.3 Team follow up

As appropriate, alternating appointments with surgeon and oncologist should be made for operated patients. alternating appointments with gastroenterologist and oncologist should be made for patients who had endoscopic palliation. Patients who had neither surgery nor endoscopic palliation should be followed up by the oncologist or the palliative care team. It is recognised that cross referral may be necessary during follow up. The clinical nurse specialist will be responsible for ensuring patients have adequate access to the appropriate team member.

11.1.4 Audit is facilitated by follow up.

Follow up appointments should be utilised as an opportunity to prospectively collect outcome audit data. This is the responsibility of the clinician consulting with the patient.

12. Data Collection and Audit

12.1.1 National oesophago-gastric cancer audit

The centres will participate with the national audit

12.1.2 Responsibility

The responsibility for the veracity of data will rest with the clinician in overall charge of the main episode of care. The responsibility for data entry and completeness will rest with the data manager. The London Cancer Pathway Board is committed to providing data for comparative audit to AUGIS of all new patients diagnosed with oesophago-gastric cancer.

12.1.3 Data manager

There will be a data manager for the input of data and its collection, cleaning, and return to AUGIS.
13. Appendices

13.1 Referral Guidelines – for Primary Care

Patients should be referred for urgent assessment for the primary diagnosis of oesophago-gastric cancer. The national guidelines for upper gastrointestinal cancer are as follows.

<table>
<thead>
<tr>
<th>Dysphagia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspepsia with Alarm Symptoms</td>
</tr>
<tr>
<td>- Weight loss</td>
</tr>
<tr>
<td>- Anorexia</td>
</tr>
<tr>
<td>- Anaemia</td>
</tr>
</tbody>
</table>

Dyspepsia with Age over 55 and High Risk features:
- Onset of dyspepsia less than 1 year ago
- Continuous symptoms since onset

Dyspepsia with known Risk Factors
- Family history of upper gastrointestinal cancer in more than 1 first-degree relative
- Barrett’s oesophagus
- Pernicious anaemia
- Peptic ulcer surgery over 20 years ago
- Known dysplasia
- Atrophic gastritis
- Intestinal metaplasia

Jaundice

Upper abdominal mass

The definition of the ambiguous term “dyspepsia” is the main weakness in these guidelines. For our purposes dyspepsia is “Discomfort associated with eating”

13.2 Referral Guidelines for Specialist Centre

13.2.1 Multidisciplinary Team (MDT) discussion

All patients with a diagnosis of oesophago-gastric cancer are to be discussed at the Local Centre Multidisciplinary meeting. Only those in whom treatment will require laser, chemotherapy, radiotherapy or surgery should be referred to Specialist Centre

13.2.2 Referral to the specialist centre should be with initial staging

Patients referred to Specialist Centre will have the following investigations completed:
- Upper GI staging endoscopy
- Prone CT scan of thorax, abdomen and pelvis for oesophageal cancer
- Supine (water inflated stomach) CT scan of thorax, abdomen and pelvis for gastric cancer

13.2.3 Targets for treatment access should be expeditious

There is no evidence that any target time improves outcome.

The date of diagnosis should be that of the first endoscopy in which subsequent histology confirms the diagnosis.

Treatment should begin within 2 weeks of diagnosis.
13.3 Classification of oesophago-gastric tumours

13.3.1 Site of oesophago-gastric junctional tumours (Siewert Classification)

<table>
<thead>
<tr>
<th>Type</th>
<th>Tumour Type</th>
<th>Location Relative to Anatomical Cardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>Lower oesophagus adenocarcinoma</td>
<td>Centre lies 1-5 cm above anatomical cardia</td>
</tr>
<tr>
<td>Type II</td>
<td>True cardia adenocarcinoma</td>
<td>Centre lies between 1 cm above and 2 cm below anatomical cardia</td>
</tr>
<tr>
<td>Type III</td>
<td>Gastric adenocarcinoma</td>
<td>Centre between 2 and 5 cm below anatomical cardia</td>
</tr>
</tbody>
</table>

The cardia is the transitional point between the columnar lined stomach and the squamous lined oesophagus. In pathological specimens, the peritoneal reflection reflects this point where the junction has been obliterated by tumour. During surgery, on-table endoscopy can help define the true level of the tumour in relation to this landmark.

The measurements are those in situ.
13.3.2 Japanese Cancer Society lymph node stations

<table>
<thead>
<tr>
<th>Number</th>
<th>Station Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Right cardial lymph nodes</td>
</tr>
<tr>
<td>2.</td>
<td>Left cardial lymph nodes</td>
</tr>
<tr>
<td>3.</td>
<td>Lymph nodes along the lesser curvature</td>
</tr>
<tr>
<td>4.</td>
<td>Lymph nodes along the greater curvature</td>
</tr>
<tr>
<td>5.</td>
<td>Suprapyloric lymph nodes</td>
</tr>
<tr>
<td>6.</td>
<td>Infrapyloric lymph nodes</td>
</tr>
<tr>
<td>7.</td>
<td>Lymph nodes along the left gastric artery</td>
</tr>
<tr>
<td>8a</td>
<td>Lymph nodes in the anterosuperior group along the common hepatic artery</td>
</tr>
<tr>
<td>9.</td>
<td>Lymph nodes around the celiac artery</td>
</tr>
<tr>
<td>10.</td>
<td>Lymph nodes at the splenic hilum</td>
</tr>
<tr>
<td>11.</td>
<td>Lymph nodes along the splenic artery</td>
</tr>
<tr>
<td>8b</td>
<td>Lymph nodes in the posterior group along the common hepatic artery</td>
</tr>
<tr>
<td>12.</td>
<td>Lymph nodes in the hepatoduodenal ligament</td>
</tr>
<tr>
<td>13.</td>
<td>Lymph nodes on the posterior surface of the pancreatic head</td>
</tr>
<tr>
<td>14V</td>
<td>Lymph nodes along the superior mesenteric vein</td>
</tr>
<tr>
<td>14A</td>
<td>Lymph nodes along the superior mesenteric artery</td>
</tr>
<tr>
<td>15.</td>
<td>Lymph nodes along the middle colic vessels</td>
</tr>
<tr>
<td>16a</td>
<td>Lymph nodes around the abdominal aorta</td>
</tr>
<tr>
<td>16a</td>
<td>Lymph nodes around the abdominal aorta</td>
</tr>
<tr>
<td>16b</td>
<td>Lymph nodes around the anterior surface of the pancreatic head</td>
</tr>
<tr>
<td>17.</td>
<td>Lymph nodes along the inferior margin of the pancreas</td>
</tr>
<tr>
<td>18.</td>
<td>Lymph nodes along the inferior margin of the pancreas</td>
</tr>
<tr>
<td>19.</td>
<td>Infradiaphragmatic lymph nodes</td>
</tr>
<tr>
<td>20.</td>
<td>Lymph nodes in the oesophageal hiatus of the diaphragm</td>
</tr>
<tr>
<td>110</td>
<td>Paraesophageal lymph nodes in the lower thorax</td>
</tr>
<tr>
<td>111</td>
<td>Supradiaphragmatic lymph nodes</td>
</tr>
</tbody>
</table>
13.4 TNM Staging and stage grouping (6th Edition)

13.4.1 Oesophagus TNM 6 Definitions

TX Primary tumour cannot be assessed
T0 No evidence of primary tumour
Tis Carcinoma in situ
T1 Tumour invades lamina propria or submucosa
T2 Tumour invades muscularis propria
T3 Tumour invades adventitia
T4 Tumour invades adjacent structures
NX Regional lymph nodes cannot be assessed
N0 No regional lymph node metastasis
N1 Regional lymph node metastasis
MX Distant metastasis cannot be assessed
M0 No distant metastasis
M1 Distant metastasis

Tumours of the lower thoracic oesophagus:
M1a Metastasis in celiac lymph nodes
M1b Other distant metastasis

Tumours of the mid thoracic oesophagus:
M1aa Not applicable
M1b Nonregional lymph nodes and/or other distant metastasis

Tumours of the upper thoracic oesophagus:
M1a Metastasis in cervical nodes
M1b Other distant metastasis

Biopsy of metastatic site performed Yes/No
Source of pathologic metastatic specimen

Stage Grouping

<table>
<thead>
<tr>
<th>Stage</th>
<th>Tis</th>
<th>N0</th>
<th>M0</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>T1</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>IIA</td>
<td>T2</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>IIB</td>
<td>T1</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T2</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td>III</td>
<td>T3</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T4</td>
<td>Any N</td>
<td>M0</td>
</tr>
<tr>
<td>IV</td>
<td>Any T</td>
<td>Any N</td>
<td>M1</td>
</tr>
<tr>
<td>IVA</td>
<td>Any T</td>
<td>Any N</td>
<td>M1a</td>
</tr>
<tr>
<td>IVB</td>
<td>Any T</td>
<td>Any N</td>
<td>M1b</td>
</tr>
</tbody>
</table>

Histological grade
Gx Grade cannot be assessed
G1 Well differentiated
G2 Moderately differentiated
G3 Poorly differentiated
G4 Undifferentiated

Residual Tumour (R)
Rx Presence of residual tumour cannot be assessed
R0 No residual tumour
R1 Microscopic residual tumour
R2 Macroscopic residual tumour

Additional Descriptors
For identification of special cases of TNM or pTNM classifications, the m suffix and "y" "r", and "a" prefixes are used. Although they do not affect the stage grouping, they indicate cases needing separate analysis.

"m" suffix indicates the presence of multiple primary tumours in a single site and is recorded in parentheses: pT(m)NM.

"y" prefix indicates those cases in which classification is performed during or following initial multimodality therapy. The cTNM or pTNM category is identified by a "y" prefix. The ycTNM or ypTNM categorizes the extent of tumour actually present at the time of that examination. The "y" categorisation is not an estimate of tumour prior to multimodality therapy.

"r" prefix indicates a recurrent tumour when staged after a disease-free interval, and is identified by the ‘r’ prefix: rTNM.

"a" prefix designates the stage determined at autopsy. aTNM.

Notes

Lymphatic vessel Invasion (L)
Lx Lymphatic vessel invasion cannot be assessed
L0 No lymphatic vessel invasion
L1 Lymphatic vessel invasion

Venous Invasion (V)
VX Venous invasion cannot be assessed
V0 No venous invasion
V1 Microscopic venous invasion
V2 Macroscopic venous invasion
13.4.2 Stomach TNM 6 Definitions

<table>
<thead>
<tr>
<th>Code</th>
<th>Definition</th>
<th>Stage Grouping</th>
</tr>
</thead>
<tbody>
<tr>
<td>TX</td>
<td>Primary tumour cannot be assessed</td>
<td>Tis N0 M0</td>
</tr>
<tr>
<td>T0</td>
<td>No evidence of primary tumour</td>
<td>T1 N0 M0</td>
</tr>
<tr>
<td>Tis</td>
<td>Carcinoma in situ: Intraepithelial tumour without invasion of the lamina propria</td>
<td>T2a N0 M0</td>
</tr>
<tr>
<td>T1</td>
<td>Tumour invades lamina propria or submucosa</td>
<td>T2a/b N0 M0</td>
</tr>
<tr>
<td>T2a</td>
<td>Tumour invades muscularis propria</td>
<td>T2a/b N1 M0</td>
</tr>
<tr>
<td>T2b</td>
<td>Tumour invades subserosa</td>
<td>T2a/b N2 M0</td>
</tr>
<tr>
<td>T3</td>
<td>Tumour invades serosa (visceral peritoneum) without invasion of adjacent structures</td>
<td>T2a/b N1 M0</td>
</tr>
<tr>
<td>T4</td>
<td>Tumour invades adjacent structures</td>
<td>T3 N0 M0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T4 N0 M0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T4 N1-3 N M0</td>
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<td></td>
<td></td>
<td>T4 N3 N M0</td>
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<tr>
<td></td>
<td></td>
<td>Any T Any N M1</td>
</tr>
</tbody>
</table>

Histological grade

<table>
<thead>
<tr>
<th>Code</th>
<th>Grade</th>
<th>Residual Tumour (R)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GX</td>
<td>Grade cannot be assessed</td>
<td>Presence of residual tumour cannot be assessed</td>
</tr>
<tr>
<td>G1</td>
<td>Well differentiated</td>
<td>No residual tumour</td>
</tr>
<tr>
<td>G2</td>
<td>Moderately differentiated</td>
<td>Microscopic residual tumour</td>
</tr>
<tr>
<td>G3</td>
<td>Poorly differentiated</td>
<td>Macroscopic residual tumour</td>
</tr>
<tr>
<td>G4</td>
<td>Undifferentiated</td>
<td></td>
</tr>
</tbody>
</table>

Additional Descriptors

- **“m” suffix** indicates the presence of multiple primary tumours in a single site and is recorded in parentheses: PT(m)NM.
- **“y” prefix** indicates those cases in which classification is performed during or following initial multimodality therapy. The cTNM or pTNM category is identified by a “y” prefix. The ycTNM or ypTNM categorizes the extent of tumour actually present at the time of that examination. The “y” categorization is not an estimate of tumour prior to multimodality therapy.
- **“r” prefix** indicates a recurrent tumour when staged after a disease-free interval, and is identified by the ‘r’ prefix: rTNM.
- **“a” prefix** designates the stage determined at autopsy. aTNM.

Notes

<table>
<thead>
<tr>
<th>Lymphatic vessel Invasion (L)</th>
<th>Venous Invasion (V)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lx</td>
<td>VX</td>
</tr>
<tr>
<td>L0</td>
<td>VO</td>
</tr>
<tr>
<td>L1</td>
<td>V1</td>
</tr>
</tbody>
</table>

Approved 27/11/2013

London Cancer Upper GI (OG) Pathway Board
Guidelines for the management of oesophago-gastric cancer

London Cancer

13.5 TNM-7 (December 2009)

TNM-7 Oesophago-gastric junction tumours

A tumour the epicentre of which is within 5 cm of the oesophago-gastric junction and also extends into the oesophagus is classified and staged according to the oesophageal scheme.

All other tumours with an epicentre in the stomach greater than 5 cm from the oesophago-gastric junction or those within 5 cm of the EGJ without extension into the oesophagus are staged using the gastric carcinoma scheme.

Oesophagus 7th edition TNM definitions: AJCC = UICC

<table>
<thead>
<tr>
<th>Tis</th>
<th>Carcinoma in situ/High-grade dysplasia</th>
<th>N0</th>
<th>No regional lymph node metastases</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>Lamina Propria or submucosa</td>
<td>N1</td>
<td>1-2 regional lymph node metastases</td>
</tr>
<tr>
<td></td>
<td>T1a Lamina propria or muscularis mucosae</td>
<td>N2</td>
<td>3-6</td>
</tr>
<tr>
<td></td>
<td>T1b submucosa</td>
<td>N3</td>
<td>&gt;6</td>
</tr>
<tr>
<td>T2</td>
<td>Muscularis propria</td>
<td>N1</td>
<td>(N1 was site dependent)</td>
</tr>
<tr>
<td>T3</td>
<td>Adventitia</td>
<td>M1</td>
<td>Distant metastases</td>
</tr>
<tr>
<td>T4</td>
<td>Adjacent structures</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>T4a pleura, pericardium, diaphragm, or adjacent</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>peritoneum.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>T4b other adjacent structures, e.g. aorta, vertebral body, peritoneum.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Stage Grouping (anatomical – adeno and squamous) UICC

<table>
<thead>
<tr>
<th>IA</th>
<th>T1</th>
<th>N0</th>
<th>M0</th>
</tr>
</thead>
<tbody>
<tr>
<td>IB</td>
<td>T2</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>IIA</td>
<td>T3</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>IIIA</td>
<td>T1, T2</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td>IIIIB</td>
<td>T4a</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T3</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T1, T2</td>
<td>N2</td>
<td>M0</td>
</tr>
<tr>
<td>IIIIB</td>
<td>T3</td>
<td>N2</td>
<td>M0</td>
</tr>
<tr>
<td>IIIIC</td>
<td>T4a</td>
<td>N1, N2</td>
<td>M0</td>
</tr>
<tr>
<td>T4b</td>
<td>Any T</td>
<td>N3</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>Any T</td>
<td>Any N</td>
<td>M1</td>
</tr>
</tbody>
</table>

Stomach 7th edition TNM definitions: AJCC = UICC

<table>
<thead>
<tr>
<th>T1</th>
<th>Lamina Propria or submucosa</th>
<th>N0</th>
<th>No regional lymph node metastases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T1a Lamina propria</td>
<td>N1</td>
<td>1-2 regional lymph node metastases</td>
</tr>
<tr>
<td></td>
<td>T1b submucosa</td>
<td>N2</td>
<td>3-6 (was N1)</td>
</tr>
<tr>
<td>T2</td>
<td>Muscularis propria</td>
<td>N3a</td>
<td>7-15 nodes (was N2)</td>
</tr>
<tr>
<td>T3</td>
<td>Subserosa ( was T2b)</td>
<td>N3b</td>
<td>16 or more (was N3)</td>
</tr>
<tr>
<td>T4a</td>
<td>Perforates serosa (was T3)</td>
<td>M1</td>
<td>Distant metastases</td>
</tr>
<tr>
<td>T4b</td>
<td>Adjacent structures</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>IA</th>
<th>T1</th>
<th>N0</th>
<th>M0</th>
</tr>
</thead>
<tbody>
<tr>
<td>IB</td>
<td>T2</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>IIA</td>
<td>T3</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>IIB</td>
<td>T1</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T4a</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T3</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T2</td>
<td>N2</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T1</td>
<td>N3</td>
<td>M0</td>
</tr>
<tr>
<td>IIIA</td>
<td>T4a</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T3</td>
<td>N2</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T2</td>
<td>N3</td>
<td>M0</td>
</tr>
<tr>
<td>IIIIB, IIIIC, IV</td>
<td>Unchanged</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>T1-3</td>
<td>N3</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>Any T</td>
<td>Any N</td>
<td>M1</td>
</tr>
</tbody>
</table>
Datasets

13.5.1 Network dataset (Now reduced to new National Oesophago-gastric Cancer Audit dataset)

13.5.2 National Oesophago-gastric Cancer Audit dataset data forms (AUGIS, BSG, RCSE, NHS information)

These may be found at the following URL:
www.ic.nhs.uk/services/national-clinical-audit-support-programme-ncasp/cancer/oesophago-gastric/user-information/useful-documents

13.6 Clinical Information Required on Specimen Request Form

In the UK, most gastric resections for carcinoma contain a palpable tumour, which is readily identifiable on visual inspection of the mucosal aspect of the specimen. However, in some specimens tumour may not be macroscopically obvious. This is becoming increasingly the case with the widespread use of neoadjuvant chemotherapy. In all cases, and especially those without obvious macroscopic tumour, clinical information may be useful in optimising specimen sampling. Clinical information that may be helpful includes:

- site of tumour
- type of tumour (if known)
- previous histology (where performed and case number if available)
- any history of neoadjuvant chemoradiotherapy
NATIONAL DATASET FOR GASTRIC CARCINOMA HISTOPATHOLOGY REPORTS

<table>
<thead>
<tr>
<th>Surname ...............................................</th>
<th>Forenames ...............................................</th>
<th>Date of birth .......................</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital ............................................</td>
<td>Hospital no ..........................................</td>
<td>NHS no ................................</td>
</tr>
<tr>
<td>Date of receipt .....................................</td>
<td>Date of reporting ....................................</td>
<td>Report no ................................</td>
</tr>
<tr>
<td>Pathologist ..........................................</td>
<td>Surgeon ...............................................</td>
<td>Sex .........................................</td>
</tr>
</tbody>
</table>

**GROSS DESCRIPTION**

<table>
<thead>
<tr>
<th>Type of specimen</th>
<th></th>
<th>Specimen dimensions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oesophago-gastrectomy</td>
<td></td>
<td>Length of stomach - greater curve ............ mm</td>
</tr>
<tr>
<td>Total gastrectomy</td>
<td></td>
<td>Length of stomach - lesser curve ........ mm</td>
</tr>
<tr>
<td>Local resection</td>
<td></td>
<td>Length of oesophagus ................................ mm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Length of duodenum ................................ mm</td>
</tr>
<tr>
<td>Type of tumour</td>
<td></td>
<td>Site of tumour .......................................</td>
</tr>
<tr>
<td>Polyoid, ulcerating or fungating</td>
<td></td>
<td>Maximum tumour diameter ................ mm</td>
</tr>
<tr>
<td>Diffusely infiltrating</td>
<td></td>
<td>Distance of tumour to nearest margin (cut end)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>................................................ mm</td>
</tr>
</tbody>
</table>

**HISTOLOGY**

<table>
<thead>
<tr>
<th>Type of tumour</th>
<th>Proximal margin involved</th>
<th>Distal margin involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenocarcinoma</td>
<td>Yes □ No □</td>
<td>Yes □ No □ □</td>
</tr>
<tr>
<td>Other (specify)</td>
<td>..................................................</td>
<td>□</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lauren classification</th>
<th>Circumferential margin lower oesophagus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intestinal □</td>
<td>Involvement (&lt; 1 mm): Yes □ No □ N/A □</td>
</tr>
<tr>
<td>Diffuse/mixed □</td>
<td>(If no, distance of tumour to nearest</td>
</tr>
<tr>
<td></td>
<td>circumferential margin ................ mm)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Differentiation by worst area</th>
<th>Lymphatic/vascular invasion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Well/moderately □</td>
<td>Yes □ No □</td>
</tr>
<tr>
<td>Poorly □</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Local invasion</th>
<th>Proximal margin involved</th>
<th>Distal margin involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>T0 No tumour identified □</td>
<td>...........................................</td>
<td>□</td>
</tr>
<tr>
<td>Tis Carcinoma in situ □</td>
<td>...........................................</td>
<td>□</td>
</tr>
<tr>
<td>T1 Invasion of lamina propria/submucosa □</td>
<td>...........................................</td>
<td>□</td>
</tr>
<tr>
<td>T2a Invasion of muscularis propria □</td>
<td>...........................................</td>
<td>□</td>
</tr>
<tr>
<td>T2b Invasion into subserosa □</td>
<td>...........................................</td>
<td>□</td>
</tr>
<tr>
<td>T3 Invasion of serosa □</td>
<td>...........................................</td>
<td>□</td>
</tr>
<tr>
<td>T4 Invasion of adjacent structures □</td>
<td>...........................................</td>
<td>□</td>
</tr>
</tbody>
</table>

**PATHOLOGICAL STAGING**

<table>
<thead>
<tr>
<th>Complete resection</th>
<th>TNM</th>
<th>(y)...... pT □ N □ M □</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes (R0) □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No (R1 or R2) □</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>History of neoadjuvant therapy (y)</th>
<th>Signature ...............................................</th>
<th>Date....../....../......</th>
<th>SNOMED codes T............./M..........</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes □</td>
<td>..........................................................</td>
<td>□</td>
<td></td>
</tr>
<tr>
<td>No □</td>
<td>..........................................................</td>
<td>□</td>
<td></td>
</tr>
</tbody>
</table>
### NATIONAL DATASET FOR OESOPHAGEAL CARCINOMA HISTOPATHOLOGY REPORTS

<table>
<thead>
<tr>
<th>Surname</th>
<th>Forenames</th>
<th>Date of birth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital</td>
<td>Hospital no</td>
<td>NHS no</td>
</tr>
<tr>
<td>Date of receipt</td>
<td>Date of reporting</td>
<td>Report no</td>
</tr>
<tr>
<td>Pathologist</td>
<td>Surgeon</td>
<td>Sex</td>
</tr>
</tbody>
</table>

#### GROSS DESCRIPTION
- **Maximum length of specimen:** mm
- **Length of oesophagus:** mm
- **Length of stomach:** mm
- **Type of tumour:**
  - Polypoid
  - Pinned
  - Other
- **Tumour edge to nearest distal margin:** mm
- **Tumour edge to nearest proximal margin:** mm
- **Length of tumour:** mm
- **Siewert tumour type:**
  - Cardiac cancers only
- **Width of tumour:** mm

#### HISTOLOGY
- **Type of tumour**
  - Squamous
  - Adenocarcinoma
  - Other (specify)
- **Differentiation by worst area:**
  - Well
  - Moderately
  - Poorly differentiated
- **Depth of invasion**
  - Tis high-grade dysplasia
  - T1 invasion of lamina propria/submucosa
  - T2 invasion of muscularis propria
  - T3 invasion beyond muscularis propria
  - T4 invasion of adjacent structures
- **Proximal margin**
  - Normal
  - Dysplasia
  - Carcinoma
  - Barrett’s
- **Distal margin**
  - Normal
  - Dysplasia
  - Carcinoma

#### PATHOLOGICAL STAGING
- **Complete resection**
  - Yes (R0)
  - No (R1 or R2)
- **Circumferential margin**
  - Involvement (<1 mm): Yes
  - N/A
- **Other features**
  - Vascular invasion
  - Barrett’s metaplasia
- **Lymph nodes**
  - Number examined
  - Number positive
- **Distant metastases**
  - Coeliac axis node positive
  - Cervical node positive
- **SNOMED codes T**

#### COMMENTS

---

**APPENDIX D**

**Guidelines for the management of oesophago-gastric cancer**

*London Cancer*

*Approved 27/11/2013*

*Upper GI (OG) Pathway Board*