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Welcome Note 2015

Once again welcome to the 2015 19th Annual Controversies and Problems in Surgery Symposium.

I should thank those patrons who participate year in year out for your loyalty, and welcome especially our novices who are attending for the first time.

I need to explain the move away from a Friday to a Sunday. The sole reason is economic. In our endeavour to keep the symposium affordable we had to book a venue within the University of Pretoria precinct, having tried other possible venues including the CSIR which turned out to be much more expensive. However the University venue was not available on the Friday, it being used for lectures. The only possible solution which we adopted was to move the programme to include the Sunday instead of the Friday. This may inconvenience some of you for which we apologize profusely.

The theme of this year is “Complications of common surgical procedures and their management”. We hope this will arm you in your practices to effectively manage such complications. The adage in the medicine that, “if you have not had a complication from whatever procedure, it means you have not done enough”, is true. All of us will experience a complication at some stage in our careers, even from a commonly performed procedure. We should therefore arm ourselves with adequate knowledge of how to approach the management of such complications. It is the object of this year’s symposium to arm you with up to date management approach to possible complications.

I should again thank the presenters for their preparation and timeous submission of their monograms for inclusion in the proceedings. I know this takes time and effort. Please continue your contribution to this important continued professional development exercise.

One needs to once again thank the Trade for their continued support of this aspect of continuing professional development. We specifically welcome and thank new sponsors and implore them to join the coterie of our loyal supporters and sponsors. We recognise that the economy in our country is struggling as a reflection of a worldwide economic downturn. Your support is therefore even more appreciated.

Lastly I should like to thank the members of my staff for their stellar performance as usual preparing for the symposium.

Enjoy.

Prof Taole Mokoena
October 2015
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Trauma remains a leading cause of death among people under the age of 44 (Simmons 2014, Hess 2008, Thorsen 2011). Haemorrhage, especial from pelvic and abdominal trauma is a major contributing factor to trauma related deaths in the first 48hrs of injury. Trauma and major surgery is often complicated by a coagulopathy that makes management of the patient a formidable challenge.

The paper discusses mechanism, identification and suggested management of this acute coagulopathy. It is my aim to provoke a thought about the role of coagulation system in that bleeding patient during abdominoperineal surgery; following a ‘cholecystectomy gone wrong’ or ‘the severely injured, hypotensive patient with an open abdomen, a pH of 7.0, oozing everywhere, whose bleeding will not stop nor blood made to clot’.

**Definition**

Acute coagulopathy complicating trauma or major surgery has been described over a decade ago and is gaining greater recognition as a clinical problem. It has been identified as a unique derangement that is distinct from disseminated coagulopathy (DIC) as commonly seen in sepsis or other conditions (Levi, 2007). Recognizing that applying generic terminology of "DIC" to this phenomenon is unhelpful and potentially counterproductive, various studies have suggested alternative names such as “Trauma-induced Coagulopathy" (TIC) (Spivey, 2005), “Early Coagulopathy of Trauma" (MacLeod, 2003), “Acute Traumatic Coagulopathy" (Brohi, 2003) and “Acute Coagulopathy of Trauma Shock" (ACoTS) (Hess, 2008). These names will be used in this presentation.

**Significance**

Acute Coagulopathy of Trauma Shock complicates management of major trauma or surgery and is usually associated with hypoperfusion; it is present in a third to 25% of trauma patients on admission to the emergency unit and is associated with four fold increase in mortality (Brohi 2003, MacLeod 2003, Maegele 2007, Frith 2012). ACoTS is an independent predictor of massive transfusion, preventable death, and increased hospital length of stay and development of multiple organ dysfunction (Cannon 1918, Maegele 2007, Brohi 2007)

**Why the fuss? This is just DIC**

Some scholars suggest that seeking to distinguish between ACoTS and DIC is a fuss, splitting hairs and is only of academic interest. This is perhaps true, ACoTS is marked by systemic or disseminated coagulopathy and there are similarities with DIC; however, acute coagulopathy of trauma is not entirely an impairment of the coagulation cascade, it is characterized by increased fibrinolysis and systemic anticoagulation; it is also exclusively found in patient with shock. Both fibrinolysis and anticoagulation are exaggerated with worsening base deficit (Hess 2008, Cohen 2013). Understanding of this underlying pathophysiological differences is pivotal to the management this derangement.

**Myth or reality**

Brohi and colleagues (2003) found clinically significant coagulopathy in 25% of the 1088 patients admitted to emergency department; coagulopathy developed long before administration of large volumes of fluids.
The existence of early coagulopathy has been verified in various other studies involving more than 20 thousand patients (Table 1)

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<thead>
<tr>
<th></th>
<th>Number</th>
<th>Percentage</th>
<th>ISS</th>
<th>Mortality No coagulopathy</th>
<th>Mortality Coagulopathy</th>
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<td>1088</td>
<td>24</td>
<td>20</td>
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<td>10790</td>
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<td>10</td>
<td>17</td>
<td>8</td>
<td>62</td>
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Table 1 Summary of studies of acute coagulopathy of trauma. ISS Injury Severity Score

**Pathophysiology of ACoTS**

Classically in DIC, there is impairment of the coagulation cascade; contrary to that simple explanation, the causes of acute coagulopathy of major trauma are multifactorial. Various endogenous, biological processes as well as iatrogenic and environmental factors are important in the initiation and progress of ACoTS.

There is greater appreciation and respect for endothelium as an important driver of the main systems responsible for ACoTS (fibrinolysis and anticoagulation). The endothelium, activated by tissue factor (in the setting of hypoperfusion), produce high levels of thrombomodulin. Thrombomodulin forms a complex with thrombin reducing formation of fibrin. The thrombin-thrombomodulin complex converts protein C into activated protein C (APC), an anticoagulant serine protease enzyme, which may have a central role in the acute coagulopathy of trauma. APC modulates clotting in multiple ways, including increasing levels of plasmin and promoting fibrinolysis (Brohi 2007, Lier 2004).

APC also impairs the clotting cascade by inhibiting activated clotting factors V and VIII. Further, it has anti-inflammatory and antiapoptotic properties that may reduce secondary insults after localized tissue damage (Mosnier 2004). Unfortunately, the anticoagulant aspects of APC outweigh its protective properties after massive tissue damage.

Pathological thrombosis accompanies the coagulopathy of injury when tissue factor, a procoagulant subendothelial protein, is exposed in injured organs, particularly in cases of traumatic brain injury.
Making a diagnosis and monitoring ACoTS

Anticipation, early diagnosis and monitoring of coagulopathy is vital to improved outcome; when clinical signs appear, it is late in the process and exceptional efforts must be taken to regain control. Traditional tests like prothrombin time (PT), partial thromboplastin time (PTT), platelets, and internationalized normalised ratio (INR) have been used in many studies and clinical practice to diagnose, monitor and manage coagulopathy of trauma; the predictive value of these tests are controversial and debatable (Simmons 2014, Schochl 2012).

An ideal test that is rapid, cheap, user friendly, reproducible and reliable does not exist. It is possible that viscoelastic haemostatic tests such as thromboelastography (TEG) and rotational thromboelastometry (ROTEM) come close.

These tests provide information on the time course of clot formation in whole blood and offer advantages over the conventional tests for evaluation of coagulation status in patients with massive bleeding. They offer rapid results, increased sensitivity in detecting abnormalities of haemostatic function, and perhaps the most important advantage is the ability to detect fibrinolysis (Johansson 2009).

Management of coagulopathy of trauma

In the traditional approach to trauma patient resuscitation, the goal was to achieve and maintain a certain arbitrary blood pressure, urine output and to reverse metabolic acidosis. This entailed huge amount of crystalloids, colloids and blind administration of blood and blood products. This approach, sensible and noble as it may be failed to address coagulopathy and the outcome has been poor with early deaths, prolonged intensive care stay, lung injury, ARDS and septic complications.

One major progress in trauma care was the introduction of damage control surgery (DCS), an abbreviated laparotomy whose sole purpose is to control haemorrhage and contamination while
delaying definitive surgery for later when the patient is stable. Damage control resuscitation (DCS) on the other hand, is the analogous term for treating haemorrhagic shock by correcting coagulopathy, acidosis, and hypothermia as efficiently as possible, with concurrent efforts to control the bleeding source and avoid iatrogenic injury (Beekly 2008). The following is a brief discussion of the key steps in the management of ACoTS:

**Permissive hypotension**

Aggressive fluid resuscitation is indicated in injured patients to ensure tissue perfusion (Davis 1996, Moore 2006). It has however been demonstrated in several studies that prehospital administration of less than 1.5 litres is associated with greater chance of survival whereas larger amounts of fluids increases the incidence of ACoTS up to 40% (MacLeod 2003, Sihler 2009, Madigan 2008) and abdominal compartment syndrome (Madigan 2008).

With permissive hypotension a mean blood pressure of 65mmHg or systolic pressure of 90mmHg is targeted until surgical intervention to stop haemorrhage is achieved. It is contraindicated in traumatic brain injury, coronary disease and hypertension (Berry 2012) and should limited to 120 minutes.

**Blood and blood products**

Administration of blood in trauma resuscitation cannot be avoided; and there is overwhelming evidence of harm and mortality with use of blood and its products like fresh frozen plasma (FFP) and platelets, notwithstanding morbidity, increased length of stay, multiorgan failure, infections, transfusion overload, transfusion related lung injury (TRALI) and sepsis (Lelubre 2009).

The focus has shifted to questions such as the right trigger haemoglobin, the ratios of fresh frozen plasma (FFP) to packed red blood cells (PRBC). Literature seems to suggest that higher ratios of FFP to PRBC favour reduced mortality (Mitra 2012, Holcomb 2007, Maegele 2007). There are however no randomised controlled trials to proof this.

More than the debates about the right ratio of plasma to blood, the focus should be on identifying patients who will require massive transfusion. Massive transfusion is actually an important determinant of outcome and implementation of massive transfusion protocols (MTP) has been shown to improve outcome with decrease in early deaths due to bleeding (Dente 2009, Griffie 2010).

Clinical criteria to identify patients who will benefit from massive transfusion protocol lack ability to adequately pick these patients; several scores have been devised with varied predictive value. The Trauma Associated Severe Haemorrhage (TASH) score and Shock Index (SI) seem to offer high predictive value for massive transfusion (Brogman 2011, Mutschler 2013). Finally, although the recommended ratio of plasma, blood and platelets is 1:1:1, massive transfusion protocol gives a logical sequential approach to blood transfusion and blood product replacement.

**Rewarming**

Hypothermia, defined as core body temperature lower than 35 degrees C, has an incidence of 1.6 to 8.2 percent in trauma victims (Holcomb 2007). It is not clear if the low temperatures in patients with poor outcome are a marker of severity of injury and physiologic derangement or cause of mortality; however, patients with core temperatures of less than 32 °C have high mortality (Shafi 2013). Early attempts should be made to limit heat loss; use of warmed fluids and both passive
and active warming should be employed.

**Correction of acidosis**
Serum lactate and base deficits measurements give good idea of the severity of shock and hypoperfusion. In clinical practice, serial values of lactate are useful in predicting survival and have been used to assess response to therapy. Similarly, base deficit can independently predict mortality and is especially useful in inebriated patients likely to have falsely elevated lactate levels (Cherian 2014). Restoration of normal perfusion aimed at correcting base deficit and pH is the mainstay of treating and preventing acidosis. It may be necessary to restore volume with blood and blood products to manage acidosis and avoid giving excessive amounts of fluids crystalloids or colloids).

Pharmacological methods of controlling bleeding

**Antifibrinolytics**
Tranexamic acid (TxA) was studied in a double blinded, randomised, multicentre trial (clinical randomisation of an antifibrinolytic in significant haemorrhage, CRASH 2) involving over ten thousand adult trauma patients, and shown to significantly reduce “all cause” mortality and mortality due to bleeding (Roberts 2011). Greatest benefit is when administered within 3 hours of trauma, in a dose of 1-2g over 10min and repeat 1g over 8 hours (Lier 2011, Spahn 2013).

**Recombinant factor VII**
Very high dose of recombinant factor VIIa (rFVIIa) are required for the formation of tissue factor complex to activate the clotting system. rFVIIa bypasses several steps of coagulation and interacts directly with activated platelets to form thrombin. Early use of rFVIIa was associated with a decreased 24 hour and 30 day mortality in severely injured combat patients (Spinella 2008, Boffard 2005). It has also been shown to decrease transfusion requirements in blunt trauma patients (Duchesne 2008, Berkhof 2009).

Recombinant FVIIa is used as an off label agent in many massive transfusion protocols, and the reported rate of thromboembolism is small.

**Combination of fibrinogen and prothrombin complex concentrates**
Use of prothrombin complex concentrates (PCC) can reduce the risk of transfusion associated acute lung injury and other viral infections. In a study on combat related trauma requiring massive transfusion, high fibrinogen to RBC ratio (> 1 g/L to 5 units packed RBCs) was found to decrease death from haemorrhage. A fibrinogen level of > 1.5 g/L should be maintained following trauma (Lier 2008) and transfusion of fibrinogen or cryoprecipitate may be considered if it is below this level. PCC or a complex of factors II, VII, IX, X is found to shorten the time to coagulation and reduce blood loss following trauma (Lier 2011).

Rapid control of bleeding
Haemostatic resuscitation and damage control surgery are vital components in the management and prevention of ACoTS and these strategies are best employed early before patients are at the end of their physiological reserves.
CONCLUSION

The last five years have seen tremendous advances in our understanding of acute coagulopathy of trauma. Although studies determining the influences of single preconditions to traumatic coagulopathy are interesting for scientific purposes, in the clinical setting, ACoTS and the deterioration of haemostasis in trauma is always caused by multiple and concurring ones.

We maybe far from having all the answers, but we understand that coagulopathy sets in much earlier than traditionally believed. Trauma shock and tissue hypoperfusion are central to the causati on of the early ACoTS. Routine tests of coagulation are inadequate in diagnosing or monitoring coagulopathy and should be replaced by better tests. Presently viscoelastic hemostatic assays are the most reliable among existing tests in monitoring coagulopathy. Damage control resuscitation is the umbrella term to several measures, including but not limited to permissive hypotension, blood, platelets and plasma transfusions, recombinant factors, that can counter worsening of coagulopathy and result in a reduction of morbidity and mortality.

References and suggested reading

28. Rizoli SB, MD, PhD, FRCSC, Tetsuo Yukioka, MD, David B. Hoyt, MD, FACS, and Bertil Bouillon, MD


**Invited Commentary: Dr S Motilal, Head of Trauma, UP**

**Protocol for Management of Massive Transfusion**

**Sequence of Components**

Profound hypotension should be treated speedily. Administer crystalloid or colloid infusions rather than delay fluid administration. Initial red cell replacement is in the form of packed red cells.

**Laboratory Samples**

At the start of resuscitation, blood should be taken for group and crossmatch, coagulation tests, full blood count and biochemistry. These must be properly labelled and identified in all situations.

**Blood Bank Arrangements**

Routine procedures should be followed until it becomes obvious that massive transfusion is likely. The blood bank should be informed as soon as possible that a major trauma is arriving or in the building.

For extreme emergencies group O blood should be supplied first. Rhesus D negative blood should be supplied to all women of childbearing age. Type specific (ABO Rh D matched) blood should be available in 5 minutes and the switch should be made promptly so as not to deplete stores of group O blood. Continue transfusing blood on this basis until time is available to crossmatch on the original serum sample. If an antibody screen is negative and more than one blood volume has been administered there is no point attempting compatibility tests except to exclude ABO mismatches.

**Monitoring**

During massive transfusion, regular monitoring of haemoglobin, platelet count, prothrombin time (PT), partial thromboplastin time (PTT) and fibrinogen levels should take place and be used to guide component replacement.

**Components**

Component replacement should occur only in the presence of active bleeding or if interventional procedures are to be undertaken.
Platelet concentrates (1 pack/10kg) are given if platelet count falls below 50. Each platelet concentrate also provides around 50ml of fresh plasma.
Fresh frozen plasma (12ml/kg) is administered if PT or PTT are running higher than 1.5 times control levels.
Cryoprecipitate (1-1.5 packs/10kg) is given for Fibrinogen levels < 0.8g/l.

For massive uncontrolled traumatic haemorrhage, maintenance of full hemostatic ability is usually unrealistic. **The priority is for definitive surgical arrest of haemorrhage from major vessels.**
Combinations of stored whole blood, packed cells, colloids & crystalloids are given to maintain blood volume or pressure at adequate levels and haemoglobin at around 7g/dl or haematocrit at 0.25. Conserve limited supplies of fresh blood, plasma or platelets until the bleeding is controlled. When blood loss has lessened (0.5l/hour) and major vessels have been controlled, it becomes worthwhile correcting haemostasis.

**Further Reading**

CHALLENGES IN MANAGEMENT OF INHALATION INJURY
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Background
Many victims of fire accident have both inhalation and thermal injury. The combination of bronchopulmonary injury with cutaneous burns that exceed 40 % of the total body surface area (TBSA) causes mortality to increase more than 70 % in South Africa. Smoke inhalation may produce injury through different mechanisms. Heated air may induce significant thermal injury to upper airway. Particulate matters produced during combustion can obstruct and irritate the airways, causing bronchoconstriction. Numerous gases released from burning materials like carbon monoxide (CO), Hydrogen cyanide have toxic effect to many cells. More other lethal gases are used in military warfare with significant mortality related.

Three primary mechanisms that lead to injury in smoke inhalation:

- **Thermal damage**
  It is usually limited to oropharyngeal area. The laryngeal reflexes help to protect the lower airway from heat. Steam and aspiration of hot liquids provide some exception can affect the lower airway as moist air has much greater heat-carrying capacity than dry air.

- **Pulmonary injury**
  Inhalation of toxic products triggers a cascade of effects in the lower airway by activation of body’ inflammatory response, direct tissue injury, mucus oversecretion, acute bronchospasm and obstruction of small airways. Damage to the alveolar capillary membrane increase permeability and intravascular leakage into the pulmonary interstitium. Loss of compliance, further atelectasis, increase edema can result in severe ventilation-perfusion mismatch and hypoxia.

- **Asphyxiation**
  Tissue hypoxia can occur via several mechanisms. Combustion in a closed space can consume significant amounts of oxygen, decreasing the ambient concentration of oxygen to as low as 10-13%. For victims in that setting, the decrease in fraction of inspired oxygen (\(\text{FIO}_2\)) leads to hypoxia, even if they have adequate circulation and oxygen-carrying capacity. If sufficiently severe, hypoxia can lead to multiorgan dysfunction, which substantially raises morbidity and mortality.

*Carbon monoxide* 

CO causes tissue hypoxia by decreasing the oxygen-carrying capacity of the blood. Hemoglobin binds CO with an affinity more than 200 times greater than the affinity for oxygen. Other mechanisms contribute, as well. CO causes a left shift in the oxyhemoglobin saturation dissociation curve, which reduces the ability of hemoglobin to unload oxygen.
The heart is particularly affected because CO binds with the heme molecules in myoglobin, decreasing facilitated diffusion of oxygen into muscle. Interaction of CO with myocardial myoglobin results in decreased myocardial contractility.

The literature suggests that hypoxic encephalopathy secondary to CO poisoning results from a reperfusion injury in which the products of lipid peroxidation and free radical formation contribute to morbidity and mortality.

Cyanide

Cyanide (CN) gas can be produced by combustion of the following: plastics, paper products, rubber, wood and others

CN directly stimulates chemoreceptors of carotid and aortic bodies, leading to a brief period of hyperpnea. CN is a small lipophilic molecule and a chemical asphyxiants that interferes with cellular metabolism by binding to the ferric ion on cytochrome a3, subsequently halting cellular respiration. Affected cells convert to anaerobic metabolism, and lactic acidosis ensues.

The organs most sensitive to cellular hypoxia are the central nervous system (CNS) and the heart. The CNS reacts to low concentrations of cyanide by promoting hyperventilation, thereby increasing exposure.

Consider CN toxicity in all patients with smoke inhalation who have CNS or cardiovascular findings. Cyanide toxicity is difficult to confirm but is frequently concomitant with CO toxicity. Its presence can be inferred by the presence of lactic acidosis in the right clinical setting. Even mild degrees of CN poisoning can cause delayed neurological sequelae in survivors and permanent disability including the following:

- Seizures
- Various extrapyramidal syndromes
- Dystonia
- Postanoxic coma

Methemoglobinemia

Methemoglobinemia occurs in fire due to heat denaturation of hemoglobin, oxides produced in fire, and methemoglobin-forming materials such as nitrites. Methemoglobinemia is less common in smoke inhalation injury than CN and CO toxicity. The pathophysiologic consequences of methemoglobin formation are a decrease in the oxygen-carrying capacity of the blood and a shift of the oxyhemoglobin dissociation curve to the left, similar to carboxyhemoglobin

Workup

In the workup of inhalation injuries caused by toxic smoke, the primary investigation focuses on the pulmonary system. Other tests may be clinically indicated based on history, physical examination, and underlying health problems. Carbon dioxide levels also may be monitored, since patients with prior lung disease such as asthma and chronic obstructive pulmonary disease may be affected more severely and are at greater risk to retain carbon dioxide.

Studies may include the following:

- Pulse oximetry and CO-oximetry
- Arterial blood gases (ABGs)
- Carboxyhemoglobin level
• Lactate
• Complete blood cell count (CBC)
• Chest radiography
• Electrocardiogram
• Serial cardiac enzymes (in patients with chest pain)
• Pulmonary function testing
• Direct Laryngoscopy and fiberoptic bronchoscopy

Pulse Oximetry and CO-oximetry
Pulse oximetry readings can be misleading in the setting of carbon monoxide (CO) exposure or methemoglobinemia because these devices use only 2 wavelengths of light (the red and the infrared spectrum), which detect oxygenated and deoxygenated hemoglobin only and not any other form of hemoglobin. Readings are falsely elevated by CO-bound hemoglobin (carboxyhemoglobin).

Arterial Blood Gases
Arterial oxygen tension (partial pressure of arterial oxygen [PaO₂]) does not accurately reflect the degree of CO poisoning or cellular hypoxia. The PaO₂ level reflects the oxygen dissolved in blood that is not altered by the hemoglobin-bound CO. Because dissolved oxygen makes up only a small fraction of arterial oxygen content, a PaO₂ level within the reference range may lead to serious underestimation of the decrement in tissue oxygen delivery and the degree of hypoxia at the cellular level that occurs when CO blocks the delivery of oxygen to the tissues.

ABG measurements are nonetheless useful to assess the adequacy of pulmonary gas exchange. Although the presence of a PaO₂ level that is within the reference range may not exclude significant tissue hypoxia due to the effects of CO, the presence of a low PaO₂ (< 60 mm Hg in room air) or hypercarbia (alveolar [arterial] carbon dioxide pressure [PaCO₂] level of 55 mm Hg) indicate significant respiratory insufficiency. Metabolic acidosis suggests inadequate oxygen delivery to the tissues. Lactate levels associated with CN poisoning have been reported as being above 8 mmol/L. The concentration of lactate increases proportionally with the amount of CN poisoning, and lactate levels higher than 10 mmol/L are a sensitive indicator of CN levels higher than 1 mg/mg. Note that in most institutions, CN levels can take hours to days for results; therefore, one must rely on clinical and indirect laboratory data. Other Blood Studies
Electrolyte testing can identify an anion gap acidosis. In patients who require large-volume fluid resuscitation, measure electrolytes at regular and frequent intervals to monitor for the electrolyte abnormalities that may occur in these patients. Use results to adjust both fluid and electrolyte replacement.

A baseline CBC is warranted, as certain types of smoke are associated with a significant drop in hemoglobin and hematocrit beginning at 1 week postexposure. A baseline white blood cell count can also be used for comparison when concerns arise about infection.

Cyanide levels
Cyanide levels correlate closely with the level of exposure and toxicity, but these values may not be readily available. Many hospitals send out tests for cyanide levels, and results may not return for several days to a week. In a setting consistent with potential cyanide exposure, institute specific empiric therapy while waiting for laboratory confirmation of the diagnosis.

Findings indicative of cyanide intoxication include the following:

• Persistent neurologic dysfunction unresponsive to use of supplemental oxygen
• Cardiac dysfunction
- Severe lactic acidosis, particularly in the presence of high mixed venous oxygen saturation
- "Arterialization" of the venous blood gas, with PO$_2$ values similar to arterial levels due to lack of oxygen utilization by tissues

**Radiography**
Obtain chest x-ray films in patients with a history of significant exposure or pulmonary symptoms. The chest film is likely to be normal—initial studies have only 8% sensitivity for smoke inhalation—but it provides a baseline for subsequent comparison in cases of significant injury. Radiographic evidence of pulmonary injury typically does not appear until 24-36 hours after the inhalation.

When present, abnormal findings may include atelectasis, pulmonary edema, and acute respiratory distress syndrome (ARDS). Hyperinflation may suggest injury of the smaller airways and air trapping.

In phase III of oxides of nitrogen exposure, a noncardiogenic pulmonary edema pattern may be seen on the chest radiograph. The chest radiograph may also show a pattern similar to military tuberculosis, which corresponds to a pathologic finding of classic bronchiolitis fibrosa obliterans. Fibrotic changes either may clear spontaneously or proceed to severe respiratory failure.

**Computed Tomography**
Chest computed tomography (CT) scans may show ground-glass opacities in a peribronchial distribution and/or patchy peribronchial consolidations. These findings may be present on CT scan as early as a few hours after inhalation injury.

A CT scan of the brain may show signs of cerebral infarction due to hypoxia, ischemia, and hypotension. An interesting and well-reported finding for severe CO toxicity is bilateral globus pallidus low-density lesions. These lesions may not appear until several days after the exposure days. This finding is highly specific for CO insult—unlike focal cortical hypoperfusion, which is nonspecific.

**Radionuclide Scintigraphy**
Delayed or inhomogeneous clearance of 133Xenon can be used to detect small-airway parenchymal injury. However, this study adds little to the clinical management and is not known to offer any particular therapeutic advantage.

**Direct Laryngoscopy and Fiberoptic Bronchoscopy**
A significant number of patients may present with a paucity of upper airway signs or symptoms but may still have serious subglottic injury. The threshold for performing diagnostic bronchoscopy should be low. Bronchoscopy can be diagnostic as well as therapeutic, particularly when lobar atelectasis is present.

Bronchoscopy is the criterion standard for diagnosis of smoke inhalation injury. This procedure examines the airways from the oropharynx to the lobar bronchi. Although it may be performed in the ED, the intensive care unit or burn unit may be a more appropriate setting, especially in patients who are intubated.

Erythema, charring, deposition of soot, edema, and/or mucosal ulceration may be present, although severe vasoconstriction from hypovolemia may mask significant injury. Impending airway obstruction may be inferred. Diagnostic accuracy is reported to be 86%. Fiberoptic bronchoscopy can also be used to facilitate endotracheal tube placement, even in the technically difficult airway.
Studies have shown up to a 96% correlation between bronchoscopic findings and the triad of closed-space smoke exposure, carboxyhemoglobin levels of 10% or greater, and carbonaceous sputum. In another study, serial bronchoscopy was twice as sensitive for diagnosing inhalation injury as clinical findings alone. Bronchoscopy is more sensitive and accurate than clinical examination alone in diagnosing inhalation injury and is, therefore, particularly useful in cases in which the decision to perform endotracheal intubation is unclear.

The use of bronchoscopy in patients with inhalation injury complicated by pneumonia is associated with a decreases in the duration of mechanical ventilation, length of intensive care unit stay, and overall hospital cost. Serial bronchoscopy can help remove debris and necrotic cells in cases with aggressive pulmonary toilet or when suctioning and positive pressure ventilation are insufficient.

Bronchoscopy in children requires the use of a bronchoscope with a relatively small diameter, in order to accommodate the narrow pediatric airway. Extremely small diameter fiberoptic bronchoscopes with a suction port (capable of entering an endotracheal tube sized for a small toddler or infant) have only recently become available, and whether these limit the ability to remove heavy particulate matter is unclear.

Emergency Department Care

Presently, no specific treatment exists to ameliorate the tissue damage and reduce the vulnerability to infection induced by smoke inhalation. Administer 100% oxygen because of the likelihood of CO inhalation in fires. Once CO toxicity, cyanide (CN) toxicity, and methemoglobinemia have been addressed, subsequent treatment is predominantly supportive.

The most urgent concern in patients is the patency of the upper airway and adequacy of ventilation. Check for exposure to heat and thermal injury to the nose, mouth, face, and singed hair. Consider smoke involvement if soot is on the face and in sputum, although smoke inhalation is possible without evidence of soot. The proportion of patients with an inhalation injury who require endotracheal intubation is higher for those who also have a burn injury: 62% with a burn versus 12% without a thermal injury and the incidence of inhalation injury increases with the size of the burn.

Oxidant injury eventually leads to cast formation of cellular debris in the airways, thus contributing to pulmonary failure.

Mechanical Ventilation

Mechanical ventilation may be necessary in patients with inhalation injury. Use of positive pressure ventilation with low tidal volumes (3-5 mL/kg) and positive end-expiratory pressure (PEEP) and maintenance of plateau pressures below 30 cm water significantly increases short-term survival and is associated with decreased tracheobronchial cast formation. In fact, this treatment has been shown to increase the intensive care unit (ICU) survival rate.

PEEP may assist in opening obstructed closed alveoli and help ventilation in those patients with poor compliance by increasing functional residual capacity. Ideally, PEEP stents alveoli open, preventing the atelectasis and alveolar flooding that can result from surfactant dysfunction, increasing interstitial fluid, and third-spacing.

High-frequency percussive ventilation (HFPV), while not as commonly used in the ED, is considered standard therapy in many burn centers. HFPV generates pulsatile flow at up to 600 cycles per minute, which entrains the humidified gas by its effect on molecular diffusion. It can improve clearance of airway secretions and allow continued patency of the lower
airways. In patients with inhalation injury and burns involving less than 40% of total body surface area, HFPV decreases both morbidity and mortality.

Not proven to improve survival compare to standard mode of ventilation with high PEEP

**Pulmonary Toilet**

As with many respiratory conditions, the use of chest physiotherapy is widely accepted in inhalation injury. The use of percutaneous cupping and postural drainage seem reasonable to clear airways of cellular debris and soot, thereby preventing atelectasis and obstruction. Obviously, care must be taken in attempting this in the presence of significant chest wall burns.

Encourage extubated patients to cough and deep breathe. In patients who are intubated, use gentle suctioning to remove mucus, debris, and sloughed epithelium. Fiberoptic bronchoscopy may be helpful in removing the debris and in facilitating pulmonary toilet.

**Tracheostomy**

The timing of tracheostomy continues to be controversial. Certainly, tracheostomy can be lifesaving for patients in whom endotracheal intubation is not possible, because of severe airway edema or burns. With early recognition of upper airway injury, this should be a rare occurrence.

Tracheostomy, especially through burned tissue, has an increased complication rate and risk of sepsis when compared with endotracheal intubation. Thus, most patients can be effectively managed with endotracheal intubation through the mouth or nose. In patients expected to have a long period of convalescence because of severe neurologic or pulmonary injury, however, tracheostomy may be desirable for patient comfort and is easy to maintain.

**Hyperbaric oxygen therapy (HBO)**

HBO therapy also displaces CO from intracellular stores and may improve mitochondrial function. HBO requires special facilities that are not available at all centers, resulting in a delay in treatment while the patient is transported to facility with HBO.

Hyperbaric therapy should be considered in those patients who have high carboxyhemoglobin levels >25%, who are unconscious, have other neurologic findings, or have severe metabolic acidosis (pH < 7.1). The benefit of treating patients 12 hours or more after CO exposure remains unproven.

**Epinephrine**

Adrenaline is used for severe bronchoconstriction, especially in patients with underlying reactive airways disease. This agent has alpha-agonist effects that include increased peripheral vascular resistance, reversed peripheral vasodilatation, systemic hypotension, and vascular permeability. The beta-agonist effects of epinephrine include bronchodilation, chronotropic cardiac activity, and positive inotropic effects. We are limited at this stage for use as adrenaline nebuliser with normo saline.

**Other Nebulizers**

Some studies have shown the use of Heparin in the form of aerosol provide a protective function and allow to clear different casts in airway. Its use is controversial.

**Fluid resuscitation in inhalation injury**
It has been a topic of controversy, most of these patients have combine smoke injury and skin burns requiring fluid resuscitation. Parklane Formula is the standard guideline to provide fluid at 4ml/kg/% Burns. However some studies have suggested to give 6 ml instead of 4 ml. This approach has been criticized because of risk of overloading lungs with fluid when there is already impairment function due to smoke injury. Thus others have suggested to reduce fluid resuscitation in this case to 2-3 ml/kg/% burns.

Prognosis

Most inhalation injuries are self-limited and resolve within 48-72 hours. The severity of direct pulmonary parenchymal injury depends on the extent of exposure and the type of inhaled toxins produced during combustion. Most patients do not manifest spirometry changes. Rare long-term sequelae include tracheal stenosis, bronchiectasis, interstitial fibrosis, and bronchiolitis obliterans.

The prognosis for mild-to-moderate exposures of toxic smokes is generally very good, with the usual outcome return to full recovery without sequelae. Metal fume fever is self-limited and usually resolves after a short period of observation. Exposure to white smoke in a military setting can lead to acute lung injury and in severe cases to ARDS. With more severe exposures, lungs may become severely damaged and develop chronic pulmonary fibrosis.

Children with acute pulmonary injury from toxic inhalations generally do well once supported through the initial period of inflammation and damage. Most of the pulmonary damage is self-limited and resolves within 2-3 days. The degree of recovery depends on the extent of the pulmonary parenchymal injury and subsequent hypoxic damage to the organs.
Perforated duodenal ulcer is one of the common surgical emergencies. The mortality following surgery for perforated duodenal ulcer (PDU) range between 6.9 – 10% globally.

Whereas the risk factors that are consistently implicated in mortality associated after surgery for PDU are well described, (table 1) an important cause of mortality (up to 56%) is the development of re-leak after PDU omental repair.

**TABLE 1: RISK FACTORS ASSOCIATED WITH MORTALITY FOLLOWING SURGERY FOR PDU**

<table>
<thead>
<tr>
<th>Risk Factor</th>
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<tbody>
<tr>
<td>presence of shock at admission</td>
</tr>
<tr>
<td>coexistence of significant illnesses</td>
</tr>
<tr>
<td>age &gt; 60 years</td>
</tr>
<tr>
<td>undertaking resection surgery</td>
</tr>
<tr>
<td>time delay between perforation and operation</td>
</tr>
<tr>
<td>preoperative blood urea and serum creatinine</td>
</tr>
<tr>
<td>size of perforation</td>
</tr>
</tbody>
</table>

**Re-leak following omentoplasty: the problem**

The rate of re-leak following omentoplasty has been reported to be between 2 – 7.6%. The literature on the mechanisms that predispose to re-leaks after PDU omental repair and the therapeutic options to address this catastrophe is sparse; one is reliant on guidelines based on retrospective reports.

A study by Kumar et al cited risk factors for re-leak following a Graham patch closure (operative techniques not detailed) (table 2)

**TABLE 2: FACTORS CONTRIBUTING TO RELEAK AFTER SURGICAL CLOSURE OF PERFORATED DUODENAL ULCER BY GRAHAM’S PATCH.**

<table>
<thead>
<tr>
<th>Risk Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 60 years</td>
</tr>
<tr>
<td>Pulse rate &gt; 110/minute</td>
</tr>
<tr>
<td>Blood pressure &lt; 90 mmHg</td>
</tr>
</tbody>
</table>
Haemoglobin < 10g/dl
Serum albumin <2.5 g/dl
Total lymphocyte count < 1800 cells/mm$^3$
Size of perforation > 5mm

Haemoglobin level, serum albumin and size of PDU were independent risk factors for prediction of re-leak on multivariate analysis. Low haemoglobin levels and serum albumin are well known factors influencing wound healing; correction of these factors (notwithstanding the urgent correction of haemoglobin level) is impractical. For this reason, the size of the PDU and the appropriate omentoplasty technique deserve consideration.

Omental patch techniques

The classic pedicled omental patch that is commonly performed for the ‘plugging’ of PDU was first described by Cellan-Jones in 1929$^5$ is accepted as the gold standard treatment, even though this technique has been erroneously attributed to Graham who (in 1937), described the use of a free graft of the omentum to repair PDU.$^6$ Prior to the Cellan-Jones technique, the standard treatment was, following excision of friable edge, the application of purse string sutures covered with an omental graft. In order to obviate narrowing of the duodenum, Cellan-Jones advocated an omentoplasty without primary closure of the defect. His technique consisted of placing 4–6 sutures, selecting a pedicled omental strand with a suture through it used to anchor the omental strand in the perforation; the sutures are then tied (figure 1). In the technique described by Graham a free omental graft was used; three sutures were classically used with a piece of omentum graft laid over these sutures, which are then tied. In both techniques, no attempt is made to actually close the perforation. The technique described by Cellan-Jones was modified by Karanjia et al$^7$; in the latter technique the omental pedicle is secured to the tip of a nasogastric tube passed through the PDU. The nasogastric tube is withdrawn for 5-6 cms before the omentum is secured to healthy serosa (figure 1).

A technique described as omentopexy whereby the PPU is sutured in one layer by three interrupted sutures with a patch of pedicled omentum used to reinforce the suture line is widely used (figure 1). This technique was noted to be associated with a greater morbidity and mortality compared to omental plugging (as described by Karanjia)$^7$. Regardless of the omentoplasty technique employed, careful securement and approximation of the sutures are important; in their report of 17 patients with re-leaks, Maghsoudi and Ghaffari$^8$ noted that the omental patch had gangrenous appearance in five patients and partial or complete separation of omental patch in all the patients who underwent re-laparotomy.
FIGURE 1: OPTIONS OF PERFORATED DUODENAL ULCER REPAIR

<table>
<thead>
<tr>
<th>Omental patch challenges</th>
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| An important decision, one of 5 (as structured by Feliciano\textsuperscript{9}) facing the surgeon treating the PDU, is whether an omentoplasty is sufficient or is a definitive ulcer operation required? The former brings to light the occasional large PDU, closure of which by the simple described method may predispose to a re-bleak. “Giant” PDU are technically difficult to repair due to the complex anatomy of the duodenum, and its marginal blood supply shared with the pancreas together with extensive tissue loss and surrounding inflammation. However, there are several reports that attest to the efficacy of omentoplasty in the management of a PDU up to a diameter of 3 cms. \textsuperscript{4,7,10}. While Schein’s comment that “do not stich the perforation but plug with viable omentum and patch a perforated ulcer if you can, if you cannot, then you must resect”\textsuperscript{11} has wide currency, conservative options other than omental plugging have been described; these include jejunal serosal patch, jejunal pedicled graft, proximal gastroenterology and gastric disconnection. The recommendations for the management of re-bleak following omentoplasty are based on the stability of the patient and findings at re-laparotomy. Together with reinforcement of the original omentoplasty, in the pre-proton pump era proximal gastric vagotomy without a drainage procedure was strongly recommended\textsuperscript{12}; alternate options included truncal vagotomy with antrectomy or gastroenterostomy. More recently (in 2011), in their series of 17 patients with re-bleak (from a total of 422 patients with PPD) Maghsoudi and Ghaffari\textsuperscript{8} undertook reinsertion of omental patch and subhepatic drainage in 13 patients, subhepatic drainage in 3 patients and jejunal serosal patch in 1 patient; the overall mortality was 59%. In the situation where reinforcement of the original omentoplasty is not feasible (friable, oedematous tissue, giant PDU, gross contamination), intubation of the PDU\textsuperscript{13} with a feeding jejunostomy or pyloric exclusion with gastroenterostomy should be considered. Ultimately, the choice of procedure will depend on operative findings, available technical expertise and the patient’s physiological reserve. (see Figure 2)
FIGURE 2: SUGGESTED ALGORITHM FOR LEAK OF REPAIRED PDU

PERFORATED DUODENAL ULCER

REPAIR OPTIONS

CELLEN JONES / GRAHAM / OMENTAL PLUGGING / OMENTOPLASTY

POST REPAIR LEAK

RESUSITATE PATIENT, RELOOK LAPAROTOMY / LAPAROSCOPY

STABLE PATIENT

UNSTABLE PATIENT

RE-INFORCEMENT OF OMENTOPLASTY

DEFINITIVE PROCEDURE

PYLORIC EXCLUSION GASTROENTEROSTOMY

WIDE DRAINAGE FEEDING JEJUNOSTOMY

VAGOTOMY + GASTROENTEROSTOMY

TRUNCAL VAGOTOMY + ANTERECTOMY

PROXIMAL GASTRIC VAGOTOMY

WIDE DRAINAGE FEEDING JEJUNOSTOMY
References


Introduction

Original practice of oesophagectomy for cancer was to perform an anastomosis intra-thoracically (Ivor-Lewis). Anastomotic break down in the thorax is associated with very high morbidity and mortality\(^1\),\(^2\), thus the move towards placing the anastomosis in the neck (McKeown)\(^1\),\(^2\). Although anastomotic break down may be even more frequent in cervical anastomoses, their morbidity and mortality are less severe compared to intra-thoracic break downs\(^2\).

Risk Factors for Oesophagogastric Anastomotic Break Down

There are a number of factors that can lead to anastomotic break down, some are technical and others are patient related\(^2\),\(^3\),\(^4\). The risk factors include preoperative albumin, loss of weight, poor respiratory function, poor nutrition, high intra-operative blood loss, hypoxia and hypotension. The surgeon’s objective should be to minimise the technical factors and optimise the patient’s condition pre-, intra-, and post-operatively to minimise breakdowns\(^3\),\(^15\). Uppermost in patient factors is nutritional status.

Definition of Anastomotic Breakdown\(^2\),\(^5\)

Anastomotic break down is said to have occurred if there is clinical or radiographic leak demonstrated.

Diagnosis of Anastomotic Leak\(^3\),\(^4\),\(^5\),\(^6\),\(^9\)

Anastomotic leak may be diagnosed by:

1. Demonstration of gastro-intestinal contents through the drains.
2. Such leak may be confirmed by letting the patient swallow methylene blue and this comes out through the drain.
3. Contrast study with water soluble material eg urograffin or gastrograffin. Barium should not be used.
4. Contrast CT scan.
5. Flexible oesophagogastroscoopy which can assess both the extent of anastomosis break down and stomach tube integrity or necrosis.

Pathology of Anastomotic Leak\(^6\),\(^2\)

The break down is usually at the oesophagogastric anastomosis, but can also be on the gastric tube “suture” line. If the colon or small bowel was used as the conduit, the break down is at the oesophago-colic or oesophago-intestinal anastomosis respectively. The relative frequency of anastomosis break down is that oesophago-intestinal is least frequent while oesophago-colic is most frequent. However the gastric tube is the most commonly used conduit after oesophagectomy for cancer for its simplicity\(^2\),\(^7\),\(^8\).

The leak may be contained in the mediastinum or dissipated within the pleural cavity. In the neck, it can also be confined to the area around the anastomosis or effuse through the drain or freely to the atmosphere if accompanied by cutaneous incision wound break down. It can also track down into the superior part the mediastinum. Contained leak may result in abscess formation while free pleural leak may lead to empyema thoracis.
Anastomotic breakdown may be early (less than 7 days) or late. Early breakdown is usually due to technical problems while late breakdown is usually from non-healing or necrosis of the anastomosis.

**Effects of Anastomotic Break Down** \(^{2,9,10}\)

Limited and contained leak may have no systemic effects as long as there is no florid sepsis. However if untreated it may lead to abscess formation with ensuing systemic effects. Free pleural leaks nearly always result in systemic effects such as systemic inflammatory response syndrome (SIRS), septicaemia and shock.

**Management of Anastomotic Leak** \(^{2,5,6,9,10,11}\)

A. *Intrathoracic Anastomotic Leaks*

I. **Contained mediastinal leak**

Small contained mediastinal leak may be managed non-operatively with broad spectrum antibiotics and adequate nutritional support. GI fluids must be kept away from the anastomotic site by active suction via NGT.

Large collection leaks will need draining. This can be achieved by percutaneous CT guided drainage (pigtail or similar device).

If the defect is large but not a near total disruption of the anastomosis an endoluminal self-expanding stent may be placed\(^{11,12}\).

II. **Free Pleural Leak**

Small leaks may be managed conservatively with intercostal tube drainage. GI fluids should be kept away through NGT suction and feeding should be by jejunostomy (better) if already *in situ* or TPN.

Large leaks require immediate attention to the anastomotic break down site. If only part of the suture line has broken down and there is no associated gastric tube suture line disruption or stomach necrosis a (metal) self-expanding endoluminal stent may suffice. If the anastomotic disruption involves a large part of its circumference or the gastric tube suture line is disrupted or there is necrosis operative management is mandatory. Depending on the presence and extent of necrosis or the general condition of the patient, a debridement and re-suture of the anastomosis could be undertaken. If there is stomach tube necrosis the best option is to debride and staple off the stomach remnant and staple of the oesophageal remnant as well, create a cervical diverting oesophagostomy and place a feeding jejunostomy if this had not been done. The pleural cavity is cleaned of all sepsis or decorticated as necessary. After the patient has recovered from effects of sepsis, continuity is re-established. In practice this may need oesophago gastric jejunal interposition graft or retrosternal colonic cervical oesophagogastric interposition graft.

B. *Cervical Anastomotic Leaks*

I. **Contained leaks**
Contained localised leak can be managed conservatively with broad spectrum antibiotics, nasogastric tube suction to removed or prevent intestinal content leakage and nil by mouth. Nutritional support, preferably by feeding jejunostomy if already created or TPN is crucial. If the collection is large this should be drain percutaneously.

**II. Free drainage via the neck drain**

This is managed by keeping the drain *in situ* to form a fistula. The fistula should close with adequate nutritional support via feeding jejunostomy.

**III.** The leak may track down into the superior part of the mediastinum. If the neck drain reaches there, this may suffice. However, if not then a suitable drain should be placed perhaps on high volume low pressure suction pump system. Some have described the use of T-tube designed to create a controlled fistula\(^\text{(13)}\)

**IV.** Complete disruption or gastric tube suture line disruption or necrosis needs surgical management and revision. If gastric tube necrosis to extensive, this should be take down and the remnant stapled off and the oesophagus brought out as a oesophagostomy for delayed later reconstruction with colon interposition graft.

**Prevention and Anticipation of Anastomotic Leaks after Oesophagectomy**

Because of the relative frequency of anastomotic leaks after oesophagectomy and their devastating effects especially of the intrathoracic anastomosis, certain anticipatory steps or procedures are undertaken as part of the initial operation.

There is no manner or mechanism by which anastomotic breakdown may be completely eliminated. Stapled and hand sewn anastomoses are accompanied by similar rates of break down\(^\text{(14)}\).

However a few anticipatory steps to minimise the effects of break down or provide effective management are prudent.

1. Nasogastric tube suction for 6-7 days post-op is assured by securing the NGT with nasal halter before the “routine” check contrast oesophagogram.

2. A pyloroplasty is fashioned to allow free gastric fluid drainage into the duodenum. Although gastric drainage is similar whether pyloroplasty was done or not in the long run, during the early phase gastric stasis is experienced due to the inevitable vagotomy that accompanies oesophagectomy. These patient may “vomit” and aspirate during the early phase of oral feeding.

3. A feeding jejunotomy is fashioned as an insurance that should anastomotic break down occur, good enteral feeding may be achieved. Enteral feeding is to be preferred over TPN.

4. Placement of the oesophagogastric anastomosis in the neck is to be preferred since morbidity and mortality after anastomotic break down is less than intrathoracic leak.
Summary

Anastomotic break downs after oesophagectomy may be devastating and associated with significant mortality particularly the intrathoracic anastomosis. Therefore many surgeons including the author, strive to place the anastomosis in the neck. Minor or contained leaks may be managed conservatively with or without direct drainage depending on the size of the collection with antibiotics. Major intrathoracic disruptions may be amenable to endoluminal self-expanding stent placement but near complete break down requires operative management. Surgery in fulminant leaks should follow the principle of damage control and GI continuity postponed until the patient has fully recovered from the inevitable sepsis. Establishment of continuity may entail colon interposition graft. Nutrition is paramount and anticipatory feeding jejunostomy should be fashioned during the initial operation since enteral feeding is the preferred route.

References


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At the outset I would like to refer you to the series of 3 articles on this topic published by our Critical Care group in Wound Healing Southern Africa.

Part 1. Why and when should the abdomen be left open? Wound Healing Southern Africa 2010;3(2):19-23

INTRODUCTION
At times it is crucial to leave the abdomen open following surgery for complicated abdominal pathology in critically ill patients. This is not a strategy to be taken lightly as it is a morbid, resource intensive procedure with many high risk complications. Despite surgical decompression patients with abdominal compartment syndrome still have a high morbidity and mortality rate. (1, 2, 3, 4,5) It should always be remembered that opening an abdomen necessitates having an immediate plan in place for a specific form of closure, be it short term, for instance, a modified vacuum dressing (vac pack); of indeterminate term, for instance, skin grafting; or a definitive method of closure such as component separation. The complexities of managing an open abdomen are many and vexing and our approaches continue to evolve rapidly. Many innovations have been devised in an attempt to successfully deal with some of the problems of an open abdomen. These have developed into a concept known as Temporary Abdominal Closure (TAC). TAC is any form of dressing applied to the abdomen after nonsuture of the abdominal wall. Well known versions of TAC include Bogota bags, the ABRA abdominal closure system and the modified vacuum dressing (otherwise known as the sandwich dressing or vac pack).

The short term aims of an open abdomen are to relieve intra-abdominal pressure and to achieve source control as in cases of sepsis, haemorrhage and necrosis. During this period protection of the gut against erosion should be prioritized. In patients where primary closure is not possible or is delayed, an intermediate phase develops which can be indeterminate in duration. Fistula formation risk is at its highest during this intermediate phase. In our experience split skin grafting (SSG) is the most practical solution to protect exposed bowel at this stage. It is easily accessible, maintains the physiological environment and it eventually allows patients to be managed on an outpatient basis. Patients are able to adapt well to the resulting ventral hernia. Definitive closure is still subject to the controversy of optimal timing. This indeterminate phase can be used to optimize wound maturation, normalize nutritional status and improve muscle function as conditions leading to an open abdomen are often associated with multi-organ failure during the intensive care stay.

The initial choice of abdominal closure type depends largely on the grade of open abdomen and how well it is likely to achieve the correlating goals. Commonly occurring complications used to delineate the grade of open abdomen, and how best to avoid or manage them with the use of appropriate grade dependent closure methods (TAC) will be discussed.
Table 1. Common complications of the open abdomen.

<table>
<thead>
<tr>
<th>LOCAL COMPLICATIONS</th>
<th>SYSTEMIC COMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Adhesions</td>
<td>• Derangement of fluid balance</td>
</tr>
<tr>
<td>o Causes stricture formation</td>
<td>• Electrolyte disturbances</td>
</tr>
<tr>
<td>o Form intra-abdominal loculations</td>
<td>• Promotion of a catabolic state</td>
</tr>
<tr>
<td>• Adhesions of viscera to abdominal wall and to other visceral organs make primary closure difficult and precludes subsequent surgery.(^7)</td>
<td>• Temperature regulation</td>
</tr>
<tr>
<td>• Fascial retraction</td>
<td></td>
</tr>
<tr>
<td>• Abdominal sepsis</td>
<td></td>
</tr>
<tr>
<td>• Fistula formation(^8)</td>
<td></td>
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</tbody>
</table>

THE IDEAL TEMPORARY ABDOMINAL CLOSURE METHOD

Taking the aforementioned complications into consideration, aim to utilize a dressing method with the following characteristics.\(^9,\(^10\)

1. The dressing must be readily available and inexpensive.
2. It should be easy to apply and remove, allowing access to the abdominal cavity without delay and without difficulty.
3. Nursing care must not be inhibited by the dressing.
4. It should not damage bowel, fascia or skin with repeated applications.
5. It should maintains the abdominal domain (physiological environment)
6. The dressing should prevent collection of peritoneal fluid, pus or blood, thus allow active clearance of fluid.
7. It should serve a barrier function, preventing evisceration and contamination.
8. Prevent abdominal compartment syndrome\(^7\)
9. Prevent fascial retraction and allow for a high rate of subsequent primary fascial closure
10. Prevent fistula formation
11. It must allow fistula(e) isolation if present
12. The dressing should prevent adhesion of viscera to the abdominal wall

PLANNING ONCE THE ABDOMEN IS OPEN

The choice of TAC is never a simple or standard decision. Each grade of open abdomen has specific goals. The dressing used must be best suited to fulfilling these goals. Therefore the appropriate choice of TAC is dependent on the grade of open abdomen. However the patient’s clinical condition may also influence the choice of TAC. Therefore it is crucial to plan the closure method at onset taking into consideration the grade of open abdomen and patient’s clinical condition.

Table 2: Working grades of the open abdomen\(^10\)

<table>
<thead>
<tr>
<th>Grade 1a</th>
<th>Clean abdomen without adherence of viscera to the abdominal wall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1b</td>
<td>Contaminated abdomen without adherence of viscera to the abdominal wall</td>
</tr>
<tr>
<td>Grade 2a</td>
<td>Clean abdomen with adherence of viscera to abdominal wall</td>
</tr>
<tr>
<td>---------</td>
<td>----------------------------------------------------------</td>
</tr>
<tr>
<td>Grade 2b</td>
<td>Contaminated abdomen with adherence of viscera to abdominal wall</td>
</tr>
<tr>
<td>Grade 3</td>
<td>Open abdomen with visceral fistula</td>
</tr>
<tr>
<td>Grade 4</td>
<td>The frozen abdomen (with or without entero-atmospheric fistulae)</td>
</tr>
</tbody>
</table>

From our experience, the initial management of the open abdomen typically involves short term TAC with the aim of early definitive closure. Early definitive closure is commonly accepted as primary fascial closure of the abdomen within 7 days. If you foresee abdominal closure within a very short time, for instance within 48 hours, cost vs. benefit of choice of TAC becomes an important consideration. In this case a “non-specific” vacuum dressing is useful, accessible and inexpensive. For management longer than 48 hours a more durable and robust but protective dressing may become necessary.

If closure within 7 days is not feasible, short term TAC is planned with the aim of indeterminate-term closure such as autologous split skin grafting (SSG). Grafting is then part of a planned ventral hernia strategy. If the patient’s condition does not reach a stage that allows for repeat surgery, then indeterminate-term closure may effectively become the definitive closure method.

Provided the patient recovers adequately from the initial insult, indeterminate closure may be followed by late definitive closure for example component separation. Adequate nutrition has proved to be a major difficulty during this recovery period and it may take months to years to reach this point. Time for wound maturation is also needed.

Available methods of TAC

Knowledge of the available methods of TAC enables the most appropriate choice to be made.

Making the appropriate choice of TAC can be challenging. Table 3 favours specific negative pressure dressings. From our experience it also appears that the specific negative pressure TAC methods yield the best results. It appears to be superior to the non-specific negative pressure dressing. This can be explained by the fact that specific negative pressure dressing have been adapted to cater for the precise requirements and goals of managing different types of open abdomens.

However, one must acknowledge that in open abdomens where one foresees closure within a short period of time, eg. 48 hours, the cost of using a specific negative pressure dressing may not be warranted and this is an important consideration.

Dressing Grade 1a & b open abdomens

When dressing the grade 1a & b abdomen the aim is to maintain the intra-abdominal environment to such a degree that early primary fascial closure is possible. To achieve this it is crucial to preserve the paracolic gutters (prevent adherence of viscera to abdominal wall) and prevent fistula formation. Added to that, fluids should be drained and sepsis prevented at all cost. Our preference to achieve this is using a commercially available specific type negative pressure dressing.
The dressing typically comprises of 5 parts:

1. A perforated, visceral sponge drape. This is used to envelop the viscera by extending deep into the paracolic gutters and drains fluid from the gutters and from between loops of bowel.
2. A large oval shaped sponge that fits on top of the exposed viscera and occupying the space between the two edges of the open abdominal wall.
3. A large adhesive drape to create a perfect airtight seal.
4. A connecting device that allows for negative suction and drains away excess fluid.
5. A controlled suction unit with alarms and a translucent, calibrated canister that collects drainage fluid.

If a Grade 1 abdomen is dressed with the aim of primary fascial closure, and this is not achieved within seven days, it is in the interest of general care and cost to revise the planning strategy. There is debate about whether or not continuation of the use of dressings to maintain paracolic gutters lessens the risk of abscess formation. However, it is currently commonly acceptable to change to a Grade 2 specific type negative pressure dressing, as this reduces cost, and allows for easier dressing changes. The patient will also not require as much visceral manipulation, and it is more reasonable to do these dressings outside of theatre, and in an ICU environment.

Table 3: Commonly used temporary abdominal closure (TAC) methods
<table>
<thead>
<tr>
<th>TAC</th>
<th>DESCRIPTION</th>
<th>PRO'S</th>
<th>CON'S</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bogota Bag</td>
<td>Bogota: 3-liter plastic irrigation bag is emptied and cut open so it lies flat or Silo (X-ray film bag) The edges are trimmed and sutured to the skin</td>
<td>Inexpensive Transparent Available in the operating room Ease of application Strong and able to prevent evisceration of pendulous viscera.</td>
<td>Requires suturing of peri-wound tissue resulting in tissue trauma Adherence of bowel to abdominal wall (14) No abdominal stabilization (14) Usually results in loss of fascia necessitating subsequent surgical intervention for hernia repair (14) Lack of fluid containment results in overall tissue damage Leakage from under bag can leave bed wet and increase the risk of worsening hypothermia and causes difficult nursing care No opportunity to isolate entero-atmospheric fistula High rate of fistula formation (14)</td>
</tr>
<tr>
<td>Retention sutures (ABRA)</td>
<td>Specifically manufactured for use in laparostomes – Silicone sheet, abdominal wall closure set, elastomer retainers.</td>
<td>Easy access into abdomen Sutures can be tightened at sequential dressings preventing fascial retraction (15) Primary fascial closure facilitated (15)</td>
<td>Expensive Requires measuring IAP Potential damage to peri-wound integrity No active removal of fluid No protection of visceral integrity High risk of fistula formation Needs to be used in conjunction with another dressing No abdominal stabilization</td>
</tr>
<tr>
<td>Wittman Patch</td>
<td>2 Bur Sheets are sutured to opposing fascia to fit the opening Velcro like Manual closure via tension on the sheet during dressing change</td>
<td>Prevents fascial retraction Allows stepwise re-approximation of fascial edges Permits final fascia-to-fascia closure (16) Reduces the need for hernia repair (16) Allows easy access to the abdomen for further surgical procedures</td>
<td>No active removal of fluid Requires measuring IAP No stabilization of internal contents Requires suturing of friable fascia Requires additional dressing for maintenance of rectus abdominus and dermal/epidermal reapproximation Lack of fluid containment results in overall tissue damage Liner pressure placed on abdominal wall could lead to fascial necrosis Expensive Sepsis (16)</td>
</tr>
<tr>
<td><strong>Mesh Closure</strong></td>
<td>Variety of products available: absorbable or non-absorbable ± Zipper for re-exploration Materials: PP, PTFE, Vicryl®, Marlex, Dexcon®, Polyglycolic acid, Gore®tex, Composite, Alloderm</td>
<td>Ease of placement Re-exploration Ability to open and close the abdomen at the bedside Increased strength compared to Bogota Permanent feeding tube can be placed (13)</td>
<td>Fistula formation (17) Rigid and irregular Adherence of mesh to viscera complicating further management. Very high rate of sepsis (up to 100% reported in some studies) Does not allow for fluid removal Delayed primary fascial closure Expensive Mesh extrusion Damage to surrounding fascia (13)</td>
</tr>
<tr>
<td>Non-specific Negative Pressure Dressing (aka Sandwhich/ conventional negative pressure or Vack Pack)</td>
<td>Fenestrated, non-adherent polyethylene sheet is placed over the viscera and covered with moist sterile towels / sponge eg Ligazano 2, 10 French silicone drains are placed and the wound is sealed with adhesive dressing Wall suction applied Sealed with surgical drape</td>
<td>Inexpensive Easy nursing care Intended for rapid closure Re-exploration at bedside possible Made from common materials found in the O.R.</td>
<td>Does not adequately deliver or regulate negative pressure Swabs become very hard on negative suction and can be injurious Risk of fistula formation Does not quantify drainage No alarms compared to regulation provided with V.A.C.® Therapy – large responsibility on nursing staff to ensure negative pressure is maintained Grafting required for closure much of the time Adherence of bowels to fascia probable</td>
</tr>
<tr>
<td>Specific Negative Pressure Dressing</td>
<td>Specifically designed for laparostomy Fenestrated, non-adhesive dressing covering viscera (between abdominal wall and viscera) Followed by a fenestrated sponge Covered by sterile adhesive drape Negative suction is applied and maintained</td>
<td>Prevents adhesion of viscera to abdominal wall Prevents fistula formation (13) Promotes granulation to prepare wound bed. Decreased oedema Clears fluid Provides closed, moist wound healing environment Enhances perfusion Draws wound closed Allows for fistula isolation Protects surrounding fascia (13)</td>
<td>Expensive (13) Experience of equipment required Learning curve to apply precisely Increased responsibility on nursing staff to ensure constant suction at set pressures is maintained (system is equipped with alarms) Not translucent</td>
</tr>
</tbody>
</table>
Dressing Grade 2a, 2b and Grade 4 (without fistula) open abdomens
When dressing a Grade 2 or a Grade 4 open abdomen, the method and timing of definitive closure will differ from Grade 1a and 1b dressings, due to established adherence of the viscera to the abdominal wall. The goals are to drain fluids, prevent sepsis, prevent fistula formation, and especially to achieve wound bed preparation for split skin grafting, as this is now a priority.

Split skin grafting

Once the wound bed has granulated sufficiently, SSG should be carried out on the wound. This should be done as early as possible, keeping in mind that skin takes to bowel very well, as it is highly vascular. The viscera and the abdominal wall can be grafted separately, or together. The latter can cause a problem with adherence of the graft, as the viscera and abdominal wall move independently of one another with respiration. Vacuum dressing could be applied successfully to the freshly grafted wound. This will keep the graft in place and clear excess fluid, if necessary. Remove this dressing after 4-5 days, or when soiled.

Dressing changes

Dressing changes are governed by the clinical scenario. If a patient still needs procedures in the operating room, such as bowel anastomoses, removal of packs or relook laparotomy, the dressing should be changed in theatre. It will then be carried out as frequently as the procedures are necessary. If a patient only needs simple washouts or inspection, the dressing may be changed in the ICU, provided that appropriate equipment and anaesthesia are available. Changing the dressing every 5-7 days is advised, but if the abdomen is septic, more frequent changing is preferable.

Conclusion

An open abdomen is a difficult situation to manage well. Many different temporary abdominal closure methods have been proposed in an attempt to deal with the morbidity and complications of this condition. In our experience, it appears that specific negative pressure dressings are the most effective form of TAC. They can be adapted to the needs of the various grades of open abdomen, and offer essential, practical advantages. These include fluid removal, the promotion of wound bed granulation, and the prevention of sepsis and fistulation. This promotes successful closure of the open abdomen.

References

MANAGEMENT CHALLENGES PRESENTED BY ENTEROCUTANEOUS AND
ENTEROATMOSPHERIC FISTULA IN CURRENT PRACTICE
B Singh, S Mewa Kinoo, R Naidoo
Department of Surgery, University of KwaZulu-Natal and King Edward VIII Hospital, Durban

Introduction

Enterocutaneous fistula (ECF) remains a surgical catastrophe that continue to present a formidable challenge in current surgical practice. Until 2 decades ago, issues pertaining to the development and management of ECF had attained a state of laudable clarity, notwithstanding the general absence of Level 1 evidence.¹

Advances in critical care together with a greater understanding of fistula pathology, behaviour and outcome have seen the management ECF evolve from an aggressive surgical option to a measured conservative approach. The latter approach, in addition to affording the detection and eradication of lingering intra-abdominal sepsis and correction of nutritional and metabolic imbalances, may expedite the spontaneous closure of ECF. This was prompted by Chapmans report (in 1964) that mortality associated with ECF decreased from 58% to 16% in patients who received more than 1500 kcal/d (largely delivered by the enteral route). Patients administered >3000 kcal/day were reported to have a mortality rate of 12%, and a fistula closure rate approaching 90%. Those patients unable to maintain this caloric intake continued to have a high mortality rate and low fistula closure rate (55% and 37%, respectively).² ³

The addition of technical innovations, improvements in diagnostic modalities and the availability of novel therapies to the therapeutic armamentarium of ECF management had led to the reduction in mortality rate for this condition to between ³.5% to 37%.¹ ⁴ ⁵

This landscape has been considerably altered in current surgical practice with the wide usage of open abdomen (OA) techniques to address abdominal compartment syndrome. The occurrence of an enteric “fistula” in the midst an open abdomen (OA) is called an “entero-atmospheric fistula” (EAF) - inappropriately because it does not have a fistula tract, the absence of which invariably precludes spontaneous closure of the “fistula”.

The challenges presented by EAF are considerable; the fluid, electrolyte losses with acid–base derangement and sepsis source control are much more challenging compared to ECF, resulting in an unremitting hypercatabolic state. Furthermore, there are difficulties in effective collection of enteric effluent. Spillage of enteric contents of an EAF on the adjacent OA surface continuously impairs the healing process, which aggravates local wound sepsis. These challenges have ensured that the morbidity rates of EAFs are high (compared to ECF). The principles of care for EAF are based on the tenets of ECF management that are sequential:

1. Initial resuscitation
2. Early recognition and management of sepsis
3. Nutritional support
4. Reducing the fistula output
5. Wound care / controlling the fistula
6. Surgical intervention

1. Initial resuscitation
This involves the correction of fluid, electrolyte and acid-base imbalances
2. Early recognition and management of sepsis

This involves, source control, drainage of sepsis and antibiotic therapy

3. Nutritional support

The patient with an EAF is invariably hypercatabolic due to sepsis associated with an underlying starvation metabolism. The metabolic needs of the patient can be estimated using the Harris-Benedict equations taking into account appropriate modifiers for sepsis and postoperative states. The rate of output from the fistula also greatly affects nutritional needs. The requirements for low-output fistulas range 25 to 30 kcal/kg/d with a protein need of 1.5 to 2 g/kg/d of protein; high-output fistulas may require up to 2 times the overall caloric daily requirement and 2 to 2.5 × baseline protein requirements to achieve a positive nitrogen balance; daily small bowel secretions may contain up to 75 g of protein, material that would ordinarily be reabsorbed.

Following the correction of fluid and electrolyte and eradication of sepsis, minimizing the fistula discharge and establishing a positive nitrogen balance are crucial. Total parenteral nutrition (TPN), by affording bowel rest and rapid repletion of nutrition, is recommended during this phase of the patient’s management. TPN decrease fistula volume and offers a significant improvement in mortality and fistula closure rates.

In addition to maintaining gut mucosa, enteral nutrition may also decrease fistula output. The supplementation with immunonutrition has failed to demonstrate improvement in mortality.

Following the initial period of bowel rest, it is reasonable to advocate enteral nutrition. Adequate nutritive absorption may be feasible with at least 1 – 1.5 meters of functioning small bowel remaining.

Enteral feeding must be commenced cautiously, with continuous, low volume feeds delivered via soft post-pyloric feeding tube; TPN must be maintained while nutritional goals are sought. For gastric feeding, osmolality is increased slowly to hyperosmolar targets, followed by volume targets. With small bowel feeding (via feeding jejunostomy or postpyloric feeding tube), volume tolerance needs to be achieved first; this may be difficult in the high-output fistula as enteral feeds can increase the volume of fistula output.

The use of fistuloclysis (feeding into the efferent limb of a fistula), either with enteral formulas or chyme output from the proximal fistula has gained currency (particularly in resource depleted services) and has the appeal of being undertaken at home with little.

4. Reducing fistula output

Reduction of fistula output may be achieved by keeping the patient nil per os and effective drainage of the stomach via a nasogastric tube. TPN affords adequate bowel rest as well as reducing GI secretions. Reduction of GI secretions maybe further achieved by the use of drugs; these include proton pump inhibitors to reduce acid secretion, somatostatin or analogue (octreotide) to reduce enteric and pancreatic secretions (but no evidence to show effect on fistula closure) and drugs that slow intestinal transit time such as loperamide, diphenoxylate, and opioids.

5. Wound care / controlling the fistula
Wound care management and the effective collection of the enteric effluent with isolation of the EAF opening and the prevention of the contamination of the adjacent wound are central to the management of the abdominal wound in the patients with EAF. To this end there are several methods available using the principle of negative pressure wound therapy [the “VAC (vacuum assisted closure) systems”] that have been shown to be effective in expediting the management of the OA;13 Among these methods are the “Floating stoma”14, the “Fistula VAC”15, the “Tube VAC”16, the “Nipple VAC”17 and the “Ring and silo VAC”18 techniques. The challenge presented by EAF in an OA is reflected in desperate technique of Malecot intubation of the EAF and then tunnelling this through adjacent well vascularised tissue to convert an EAF to an ECF19.

While there are anecdotal reports of low output fistulas closing spontaneously following conservative medical treatment and VAC application to the OA, the presence of factors that would preclude spontaneous closure (such as muco-cutaneous continuity, distal obstruction, length of fistula tract, among other factors) have to be considered when using VAC systems. The value of VAC systems reside in its expediting the healing of the wound around the EAF.12, 21 The measures described for the management of EAF together advances in critical care and surgical care have ensured that mortality rates have decreased from as high as 70% in the past decades to about 42%.20 The challenge presented by EAF is reflected in it being classified as Grade 3 as per Bjorck’s classification of the OA, wherein Grade 4 is the frozen and inoperable OA.22

Notwithstanding the value and advances of conservative treatment of ECF and EAF, surgery remains the cornerstone of fistulas that have not closed spontaneously. Definitive surgery for the closure of small bowel ECF or EAF is demanding in terms of the physiological reserves of the patient, surgical technical expertise and hospital resources.

The failure of small bowel ECF or EAF to consistently close spontaneously underscores the need to develop reproducible surgical strategies that would ensure a favourable outcome. However, even in experienced hands, a re-fistulation rate between 9% and 32.9% after definitive surgery has been reported. It is therefore necessary that the surgeon treating patients with small bowel ECF or EAF become facile with surgical strategies and techniques that would ensure a safe and effective procedure.

The resection of the involved enteric loop is the most definite way of treating an ECF or EAF. Surgery should only be undertaken in patients in good general status and free of infection.20 These conditions are possible to attain in some patients as early as 1–2 months,20, 23 while others accomplish this target after 6–12 months or even after a 1-year period24 and will affect the timing of the surgery.

6. The principles of surgical intervention

- With the progressive decrease in fistula output at the 6 week stage, return of bowel activity and restitution of nutritional status, it is reasonable to pursue with conservative treatment with the expectation of spontaneous closure of the fistula. However, fistulas with factors known to prevent spontaneous closure or those persisting for >2 months are unlikely to close spontaneously.1
- The timing of the surgical intervention in relation to the most recently undertaken laparotomy is crucial because it impacts the rate of further complications. Furthermore, dense, vascular adhesions, the legacy of oblitative peritonitis, are most evident between 3 and 12 weeks; surgery during this period may predispose to fistula recurrence.
• Gaining safe access into the invariably scared abdomen is facilitated by siting the incision in the sub-xiphoid area where the liver protectively overlies bowel loops.
• In mobilising the small bowel, begin in an area considered “hospitable”. Unravel the entire small bowel from the ligament of Treitz to the caecum, thereby unravelling all sites of small bowel obstruction.
• Resecting the bowel segment bearing the fistula and repairing, rather than direct suture closure is a principle that has ensured a low rate of fistula recurrence. However, depending on the extent of the fistulous opening and absence of oedema, friability or intrinsic bowel disease (such as inflammatory bowel disease), a wedge resection may be adequate.
• During the procedure, the thinned out small bowel is vulnerable to injury by overzealous handling; for this reason the use of bowel clamps is inadvisable.
• Apply diligence in ensuring that no iatrogenic perforations are overlooked.
• Repair all serosal tears because these may predispose to spontaneous perforation in the context of postoperative ileus or intestinal obstruction these patients are vulnerable to.
• Routine stenting of the small bowel has been recommended following repair of the fistula. ¹

Conclusion

Although tempting, surgery as primary management for fistula closure without adequate patient optimization is highly unsuccessful. Notwithstanding this approach, adequate patient optimization, remains a long drawn out process and requires patience from both the surgeon and patient. Furthermore while optimizing these patients for eventual surgery, fistulas may close spontaneously negating surgery and the risks associated with it. It is well documented that the success of optimization lies in providing and maintaining an adequate nutrition and preventing sepsis. In the event that a patient requires surgical repair such as in a case of EAFs, strict surgical principles should be adhered to avoid recurrence of fistulas.

References

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RECTOVAGINAL FISTULA (RVF)
Dr BH Pienaar

The term denotes an epithelial lined tract from the rectum to the vagina. The majority of RVFs will be found in close proximity to the dentate line, either at or just above this landmark.

It is important to distinguish a fistula below the dentate line from those proximal to the line as these should be called anovaginal fistula. This aspect is important as the treatment is different.

There are a number of ways in which RVF may be classified namely location, etiology and/or size.

The most practical is probably by location as this would also indicate which approach should be used to address the problem.

**Perineal approach:**
This would be used for RVF where the fistula is situated in the lower third of the rectum. Alternatively a low fistula.

**Transabdominal approach:**
This would be used for a fistula which occurs in the middle third of the rectum and posterior vaginal fornix.

Size may be used as a differentiating parameter.

1. Small < 0.5 cm
2. Medium 0.5 – 2.5 cm
3. Large > 2.5 cm

**Etiology**
1. Obstetric trauma (most common)
2. Radiation injury
3. Iatrogenic trauma
4. Neoplasm
5. Inflammatory bowel disease (Crohn’s)

**Pathophysiology**
Obstetric trauma may manifest in different ways where the failure to correct perineal defects or secondary infection of episiotomy may lead to fistula formation. In years gone by prolonged labour with pressure from the foetal head was a major cause of RVF.

Lately the stapled resection procedures performed in the vicinity of the dentate line have been associated with RVF.

Crohn disease often is associated with RVF due to perirectal abscess.
Diverticulitis and/or abscess may result in RVF.
The most vexing and difficult to manage are fistulas that result from radiation therapy in pelvic malignancies.

In all conditions but especially the latter previous pelvic surgery, smoking and diabetes increases the risk for the development of fistula.

Other rare causes include tuberculosis, lymphogranuloma venereum.

**Symptomatology**
Faeces and flatus are passed through the vagina. It is often associated with diarrhoea which makes the symptoms worse. Severe vaginitis and cystitis may be the presenting symptom. Rarely patients may be found to be asymptomatic.

The clinical picture may also present as incontinence.

**Examination**
Routine standard examination in the rooms may provide the correct diagnosis. It is important to determine sphincter function at the initial examination.

A simple test is to place a vaginal tampon whilst methylene blue is instilled into the rectum. The tampon can be inspected after 15-20 minutes when the blue discoloration of the tampon will confirm the diagnosis.

**Treatment**
Surgical correction is the only method that may result in complete healing. This may take the form of repair and in some cases diversion by colostomy. Colostomy may be used in repair of recurrent fistulas or post radiation repairs.

Biopsy of the fistula prior to repair is essential.

**Anatomy**
Thorough knowledge of the anatomy in the region is important in order to effect successful repair. The rectovaginal septum is a thin layer of tissue where the perineal body is the most caudal portion of this septum with the anal sphincter complex closely related to the perineal body.

**Imaging**
Barium enema and CT scan may be utilised in conjunction with endorectal and vaginal sonography.

**Surgical therapy**
Preparation should always include complete mechanical bowel preparation. Some authors recommend poorly absorbable antibiotics such as neomycin. Prophylactic SYSTEMIC antibiotic use is recommended.

Local repair
Transanal advancement flap repair. (Prone position & operating anoscope)

The flap consists of mucosa and submucosa. The tract is curetted whilst the fistulous opening in mucosa is excised. The fistula is closed with circular muscle. The flap is sutured in position with interrupted sutures. The vaginal opening of the fistula is left open.

Transabdominal repair

High fistula necessitates this approach especially if complicated by radiation, inflammatory bowel disease and diverticulitis. The affected bowel must be resected. Omentum may be used as an interposition buttressing structure.

Prognosis

Trans-anal advancement flap.

Expects success in 77-100% is reported in different series. It is extremely important that sphincter repair is performed simultaneously or that it is intact.

Subsequent childbirth should be by caesarean section especially if sphincter repair had been part of the initial procedure.

Recurrent fistula makes subsequent successful repair more difficult and prone to failure. Rectal sleeve advancement may play a role.

Transabdominal procedures

Due to the fact that the etiological factors are usually more advanced pathology the outcome is less favourable.

REFERENCES


Most surgeons report an increase in cholecystectomies performed especially in the public sector in SA. This reflects a change in incidence of choledocholithiasis in South Africans, however data confirming this remains limited. There is also limited data in South Africa regarding the magnitude of the CBDI after laparoscopic surgery. This includes the incidence and the management strategies. It is recommended that these cases be centralized to centers with the multidisciplinary skills to manage the problem.

There are a number of consequences of the CBDI that we need to unpack. Firstly the impact of the injury on the patient, the doctor and the medico legal process. There is clear evidence today that CBDI does have a long term impact on the patient in terms of quality of life and even a suggestion that there may be an impact on long term survival. Even a well done timeous repair has consequences. Long term stricture formation of the anastomosis is well described at about 10%.

With rising medico-legal claims the legal process needs to be explored. For patients to receive some form of compensation in SA law, there must be a finding of negligence. The implications are that an adverse event is always a consequence of negligent behavior on the part of the doctor. While surgical misadventure is nearly always present there is good evidence today that this is not commensurate with negligence. There is an impact on the surgeon during the process of litigation including significant stress, anxiety and even an impact on productivity due to a loss of self-esteem during the legal process hinged on negligence. A change in this legal structure may allow a less adversarial resolution for both the patient and the surgeon. It is however worth noting that often the legal culpability resides in issues of adequate consent and the appropriate management of the patient once the injury has occurred.

Is there something that can be done to reduce the incidence of CBDI? The literature is clear that improved training and better intraoperative strategies can reduce the incidence of CBDI. Psychomotor studies have shown that there are normal human errors brought into play which are not in themselves negligent. Better training and greater experience in laparoscopic training is required. A recent paper in the SAJS demonstrated a significant lack of laparoscopic equipment in training institutions reducing the supervised training of registrars. Data showing a greater incidence amongst less experienced surgeons supports this argument.

Strasberg and colleagues have described an operative strategy that has now been evaluated in a number of studies with a real reduction in the incidence of CBDI. This approach relies on better dissection of the anatomical structures in Calots triangle considered to be the ‘Critical view of safety’. This approach is possible in the majority of patients and when not possible it should be an indication to convert the procedure to an open laparotomy for safe completion of the cholecystectomy. Conversion should not be seen as failure by the surgeon but rather one of a number of ways to prevent CBDI. Others include subtotal cholecystectomy, preoperative prediction of difficult cholecystectomies and
the increased use of cholecystostomy in the severely inflamed Gallbladder. Predictors of the difficult cholecystectomy become less obvious as the skills of surgeons improve. Previous predictors no longer routinely apply however the presence of an impacted stone in Hartman’s pouch compressing the common hepatic duct (Mirizzi syndrome) is still considered a contraindication to LC. However obesity, cirrhosis and acute cholecystitis no longer constitute absolute contraindications. The training of registrars needs to ensure that all these approaches are well taught. In a recent fellowship exam this question was asked with the expectation that all candidates would understand the implication of this technique, yet the response was poor This suggests that there is a deficit in the training of registrars.

There are a number of other recommendations that remain controversial. The role of intraoperative cholangiograms (IOCG) remains a topic of debate. Two broad approaches are described; IOCG has not proven to reduce the incidence of CBDI especially when performed on demand. Its routine use is probably more appropriate to identify undetected CBD stones and the information regarding the anatomy probably does not prevent CBDI. There are those who believe that routine on table Cholangiograms will reduce the incidence. Most centers in SA do not practice routine IOCG.

It is well recognized that only symptomatic cholecystolithiasis requires cholecystectomy. After the introduction of Laparoscopic cholecystectomy and the acceptance that this was the standard of care, the numbers of cholecystectomies reported globally increased. It was thought that this was due to a relaxation in the indications for the procedure. Patients and health professionals must accept the current guidelines for cholecystectomy; only symptomatic or complicated gallstones should be treated by cholecystectomy.

A high index of suspicion for CBDI in patients who do not display an appropriate clinical response in the immediate post-operative period, is essential to early diagnosis. The global incidence of injuries detected post operatively, as opposed to intraoperative identification, remains high. The complex management of patients with delayed diagnosis, is responsible for a significant component of the high cost as a result of increased need for ICU, more investigations longer hospital stay and in many studies reflects the patients at risk for mortality.

There is good evidence to support the early referral of these patients to centers able to manage patients who require interventional radiology and advanced surgical skills. The training of surgeons must stress the appropriate initial management of these patients before referral to these centers. Preventing biliary peritonitis by adequate drainage of bile is far more important that the immediate repair of the injury. This does not require expert skills rather an awareness of the need for adequate drainage. Once this has been established early transfer before expensive investigations such as MRCP, CT scanning or endoscopy is important. This may result in some unnecessary transfers but overall this may improve the outcome for patients who require the skills of the experienced centers.

Strasberg A or D injuries can both be managed with endoscopic interventions. These patients should be included in the generic strategy for the recognition and management of CBDI.
There is no doubt that we can do better in the prevention of CBDI following laparoscopic cholecystectomy. The strategy must involve all agencies involved in training, assessment and accreditation. The Minister of Health has recently identified the need to address the rapidly rising number of medico-legal claims and is clear that all these agencies need to participate in addressing this trend in the best interests of patients but also to prevent depletion of scarce resources available to the health sector. We should all revisit our training programs and ensuring that everyone plays their part in the overall prevention and management of these patients to reduce the consequences to the patient and to reduce the health expenditure.

**Recommended reading**

POST-HEPATECTOMY LIVER FAILURE
Dr C Jeske, Department of Surgery, University of Pretoria and Steve Biko Academic Hospital

Introduction

Post-hepatectomy liver failure represents a major source of morbidity and mortality after liver resection. It is considered to be a devastating complication with little treatment other than vigilant supportive care. The incidence of post-hepatectomy liver failure ranges from 4-19%. In recent literature, the incidence is less than 10%, owing to improved preoperative assessment and intraoperative and postoperative management. Several studies report a lower rate of PHLF is East Asian countries at 1-2%. Mortality following partial hepatectomy in the past two decades still ranges from 0 to 6%, however, and PHLF has been implicated as contributing to mortality in the majority of cases.

Definition

An excess of 50 studies have defined PHLF. This wide variety of definitions among groups have made comparison of rates between studies challenging. The time period in which liver failure is defined is also contentious, some using 3 days others 7 days. Prior to this decade there has been no uniform definition of PHLF. A more widely accepted recent definition is that of the International Study Group of Liver Surgery (ISGLS). In 2011 they defined PHLF as the “impaired ability of the liver to maintain its synthetic, excretory and detoxifying functions, which are characterized by an increased international normalized ratio and concomitant hyperbilirubinemia (according to the normal limits of the local laboratory) on or after postoperative day 5.” The ISGLS group also advocated a grading system in which laboratory values, clinical symptoms, and need for increasingly invasive treatments define severity of PHLF (see Table 1).

<table>
<thead>
<tr>
<th>Grade</th>
<th>Clinical</th>
<th>Treatment</th>
<th>Diagnosis</th>
<th>Clinical symptoms</th>
<th>Location for</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Deterioration in liver function</td>
<td>None</td>
<td>1. UOP &gt;0.5 mL/kg/h</td>
<td>None</td>
<td>Surgical ward</td>
</tr>
<tr>
<td>B</td>
<td>Deviation from expected postoperative course without requirement for invasive procedures</td>
<td>Non-invasive: fresh frozen plasma; albumin; diuretics; non-invasive ventilatory support; abdominal ultrasound; CT scan</td>
<td>8. UOP ≤0.5 mL/kg/h: BUN &lt;150 mg/dL; &lt;90% O2 saturation despite oxygen supplementation; INR ≥1.5, &lt;2.0</td>
<td>32. Ascites: Weight gain; Mild respiratory; Confusion</td>
<td>Intermediate unit or ICU</td>
</tr>
<tr>
<td>C</td>
<td>Multi-system failure requiring invasive treatment</td>
<td>Invasive: hemodialysis; intubation; extracorporeal liver support; salvage hepatectomy; vasopressors; intravenous glucose for hypoglycemia; ICP monitor</td>
<td>• UOP ≤0.5 mL/kg/h • BUN ≥150 mg/dL • ≤85% O2 saturation despite high fraction of inspired oxygen support</td>
<td>• Renal failure • Hemodynamic Instability • Respiratory failure</td>
<td>ICU</td>
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The ‘50-50 criterion’ (PT <50% and bilirubin >50 \(\mu\)mol/L on post-op day 5) have been proposed to predict post-hepatectomy mortality.\(^{10}\) There are also comprehensive algorithms for predicting PHLF. Others have proposed a peak total serum bilirubin of greater than 7mg/dL.\(^{11}\) Table 2 summarizes current post-operative predictive models of PHLF.

Despite efforts by the ISGLS to define PHLF more accurately to predict prognosis early after hepatectomy, several studies have questioned the accuracy of the ISGLS criteria. Skrzypcyk et al. compared the ISGLS definition with the ‘50-50 criteria’ and peak bilirubin >7 mg/dL criteria among 680 patients who underwent either minor or major hepatectomies.\(^{13}\) The ISGLS definition was found to be least predictive of both the occurrence of major complications (positive predictive value of 49.2% for ISGLS vs. 78.9% for ‘50-50 criteria’ and 65% for peak bilirubin >7 mg/dL), as well as the risk of post-operative death (OR=6.9 for ISGLS vs. OR=21.1 for ‘50-50 criteria’ and OR=21.7 for peak bilirubin >7 mg/dL).\(^{13}\)

<table>
<thead>
<tr>
<th>Table 2. Predictive models of PHLF</th>
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<tr>
<td><strong>Postoperative Models</strong></td>
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<tr>
<td>MELD Score</td>
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<tr>
<td>Child-Pugh Score</td>
</tr>
<tr>
<td>50-50 Criteria(^{10})</td>
</tr>
<tr>
<td>Kim et al.(^{14})</td>
</tr>
<tr>
<td>Snap peak bilirubin&gt;7 mg/dL(^{11})</td>
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<tr>
<td>ISGLS Definition(^{9})</td>
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<tr>
<td>Hyder et al.(^{12})</td>
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Risk Factors

Identification of risk factors for PHLF is critical to help identify patients most at risk of developing PHLF and to develop strategies aimed at decreasing the incidence and mortality associated with PHLF. Independent risk factors can be categorized into three main categories: patient-related, liver-related, and surgery/postop-related factors (Table 3).

Patient-related factors

These include age, male gender, malnutrition, diabetes, and American Society of Anesthesiology (ASA) score. Some studies had implicated older age as a risk factor for PHLF; however other studies have documented the safety of liver resection in the elderly. Animal studies have suggested a loss of the liver’s regenerative capacity, as well as impaired liver function with increased age. Aldrighetti et al, Nanashima et al and Kim et al have all failed to detect a difference in age-related PHLF in their studies.

Diabetes, either alone or in combination with metabolic syndrome, has also been associated with greater risk of PHLF. The association of diabetes with risk of PHLF may be due to the important role insulin plays in the regulation of hepatocyte function and regeneration. Specifically, a lack of insulin has been noted to cause hepatic atrophy in animal models. Furthermore, 80% of peri-operative deaths in diabetic patients were secondary to PHLF. Excess mortality seen in diabetic patients undergoing major hepatic resection is likely multifactorial, with alterations in liver metabolism, decreased immune function, and hepatic steatosis contributing to post-operative liver dysfunction.
Liver-related Factors

Patients undergoing hepatectomy present with a wide range of underlying hepatic parenchymal disease including cirrhosis, steatosis, steatohepatitis, and chemotherapy induced liver injury that can affect the ability of the liver to regenerate after liver resection.

Cirrhosis is a major consideration when operating on patients, especially in the hepatocellular carcinoma (HCC) population. Animal models have demonstrated that after resection, cirrhosis is associated with decreased levels of hepatocyte growth factor, impaired transcription factors, and a reduction of DNA synthesis, leading to lower volumes of regenerated liver. In patients with cirrhosis, PHLF rates range between 2% and 19%. Child-Pugh score and Model for End-Stage Liver Disease (MELD) may give some information about the degree of cirrhosis, they may underestimate the ability of a patient to withstand resection. In these patients preoperative tests for functional liver reserve, such as Indocyanine Green clearance (ICG), are considered essential.

As neoadjuvant and conversion chemotherapeutic strategies improve, the percentage of patients undergoing liver resections after exposure to chemotherapy is increasing. Common regimens for the treatment of colorectal cancer include 5-fluorouracil combined with oxaliplatin, irinotecan, and the targeted agents bevacizumab and cetuximab. Although these regimens have helped to make previously unresectable metastases resectable, their prolonged use can result in steatosis, steatohepatitis, and sinusoidal congestion. Several studies have associated sinusoidal injury and steatohepatitis with compromised liver regeneration as well as increased morbidity following hepatic resection.

Surgery-related Factors

In addition to patient-specific factors, the performance of the surgical procedure itself influences risk of PHLF in both the immediate postoperative period and in a delayed manner. Intraoperative blood loss and transfusion requirements have been associated with an increase in postoperative complications following hepatectomy. Postoperative blood transfusions required due to intraoperative blood loss, results an immunosuppressive effect that may contribute to PHLF.

Table 3. Risk factors for PHLF

<table>
<thead>
<tr>
<th>Patient-related factors</th>
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<tr>
<td>Age (&gt;65 years)</td>
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<tr>
<td>Male gender</td>
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<tr>
<td>Metabolic disorders</td>
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<td>Preoperative chemotherapy</td>
<td></td>
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<tr>
<td>Sepsis Malnutrition</td>
<td></td>
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<tr>
<td>ASA score</td>
<td></td>
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<tr>
<td>Liver-related factors</td>
<td></td>
</tr>
<tr>
<td>Grade of the tumour</td>
<td></td>
</tr>
<tr>
<td>Hepatitis</td>
<td></td>
</tr>
<tr>
<td>Portal venous pressure</td>
<td></td>
</tr>
<tr>
<td>Cirrhosis</td>
<td></td>
</tr>
<tr>
<td>Cholestasis</td>
<td></td>
</tr>
<tr>
<td>Surgery-related factors</td>
<td></td>
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<tr>
<td>Complex operations</td>
<td></td>
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<tr>
<td>Extent of resection</td>
<td></td>
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<tr>
<td>General surgical models</td>
<td></td>
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<tr>
<td>Large blood transfusion</td>
<td></td>
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<tr>
<td>Left hepatectomy</td>
<td></td>
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<tr>
<td>Duration of Pringle Maneuvre</td>
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</table>
An important surgery-related factor is the extent of resection and avoidance of ‘small-for-size’ liver remnant following hepatectomy. Small for size livers are associated with an increase in severe graft dysfunction with increased hepatocyte injury, hyperbilirubinemia, prolonged PT, portal hypertension, and ascites. Efforts should be taken preoperatively to predict adequate future liver remnant (FRL) in an effort to decrease the risk of PHLF. In patients for whom <25% of the pre-operative liver volume is left post-resection, the risk of PHLF is 3 times that of patients with >25% of liver volume remaining.

Preoperative Risk Assessment

Given the high mortality and morbidity rate associated with PHLF, there has been great interest in techniques to preoperatively identify patients at high risk for hepatic dysfunction or failure. There are multiple different techniques available to evaluate the quality and quantity of the FLR and hence the risk for developing PHLF.

Quality assessment of the liver

Traditional liver function markers and clinical scoring systems

Two tools used widely for assessment of liver function are the Child-Pugh score and the MELD score. Both scoring systems were originally developed to grade chronic liver disease and cirrhosis in liver transplant candidates; however, both are currently also used to screen patients preoperatively for the risk of PHLF as well as to evaluate perioperative liver function. Patients with advanced Child-Pugh B and C scores are not candidates for hepatectomy due to their risk of PHLF. Child-Pugh classification falls short in predicting PHLF-related mortality in the more modest to mild cirrhosis patients. The MELD model has similarly been extensively evaluated with mixed results.

Indocyanine Green Retention at 15 min (ICG-R15)

ICG is a water-soluble, nontoxic fluorescent dye that is injected intravenously and is eliminated almost exclusively by the liver. The absorption and emission spectrum of ICG are both in the near infrared range allowing for measurements to be performed by non-invasive monitoring. The ICG-R15 test has been shown to predict more accurately PHLF compared with both the Child-Pugh classification and MELD model. There is however no clear consensus on the cut-off value for ICG-R15 allowing for safe hepatic surgery and while the test is used in the east, its adoption has not been widespread in western centers. Some studies suggest a cut off of 14% and others report higher cut-off values in relatively younger patients of 17%.

Other Liver Function Tests for the Quality Assessment

Other qualitative tests based on the principle of clearance of substrate by the liver include substances such as lidocaine, galactose, aminopyrine, amino acid, and methacetin. None have been proven to be superior to ICG-R15 for the prediction of PHLF or PHLF-related mortality. There are also tests based on the synthetic functions of the liver such as serum levels of hyaluronate and type IV collagen, energy production of the liver, and the number of receptors for asiaglycoprotein (GSA scintigraphy). The high cost and complexity of these tests has limited their clinical implementation to date.

Quantity Assessment of the Liver

Preoperative assessment of the FLR size after hepatectomy is fundamental for effective and safe hepatic resection. Currently, there is no uniform consensus regarding the limit of the FLR volume necessary to achieve a ‘safe’ liver resection or the modality most effective for evaluating FLR size preoperatively. The techniques most frequently used to assess FLR
include computed tomography (CT) and magnetic resonance imaging (MRI). CT and MRI both show excellent accuracy and precise quantification of hepatic volume. Additionally, careful evaluation of preoperative CT imaging should focus on liver attenuation, indicating steatohepatitis, as well as splenomegaly, varices, ascites, or consumptive thrombocytopenia, to raise suspicion of underlying cirrhosis.

**Strategies to Prevent PHLF**

Treatment of PHLF hinges first on its prevention. The preventive techniques are applied to those patients identified to be at risk of PHLF based on the preoperative assessment as outlined above, i.e. those patients deemed to be at high risk based on underlying patient factors, presence of cirrhosis, preoperative laboratory values, volume of liver to be resected or estimated FLR after resection.

Most preventive strategies have been aimed at increasing the FLR. Techniques aimed at increasing the FLR are portal vein embolization (PVE), portal vein ligation (PVL) and associating liver partition and portal vein ligation for staged hepatectomy (ALPPS) procedure. PVE is an ultrasound-guided percutaneous procedure that induces liver hypertrophy following embolization of the portal vein ipsilateral to the side of the disease, it thus leads to hypertrophy of the contralateral side, i.e. the FLR. Additionally, it results in an increase in the production of hepatic growth factor and tissue growth factor, along with redistributing the portal blood flow to the FRL. CT volumetry should be performed 3-4 weeks after PVE to assess the degree of hypertrophy. PVE allows for hypertrophy of 30-40% in 80% of patients within 4-6 weeks. Current guidelines recommend PVE for patients with underlying cirrhosis and an anticipated FLR of ≤40%, or patients with normal liver function and intended FLR of <20%.

In some circumstances a PVL may be preferable, especially when resecting a bilobar malignant liver lesion which requires a two-stage approach due to inadequate FLR volume. At the first operation the contralateral portal vein is ligated and in 3-6 weeks after adequate hypertrophy has taken place, the second stage is performed. At which time the extended/major hepatectomy is performed. A meta-analysis reported that there was no statistically significant difference comparing PVE and PVL in terms of increasing FLR volume, morbidity and perioperative mortality.

The ALPPS procedure was developed to decrease time between PVL and resection for patients with borderline FRL volume. ALPPS may facilitate superior hypertrophy of the FLR compared with PVE, with a reported 74% volume increase of the remnant liver in a mean of 9 days. This procedure has however been reported to have high operative morbidity (16-64% of patients) and perioperative mortality (12-23% of patients).

**Treatment of Post-hepatectomy Liver Failure**

When present PHLF is manifest by progressive multi-system organ failure, including renal insufficiency, encephalopathy, need for ventilator support, and need for pressor support. As hepatic function worsens, patients develop persistent hyperbilirubinemia and coagulopathy. Patients need to be closely monitored postoperatively to identify and treat PHLF as early as possible. Particular attention should be paid to early clinical and laboratory signs of liver failure including changes in coagulation factors and bilirubin, as well as signs of encephalopathy. Patients should also be monitored for early signs of infection, hemodynamic failure, renal failure, malnutrition, or metabolic disorders. Patients should be monitored in an ICU setting.

The management principles follow those of the American Association for the Study of Liver Diseases (AASLD) for the management of acute liver failure. The severity of the PHLF
needs to be followed using laboratory values. Resuscitative measure and organ support provide the optimal environment for liver regeneration.

It is necessary to detect and treat all triggers or aggravating factors of PHFL at a very early stage: establishment of an appropriate antimicrobial therapy (blood, ascites and bile samples), looking for vascular complication (arterial or venous thrombosis), radiological drainage if biliary fistula, optimization of vital functions (vascular filling in case of hypovolemia) and prevention of malnutrition by enteral or parenteral nutrition (needs increased by 30%, supplementation with vitamins and trace elements). The use of hepatotoxic and nephrotoxic drugs should be banned. Regarding the symptomatic treatment, the use of lactulose is usually recommended to correct encephalopathy, and administration of mannitol is possible to reduce intracranial hypertension. Compensation in platelets or clotting factors is needed in case of coagulopathy.

The development of artificial liver brought much hope in terms of treatment in fulminant hepatic failure. For the management of PHLF, few studies have been conducted, and results are disappointing since no survival improvement was found, but it may be helpful as a bridge to possible orthotopic liver transplantation (OLT).97,98

The most efficient treatment of PHLF remains OLT. This indication however is marginal due to graft shortage and the oncologic context in which most of these procedures are performed. PHLF accounts for less than 10% of liver transplantations. OLT for PHLF has only been sparsely reported.99,100 The initial indication for hepatic resection frequently involves a malignancy outside of transplantation criteria.

References:


Abdominal wall defects are divided into omphalocele and gastroschisis. While often considered together, they are distinct and separate entities.

**Embryology and Aetiology**

The current understanding of the aetiology for an omphalocele suggests that this defect is not from a failure in body wall closure or migration. It is thought that an omphalocele develops due to a failure of the viscera to return to the abdominal cavity. Other intra-abdominal viscera often found in the omphalocele sac are liver, stomach, ovary and testis. The sac consists of covering layers of the umbilical cord and includes amnion, Wharton's jelly, and peritoneum. The location of the defect is in the mid-abdomen, but may also occur in the epigastric or hypogastric regions as well.

The aetiology for gastroschisis is less clear. Currently, the ventral body fold theory, which suggests failure of migration of the lateral folds (more frequent on the right side), is most widely accepted. Due to increasing incidence of gastroschisis, there are a number of possible causative factors including tobacco, lower maternal age and low socioeconomic status. Gastroschisis occurs in 1 in 4000 live births. An increased incidence in mothers younger than 21 years has been widely documented. There is also a significant increase in incidence in all age groups over the past two decades. Preterm delivery is more frequent compared with babies without an abdominal wall defect.

**Pre- and perinatal management**

Gastroschisis: In the developed world, most gastroschisis children are diagnosed sonographically by 20 weeks gestation. Detection of freely floating bowel loops and a defect in the abdominal wall to the right of the umbilical cord are diagnostic. Intrauterine growth retardation is common in these foetuses. Some authors advocate selective preterm delivery based on the finding of bowel distention and thickening of the bowel wall on prenatal ultrasound. Studies in animal models have shown that the duration of amniotic fluid exposure is correlated with the degree of inflammatory peel seen on the bowels. Damage to the pacemaker cells and nerve plexi may be the reason for the profound dysmotility and malabsorption seen in these patients. Early delivery may mitigate these effects, but the literature is mixed. Currently available evidence does not support elective preterm delivery for gastroschisis.

**Neonatal Resuscitation**

Neonates with gastroschisis have significant evaporative water losses from the open abdominal cavity and exposed bowel. Appropriate intravenous access should be obtained and fluid resuscitation initiated after birth. Nasogastric decompression is important to prevent further gastric and intestinal distention. The neonate should be positioned on the right side to prevent kinking of the mesentery with resultant bowel ischemia. The bowel should be wrapped with plastic wrap or the infant placed partially in a plastic bag to reduce evaporative losses and improve temperature homeostasis. Although gastroschisis most often is an isolated anomaly, thorough examination of the neonate is important. In addition, the bowel must be carefully examined for intestinal atresia, necrosis, or perforation. Recent evidence suggests that excess fluid resuscitation is detrimental and results in oedema, an increase in time to closure, and an increased risk of abdominal compartment syndrome.
The primary goal is to return the viscera to the abdominal cavity while minimizing the risk of damage due to trauma or increased intra-abdominal pressure. The two most commonly used treatment options are placement of a silo followed by serial reductions and delayed closure, or primary closure. In all cases, inspection of the bowel for obstructing bands, perforation, or atresia must be undertaken. Primary closure is the preferred option if the bowels can be returned to the abdominal cavity without causing abdominal compartment syndrome. The urinary bladder pressure measured via the inserted urinary catheter should not be higher than 25 cm water after closure of the abdominal wall.

Preservation of the umbilicus is preferred because of its superior cosmetic result. In the mid-1990s, a prefabricated silo was developed with a circular spring that is positioned under the fascial opening, without the need for sutures or general anaesthesia. This has made it possible to insert the silo in the delivery room or at the bedside. After placement, the bowel is reduced daily into the abdominal cavity as the silo is shortened by sequential ligation. When the intestines are entirely reduced, the silo bag is removed and the remaining abdominal wall defect covered by a dressing and left to close by secondary intention. This process takes usually one to two weeks.

**Management of associated intestinal atresia**

Up to 10% of neonates with gastroschisis have an associated atresia, most commonly jejunal or ileal. These atresias can be treated at the time of abdominal wall closure with resection and primary anastomosis in cases where there is minimal inflammatory peel. If the condition of the bowel makes primary anastomosis inadvisable, the bowel is reduced with the atresia intact and repair is undertaken four to six weeks after the initial abdominal wall closure. If perforation is found, the perforated segment can be resected and a primary anastomosis performed if the inflammation is minimal. Alternatively, an ostomy can be created followed by ostomy closure at a later date. There is no consensus about the optimal management for these complicated problems. Patients with an atresia are considered to be ‘complex’ to differentiate them from the patients without any such association (simple).

**Postoperative course**

Most authors note worse outcomes with complex cases. Gastroschisis is associated with abnormal intestinal motility and nutrient absorption, both of which gradually improve. Introduction of enteral feeding is often delayed for weeks while awaiting return of bowel function. During this waiting period, nasogastric decompression and parenteral nutrition is required. When bowel activity occurs, enteral feeds can be started and slowly advanced. Because progression to full enteral feeding can take weeks, central venous access is important.

**Long term outcomes**

Long-term outcomes for patients born with gastroschisis are generally excellent. The presence of complex disease is the most important prognostic determinant for a poor outcome.

Most gastroschisis patients have some degree of intestinal nonrotation. This is typically not repaired at the time of closure and does not have the same incidence of midgut volvulus as other causes of malrotation. However, the parents should be cautioned regarding bilious emesis and instructed to take urgent action if that occurs.

Cryptorchidism is associated with gastroschisis in 15–30% of cases. Several retrospective analyses have shown that placement of the herniated testis into the abdominal cavity will
result in normal testicular descent into the scrotum in most cases. Most centres recommend allowing a year for spontaneous descent and then performing an orchiopexy if needed.

Omphalocele

The diagnosis of omphalocele can be made by ultrasound at the time of the normal 18-week ultrasound evaluation. It is also useful in the detection of other abnormalities which are frequent in these children: Cardiac and central nervous system abnormalities might have a serious impact on prognosis and raise questions about early termination of pregnancy.

The route of delivery is usually vaginal except in giant omphaloceles where C-section is preferred. After delivery, a thorough search for associated anomalies is important. All neonates should undergo an echocardiographic evaluation. Renal abnormalities can be detected by abdominal ultrasound. Neonatal hypoglycaemia should alert the practitioner to the possibility of Beckwith–Weidemann syndrome.

Infants with an omphalocele do not have as significant fluid and temperature losses as those with Gastroschisis. The sac itself can be covered with saline-soaked gauze and an impervious dressing to minimize these losses. An NG tube should be inserted and placed to suction.

Surgical management: Defects that are less than 1.5 cm in diameter are referred to as hernia of the cord and are repaired shortly after birth without any issues as long as there are no associated anomalies. The defects that are larger, but still easy to close without much loss of abdominal domain can also be closed soon after birth. Primary closure consists of excision of the sac and closure of the fascia and skin over the abdominal contents. If the sac is too large for primary closure we prefer nonoperative techniques. The sac is painted with an antiseptic, allowed to slough off and eventually granulation tissue forms on the intestines. From the edges of the wound skin epithelium grows in until the defect is covered by skin. At about age 1 year the abdominal cavity has enlarged enough to be able to receive the intestines from the ventral hernia which is repaired with or without mesh.
MALDESCENDED TESTIS IN ADULTS
Dr. Evelyn M Moshokoa, HOD Urology

What it is?
Cryptorchidism/Undescended testis is the absence of one or both testes in normal scrotal position.
Clinical evaluation may refer to palpable cryptorchid testes or to nonpalpable testes, which are either cryptorchid or absent. Hidden or obscure testis/testes that may either be present in an abnormal position or absent.

How common it is?

**Epidermiology**
Cryptorchidism is the most common genital disorder encountered in paediatrics.
- 1% to 4% of full-term and
- 1% to 45% of preterm male neonates (Sijstermans et al, 2008)
Spontaneous descent is more likely and may occur later in premature infants.
Spontaneous descent after the first year of life is uncommon.

Gonadal descend
*Trans abdominal descent*
1. Differential growth of vertebrae and pelvis.
2. Enlargement of gubernaculum pulls developing testes toward inguinal region
   * InsI3 cause gubernaculum enlargement
3. Regression of cranial suspensory ligament
   * Androgen cause regression of cranial suspensory ligament

**Between 10 – 15 weeks testes near future inguinal region**

Retractile and Acquired Undescended Testes
Theories:
- Presence of a fibrous remnant of the processus vaginalis that tethers or foreshortens the cord over time or mobility of the testis within an open sac.
- Testis is incompletely descended from birth. (Barthold and Gonzalez, 2003)
- Somatic growth results in relative widening of the distance between testis and scrotum. (Redman, 2005; Agarwal et al, 2006).

**Genetic Susceptibility**
- Genetic studies of cryptorchidism suggest that the disease is heritable but that susceptibility is likely polygenic and multifactorial.
- Autosomal dominance with reduced penetrance probable mode of inheritance
- Recurrence risk ratio (RR) was 10.1 in twins, 3.5 in brothers, and 2.3 in offspring and were significantly higher in maternal than in paternal half-brothers. (Schnack et al, 2008).
An adult is not a big child!

**Presenting History**
- Pain
- Hernia
- Testicular malignancy
- Infertility
- Micropenis
- Delayed puberty

**Hypogonadism**
- Loss of libido (desire)
- Fatigue / depression / loss of well-being
- ↓ Lean body muscle and mass
  - ↑ Visceral fat / mass
  - Sleep disturbances
  - ↓ Virility
  - ↑ Sweating / dry skin / anaemia
  - Osteoporosis

**Approach**
- History
- Exam
- Investigations
- Management

**An Adult with empty scrotum**

**Congenital**
- Undscended
- Retractile
- Ectopic
- Vanished
- DSD
- Agenesis

**Acquired**
- Torsion
- Orchidectomy

**Clinical Examination**
- Palpable
- Non – Palpable
- Unilateral
- Bilateral
- Associated penile abnormality
- No penile abnormality
Evaluation

- Role of Sonar
- Baseline investigations?
  - Testosterone
  - Semen analysis
- To do a biopsy or not

- Anatomy
- Physiology
- Psychosocial aspects
- Technical aspects

Anatomy

- Inguinal canal long and developed
- Cremasteric muscles thicker
- Pampiniform plexus more developed
- Testicular size

Physiology

- Testosterone production
- Spermatogenesis
- Thermoregulation dependence
  - Countercurrent mechanism
  - Cremasteric reflex

Cryptorchism is the most common disorder of sexual differentiation

Orchiolysis is a common paediatric procedure performed world wide

Laparoscopically introduced in 1976 by Cortes

Diagnostic

Therapeautic
LAPAROSCOPIC ORCHIOPEXY: PROCEDURE OF CHOICE FOR THE NONPALPABLE TESTIS?

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LAPAROSCOPIC FOWLER-STEPHENS ORCHIOPEXY FOR THE HIGH ABDOMINAL TESTIS

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Advantages of laparoscopic orchiolysis

Contraindications

- Previous extensive pelvic surgery

Laparoscopic findings

Unilateral

- Absent testis and cord structures
- Absent testis with cord structures exiting the inguinal canal
- High testis
- Testicular Nubbin
  - Orchidectomy

Bilateral

- Fowler Stephens
- Prentis Maneuvre
- Orchidectomy

Laparoscopy

- Undescended non palpable testis

Relative

- Redo cases. Can be done
- Concomitant hernia repair

Adults!

- Modifications have been discussed regarding orchiolysis
- Challenge on mobilization

- Adult with bilateral undescended testis!
  - Intra-abdominal testis
  - Fowler -Stevens
  - When to do orchidectomy?
  - When to do nothing?

Absent testis
• Treat the complications
• Hypogonadism
  – Testosterone supplement
• Family interest
• Self image
  – Testicular prosthesis
COMPLICATIONS AND SEQUELAE OF INGUINAL HERNIA REPAIR
Prof Zach Koto, Department of Surgery, Sefako Makgatho Health Sciences University

Introduction
Inguinal hernia repair one of the most commonly performed operations in surgery world-wide. The concept of watchful waiting which was initially thought of as a viable strategy has now been largely abandoned by most as data shows that on follow-up majority of these so-called asymptomatic patients do eventually need surgery. The Lichtenstein repair which epitomizes tension free repair has over the years become the benchmark against which all types of repairs are measured. The advent of laparoscopic surgery has now become a much preferred procedure that achieves the similar objectives of a tension free repair with comparable recurrence rates. The take up of laparoscopic repair has been low in many centres but the incidence of laparoscopic repair is on the rise.

Complications
Complications following hernia repair are very diverse and for purposes of this overview, I will only discuss those complications that are associated with laparoscopic repair. The anaesthetic and general complications will not be discussed here but only procedure related and long term complications.

Procedures related complications

Complications related to anatomy
Although knowledge of anatomy of the pelvis is crucial to understanding how vital associated structures are linked to the inguinal defect. This anatomy can be very challenging even to the most experienced of surgeons. In some instances even an obvious hernia can be missed by the uninitiated resulting in no repair at all.

Injury of the surrounding structures
The key landmarks should be observed at all times to avoid dissecting in the wrong plane which can result in injury to Iliac vein – this can be a catastrophic complication which can result in significant blood loss and even mortality. This complication must be divided at all costs. In the unlikely event that this happens, the surgeon must apply pressure immediately with a grasper – avoid suction – a swab introduced through a 10 mm port to apply pressure is helpful, an increase in the pneumoperitoneum pressure is also helpful and immediate conversion of the patient to open procedure must be done.

The key to avoiding this injury is identification of the landmarks and knowing at all times where you are dissecting in relation to the iliac vessels.

Bladder injury
Urinary bladder must always be emptied before an inguinal hernia is done laparoscopically – a urinary catheter may be helpful but it is associated with discomfort and possible urinary tract infection. We prefer to empty the bladder before we start the operation by asking the patient to go to the bathroom just immediately before surgery. The insertion of the suprapubic port must be done under vision and meticulous dissection is key. Knowing where you are is crucial during the dissection.

Cord structures
Identification of cord structures and the hernia sac is important. The cord structures must be handled with care and one must avoid direct handling of the cord structure to avoid injuring
the testicular vessels and lymphatics. The sac must be dissected meticulously off to avoid injury.
The vas and cord structures must be separately identified. The vas is more susceptible to
injury because it lies more medial and can be accidentally transected when dividing the sac.
It is thus very key to dissect as far medially as possible to identify the vas.

Testicular atrophy
Testicular atrophy and necrosis as a result of ischaemic orchitis is a well-recognised
complication following an inguinal hernia repair. Although it is reported rarely it does occur.
The reported incidence is about 0.5% for primary open inguinal surgery and 5% for recurrent
open inguinal hernia (1).

For laparoscopic inguinal hernia repair some investigators report 1% incidence of testicular
atrophy. This complication is thought to be more common in open repair. The possibility of
this complication must be discussed with the patient beforehand.

The nerves

There are four crucial nerves in the posterior abdominal wall that can be injured during a
total extra-peritoneal (TEP) hernia repair.

Ilio-inguinal nerve, illiohypogastric nerve that lie on quadratus lumborum muscle, lateral
cutaneous nerve of the thigh that runs between the quadratus lamorum muscle and psoas
muscle, and genito-femoral nerve that runs on psoas muscle.

These nerves when injured can result in sensory loss of the affected area or chronic groin
pain which can be quite incapacitating.

Injury to these nerves can be avoided by avoiding placement of tackers posteriorly unless
fixating the mesh or better still using fibrin glue as a fixation strategy.

Chronic inguinodynia
Post hernia repair, groin pain is expected for up to 2 months after the procedure. It becomes
chronic groin pain if it persists beyond 3 months after the operation. It certainly can be a
challenge to manage when it occurs. The management of this condition is a whole separate
discussion and will not be discussed here.

Seroma
Seroma following a laparoscopic hernia repair is fairly common. It can sometimes be
mistaken as a recurrence. Compression of the groin may help reduce this complication.

Haematoma
The incidence of haematomas following groin hernia surgery is fairly common. Meticulous
dissection and haemostasis is important to minimize this complication.

Mesh sepsis
Mesh sepsis is very uncommon. Mesh sepsis can be minimized by avoiding implantation of
the mesh in potentially contaminated area. In the unlikely event of mesh sepsis, exploration
of the mesh can be avoided by the use of light weight mesh with a large mesh size.

Summary
Complications of inguinal hernia repair can be minimized or completely avoided by attention
to details meticulous technique and adequate proctoring.
References

Problems of continence following surgery for haemorrhoids: surgical misadventure or unavoidable complication
Dr Stephen Grobler, Bloemfontein

General introduction
Pelvic floor and proctologic disorders are commonly encountered in surgical practice. Although regarded as routine pathology by many, choosing the right treatment at the right time can pose a real challenge to the treating surgeon and often involves choosing between two evils. Radical treatment leads to good symptom control but is likely to result in functional deterioration, whereas less invasive treatment is safe but often leads to inferior results. This has been the core problem in the treatment of pelvic floor and proctologic disorders for centuries. The close interaction between the anatomy and physiology of the rectum and anus is responsible for this compromise the surgeon often has to make. Historically functionality was of secondary importance to healing of the disorder, but nowadays it is considered unacceptable to cause permanent disability for a benign disorder. Many classic surgical therapies such as anal stretching and lateral internal sphincterotomy for anal fissure, haemorrhoidectomy and (extensive) lay-open procedures for anal fistula have been re-evaluated over the past decades with special respect to postoperative functionality. Reports of high rates of impaired continence, severe postoperative pain and high rates of recurrence have boosted the incentive to introduce new techniques in the field of proctology.

Anatomical considerations
The rectum constitutes the last 12-15 cm of the large bowel and starts at the level of the promontory. It is situated within the pelvic cavity. The anal canal is the last part of the rectum, starting at the anorectal junction where the rectum passes through the levator muscle, ending at the anal verge. The length of the functional anal canal is about 4 cm, whereas the anatomical anal canal, extending from anal verge to dentate line is only 2 cm in length.

The most important contributor to faecal continence is the anal sphincter, which consists of 2 muscular components: the internal anal sphincter (IAS), 0.3-cm to 0.5-cm thick continuation of the circular smooth muscle layer of the rectum and the external anal sphincter (EAS), 0.6-cm to 1.0-cm thick conjunction of the levator ani muscle complex. Morphologically, both sphincters are separate and heterogeneous. The IAS is a predominantly slow-twitch, fatigue-resistant smooth muscle. The IAS generates mechanical activity, with a frequency of 15 to 35 cycles per minute and ultra-slow waves at 1.5 to 3 cycles per minute. The IAS contributes approximately 70% to 85% of the resting sphincter pressure, but only 40% after sudden distension of the rectum and 65% during constant rectal distension. Thus, the IAS is chiefly responsible for maintaining anal continence at rest. The anus is normally closed by the tonic activity of the IAS. This barrier is reinforced during voluntary squeeze by the EAS. The puborectalis muscle forms a flap-like valve that creates a forward pull and reinforces the anorectal angle.

Anal Endovascular Cushions
The anal canal is lined with squamous epithelium below the dentate line, and columnar epithelium above. The dentate line is also marked by the lower end of the columns of Morgagni, where the ducts of the anal glands terminate. A substantial number of these glands branch out into the intersphincteric plane. Between the upper border of the anal canal and the dentate line lie three vascularised cushions. The blood-filled vascular tissue and submucosa of the anal mucosa play an important role in producing a more perfect closure of the anus. The internal sphincter ring is unable to close the anal orifice completely – this is filled by the anal cushions, which may contribute 10% to 20% of resting anal pressure by
physically acting as a sealing ‘plug’.

**Pathophysiology of evacuatory disorders**

The cause of faecal incontinence is usually multifactorial. Faecal incontinence occurs when the normal anatomy or physiology that maintains the structure and function of the anorectal unit is disrupted.

Disruption or weakness of the EAS can cause urge-related or diarrhoea-associated faecal incontinence. Obstetric trauma, the most common cause of anal sphincter disruption, may involve the EAS, IAS and pudendal nerves, singly or in combination. It remains unclear why most women who sustain obstetric injury in their 20s or 30s typically do not present with faecal incontinence until their 50s.

In contrast, damage to the IAS muscle or the anal endovascular cushions may lead to a poor seal and an impaired sampling reflex. These changes may cause passive incontinence or faecal seepage, often under resting conditions.

Both sphincters may be defective in many patients. The extent of muscle loss can influence the severity of incontinence.

Damage to the endovascular cushions may produce a poor anal “seal” and an impaired anorectal sampling reflex. The ability of the rectum to perceive the presence of stool leads to the recto-anal contractile reflex response, an essential mechanism for maintaining continence.

Anal leakage caused by mild passive faecal incontinence or soiling is not exclusively caused by diminished sphincter function. On the contrary, dysfunction of evacuation of stools may be the main cause. Many patients with obstructed or dyssynergic defecation have passive faecal incontinence, sometimes accompanied by faecal soiling, due to the incomplete evacuation of stools. They generally loose stools or liquids passively during walking without noticing. Incomplete evacuation of stools in obstructed defecation can be caused by rectocoele or internal and external rectal prolapse.

Excessive pelvic floor descent on straining may lead to progressive damage to the striated anal sphincter muscle and or pudendal nerves due to repeated stretch injury. Anal endosonography has shown that aging was associated with an increase in the thickness and echogenicity of the internal sphincter muscle.

The sacral nerves are intimately involved with the maintenance of continence. Bowel contents are periodically sensed by anorectal sampling, the process by which transient relaxation of the IAS allows the stool contents from the rectum to come into contact with specialized sensory organs. The likely role of anal sensation is to facilitate discrimination between flatus and faeces and the fine-tuning of the continence barrier. Rectal sensitivity may be too low to detect the presence of residual faeces.

Pudendal neuropathy can diminish rectal sensation and lead to excessive accumulation of stool, causing faecal impaction, mega-rectum and faecal overflow. The puborectalis muscle plays an integral role in maintaining the anorectal angle. Its nerve supply is independent of the sphincter.

Diarrhoea and constipation with overflow incontinence must be managed to achieving controlled evacuation of stools.

The consistency, volume and frequency of stool and the presence or absence of irritants in stool may also play a role in the pathogenesis of incontinence. In the presence of large volume liquid stools, which often transit the hindgut rapidly, continence can only be maintained through intact sensation and a strong sphincteric barrier. Similarly, in patients
with bile salt malabsorption, lactose or fructose intolerance or rapid dumping of osmotic material into the colon, the colonic transit is too rapid for both gaseous and stool contents and can overwhelm the continence mechanisms.

A variety of medical conditions and disabilities may predispose to faecal incontinence, particularly in the elderly, e.g. immobility and lack of access to toileting facilities. Several medications may inhibit sphincter tone, e.g. anticholinergics, some of which are used to treat urinary incontinence and detrusor muscle instability, muscle relaxants such as baclofen and antispasmodics. In contrast, stimulants such as caffeinated products, fibre supplements, or laxatives may cause diarrhoea.

**Haemorrhoidal disease**

Haemorrhoidal disease is a frequently occurring entity and constitutes the most common proctologic disorder (prevalence 4 – 10%).

Haemorrhoids are highly vascular tissue in the submucosal space in the anal canal. Haemorrhoidal tissue gives rise to symptoms such as bleeding, prolapse or pruritus when it slides downward. Aetiological factors are multifactorial and include prolonged straining, irregular bowel habits and heredity.

When conservative treatment like dietary- and defaecation habit advice fails, the primary treatment of choice should be rubber band ligation (RBL). This procedure is reported to be successful in 65-85% of the patients. The setback is that treatment often needs to be repeated in order to be successful and that grade 3 to 4 haemorrhoids are less amenable to banding. This leaves a substantial group of patients which are not treated sufficiently.

Doppler-Guided Haemorrhoidal Artery Ligation (DGHAL) or Transanal Haemorrhoidal Dearterialisation (THD) uses custom proctoscopes combined with a Doppler transducer to identify the haemorrhoidal arteries (originating from the superior rectal artery) for selective ligation and fixation. The theory that haemorrhoids occur when there is an imbalance in the blood flow of the haemorrhoidal plexus, either caused by increased inflow or decreased venous outflow underlies the inflow reduction by arterial ligation, causing the vascular plexus to recede.

Overactivity of the anal sphincter in patients with haemorrhoids may be primary or secondary and thus raise the notion of the need for anal dilatation or lateral internal sphincterotomy in addition to the surgical treatment of haemorrhoids. Tonic contraction of the sphincter muscle in patients with advanced stages of haemorrhoids is considered by many authors as a primary cause.

The nefarious practice of Lord’s stretch is banned to the annals of iniquity!

Some operators tend to complete haemorrhoid surgery with lateral internal sphincterotomy (LIS). Sequelae of lateral internal sphincterotomy cannot be excluded and may later negatively affect patient’s anal continence. Overactivity of the anal sphincter in patients with haemorrhoids seems rather to be a secondary phenomenon and therefore there is no indication for lateral internal sphincterotomy (LIS). The pathogenic circle is broken by therapy of the haemorrhoids.

Persisting hypertension of the anal sphincter after Milligan-Morgan haemorrhoidectomy may be associated with delayed healing of extensive defects of anorectal mucosa. Postoperative pain leads to increased tension of the anal sphincter and subsequently prolongation of operative wound healing. However, stapled haemorrhoidectomy significantly reduces resting anal pressures in the immediate postoperative period, allowing more rapid improvement in anorectal function compared to Milligan-Morgan haemorrhoidectomy.

Anal dilation or lateral sphincterotomy may result in permanent incontinence due to
fragmentation of the anal sphincter apparatus. Only mild dilatation of the anal canal is ever allowed. Dilatation should never exceed 40mm (~2 fingers or 4 clicks of standard Park’s anal dilator). But even mild dilatation of a stenotic anal canal or pelvic floor weakness may unmask underlying anal sphincter defects and render your patient incontinent, if not immediately, then when they become older!

If in doubt, do NOT dilate but rather refer for objective colorectal assessment (ultrasound, physiological testing) and colorectal specialist management.

Classic haemorrhoidectomy (“open” Milligan-Morgan, or the preferred “closed” Ferguson) is still an effective procedure for resolving haemorrhoidal symptoms but further affects the already altered anal anatomy and may result in disordered defaecation.

There is no doubt that haemorrhoidectomy may endanger the sphincters. This is usually due to inadvertent stripping of the internal sphincter together with the haemorrhoidal complex. Technical errors (ignorance, unfamiliarity and over exuberance) may even damage the external sphincter. Pathology reports of 16% of specimens containing muscle fibres (80% smooth and 20% striated muscle) are cause for concern! It seems that surgical anatomy needs to be included as an important syllabus while educating surgeons for haemorrhoid surgery.

Anal endosonography may reveal sphincter defects after haemorrhoidectomy – the pattern is usually specific, with patchy disruption of the IAS, but some have EAS defects too. Nevertheless, 70% of patients may be asymptomatic, without complaints. (Pre)-existence of small sphincter defects without symptoms should be realized when evaluating patients to decide on causation.

There is no excuse for “blind” surgery – get help, learn, train and teach. Ample safe and effective anorectal retractors are available and should be used.

Blind mass resection of haemorrhoids by clamping energy devices is also not safe. These energy devices should rather be used to provide haemostasis and cutting of carefully dissected mucosa and submucosa only.

Therefore, other techniques have been developed to treat patients that have not reacted to conservative treatment, rubber band ligation or HAL. The Procedure for Prolapse and Haemorrhoids (PPH) or stapled haemorrhoidopexy (SH) entails stapled transanal mucosectomy by circumferential resection of mucosa and submucosa above the haemorrhoids. PPH/SH is reported to be an effective and safe alternative for surgical haemorrhoidectomy with less postoperative pain, shorter hospital stay and greater patient satisfaction.

Devastating complications from misplacement of the purse-string in relation to the dentate line (too high) and the depth of biting (too deep bites) and the drawing in of too much tissue into the stapler housing have been reported. The basic notion is that the submucosal purse-string suture should be placed at a distance of 4 - 5 cm above the dentate line; the stapling line should lie 2 to 3 cm proximate to the dentate line. Key technical factors must be respected:

- ensure correct positioning of the dilator to facilitate visibility of the whole dentate
- tighten the purse-string firmly on the anvil and address the stapler so as to parallel the rectal axis (if firm tightness is not feasible, the procedure is converted to an open haemorrhoidectomy)
- precise synchronicity of a progressive introduction of the stapler and the closing of its jaws can sufficiently control the amount of rectal mucosa and submucosa to be resected
- check the vaginal wall before engaging the stapler to correct the actual direction of the instrument and safeguard against a possible rectovaginal fistula
- the staple line should be checked and defects should be repaired
- perform double stapled or custom stapled resections for selected grade IV haemorrhoids, anal- or rectal prolapse
- excision of haemorrhoidal components that have failed to shrivel after PPH/SH so as to ensure a more radical result and to relax anastomotic tightness
- routinely excise concomitant skin tags or redundant anoderm.

Soiling (17-28%), prolapse (3-9%), and incontinence and urgency (1.8-5.6%) have been reported after PPH/SH, but usually improve with time. Persisting incontinence is rare. Complications after using different devices are comparable. The anal dilator for introducing/using the stapling device (diameter 37 mm) could be responsible for the observations. Supposed mucosal resection may excise upper portions of the sphincters if placed too low. Endoanal ultrasound lesions of the internal anal sphincter may be detected.
Anastomotic failure is an inevitable consequence of performing an anastomosis. There are only 2 causes of anastomotic failure, inadequate surgical technique or incorrect surgical judgement. The surgeon is directly responsible for all anastomotic failures.

The consequences of anastomotic failure may vary from a patient who has a mildly prolonged postoperative recovery to overwhelming sepsis with multiple reoperations and a high mortality. In addition, anastomotic leakage in rectal cancer surgery is associated with worse long term bowel function and increased local recurrence rates with reduced survival (Mirnezami, Mirnezami et al. 2011)

What is an anastomotic leak? There are at least 29 different definitions in the literature. A reasonable definition comes from the International Study Group of Rectal Cancer (Rahbari, Weitz et al. 2010). They defined an anastomotic leak as a defect in the intestinal wall at the anastomotic site leading to a communication between the intra and extra-luminal compartments. They graded leaks as:

Grade A: Leaks that do not change patient management

Grade B: Leaks that require intervention but not re-laparotomy

Grade C: Require repeat laparotomy

Incidence: Reported leak rates for colorectal surgery vary widely from 1%(Paun, Cassie et al. 2010) to 50%(Goligher, Graham et al. 1970) depending on how they are detected and the site of the anastomosis. The closer the anastomosis is to the anus in rectal surgery, the higher the leak rate. A clinical leak rate of around 10% for coloanal anastomosis is generally accepted, but for more proximal anastomoses, leak rates should not be any higher than 1 to 2%.

Defunctioning proximal to an anastomosis with a colostomy or ileostomy does not appear to alter leak rates, but it does decrease the morbidity of a leak (Huser, Michalski et al. 2008, Montedori, Cirocchi et al. 2010). It however adds to morbidity and mortality by adding a second operation for closure.

Factors affecting leak rates
- Technical factors:
  a. Leak rates vary between surgeons.
  b. Meticulous technique
  c. No tension
  d. Good blood supply
  e. No contamination
  f. Single layer better than double layer
  g. Lower leak rate for stapled anastomosis
  h. Appropriate suture material
i. High IMA ligation

- Management factors:
  a. Peri-operative hypotension
  b. NSAIDs
  c. Over use of perioperative fluids
  d. Inotropes
  e. Long operation time
  f. Blood loss
  g. Transfusions

- Patient factors
  a. Emergency surgery
  b. Significant pre-operative weight loss
  c. Low albumin
  d. Obesity
  e. Diabetes
  f. Liver disease
  g. Renal failure
  h. Myocardial infarction
  i. Cardiac failure
  j. Peripheral vascular disease
  k. Cerebrovascular disease
  l. Dementia
  m. Chronic pulmonary disease
  n. Any tumour
  o. Immunosuppression
     i. Steroids
     ii. Other causes
  p. Crohn’s disease
  q. Peritonitis

The important questions a surgeon needs to ask when performing an anastomosis are:

1. How important for the patient is this anastomosis?
2. What is the likely chance of a leak in this patient?
3. Does the patient have the reserve to survive a leak should it occur?

On re-operating or a patient who has had a leak, a surgeon has to establish the cause of the leak. If it is due to a technical error and there is little contamination, it may be reasonable to redo the anastomosis. In most other situations, the surgeon has to change the approach and take the anastomosis down.

References:


COMPLICATIONS OF PERIANAL ABSCESSES AND THOSE OF THEIR MANAGEMENT
Prof OD Montwedi

- Perianal abscess remains a common surgical problem
- Incident high in males

ETIOLOGY
A: TRAUMA   penetrating
   Local trauma (Abrasions)

B: Local Infection
- Hair follicle
- Haemorrhoid
- Fissure
- Post OP (Sphincterotomy)
- Other diseases (Chron’s disease)

C: CRUPTOGLANDULAR THEORY

1. Perianal, 2 Ischiorectal, 4 Submucus, 5 Marginal, 6 Supralevator, 7 Intersphincteric
- Management
Incision and Drainage

**PRINCIPLES**
- Criss cross incision over an area of maximal fluctuation
- Break all loculations
- Packing/ no packing
- Antibiotics only for certain situations
- Post operative sitz bath
- Dressings/ no dressings

**COMPLICATIONS**
- This can occur before or after surgery
- **PREOPERATIVE COMPLICATIONS**
  1. Rupture with subsequent recurrences or fistula formation
  2. Necrotising fasciitis
- common in immune compromised patients
- Upward spread of infection along skin, subcutaneous tissue, fascia and muscles
- Occurs in about---- of patients
- Diagnosis requires a high index of suspicion
- Rapid spread, tissues necrosis, excessive pain, no classical signs of inflammation

**PRINCIPLES OF MANAGEMENT**
- Resuscitate the patient
- Antibiotics: Broad spectrum
- Surgery: Aggressive debridement with faecal and urinary diversion
- Hyperbaric oxygen: shown to help but not a substitute for surgery
- Prognosis: Depends on severity. Can be as high as 70% in diabetic patients.

**POST OPERATION COMPLICATIONS**

**IMMEDIATE**
- A. Bleeding: Rare, but if it does occur simple packing is adequate
- B. Inadequate drainage
- C. Missed abscesses: In case of horse shoe abscess or submucous abscess
- D. Recurrent abscesses/ fistula This remains a common complication.
  
  Occurs in crypto glandular abscesses

**PREVENTATIVE STRATEGIES FOR RECURRENCES / FISTULAS**
- Antibiotics with incision and drainage (No evidence to support this)
- Combination of medical therapy for chron’s diseases and prolonged seton use
- Fistulotomy at same time with incision and drainage
• Plus swab during incision and drainage and fistulotomy in same hospital admission if GIT bacteria are cultured

MANAGEMENT OF FISTULA

1.) DEFINE ANATOMY? CLASSIFICATION (Parkes Classification)

A.) Superficial
B.) Intersphincteric
C.) Transsphincteric
D.) Suprasphincteric
E.) Extrasphincteric

2.) Fistulotomy/ Fistulectomy

3.) Preservation of sphincter function

4.) Post operative wound care

METHODS OF ASSESSING FISTULA

• Palpation
• Probes
• Injection: H2O2, methylene blue

IMAGING

• Endo anal u/s
• CT
• MRI
• Fistulography

Surgical option

• Fistulectomy: simple, intersphincteric
• Fistula plug (collagen plus) Less success rate
• Fibrin glue: recurrence high, Procedure can be repeat
• Seton: Transsphincteric, suprasphincteric
• Flap advancement
• Colostomy: chron’s disease
• Ligation of intersphincteric fistula tract (LIFT)

E. INCONTINANCE

Incontinence following treatment of perianal abscesses is rare

Increase incidence:

- Repeated drainage for recurrences
- Fistulectomy done same time as drainage
- Patients with necrotising fasciitis

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Appendicitis was first described in the 16th century as perityphlitis (inflammation of the connective tissue around the caecum)\(^1\). In 1886, Reginald H. Fitz described the pathogenesis starting with acute inflammation of the appendix to peritonitis and iliac-fossa abscess. Fitz also recommended surgical remedy for most cases\(^2\). In 1889, McBurney described the clinical findings. Appendicectomy is still the mainstay of treatment\(^1\).

### Pathology and pathogenesis

- **Appendix lumen obstruction** leads to congestion within the appendix.
- **Secretion of inflammatory exudate and mucous** causes increase in luminal pressure.
- **Obstruction of lymphatic drainage** leads to further oedema, ulceration, bacterial growth and formation of pus leading to ischaemia of the appendix.
- **The loss of integrity** leads to bacterial invasion of the submucosa and muscularis propria resulting in acute appendicitis.
- **On the rare occasion when the initial stages of infection resolves**, the appendix distends with mucus resulting in a mucocele\(^1\).

### Appendicitis complications

- **Gangrenous Appendicitis**: Thrombosis of the appendiceal artery and veins leads to ischaemic necrosis\(^3\).
- **Perforation**: The morbidity and mortality increase significantly following rupture of the appendix, with complication rates as high as 58 %\(^5\). A review of 653 cases of pathologically diagnosed appendicitis showed a perforation rate which is increased at both ends of the age spectrum. This is most likely due to delays in presentation and diagnosis\(^4\).
- **Peri-appendiceal abscess**: The most frequent complication of perforation. The abscess remains localized if peri-appendiceal fibrinous adhesions develop before rupture\(^3\).
- **Peritonitis**: Bacterial peritonitis is due to perforation before formation of inflammatory adhesions\(^3\). A study of 2522 cases intraoperative cultures of peritoneal fluid showed no growth in 64% of normal cases, 65% with acute appendicitis and 24% with perforated appendicitis. Cases with positive cultures isolated most commonly *Escherichia coli* followed by *Enterococcus* and other *Streptococcus* species. Less commonly encountered organisms cultured were *Pseudomonas*, *Klebsiella*, and *Bacteroides* species\(^4\). Refer to figure 1.
- **Bowel Obstruction**: Mechanical obstruction, likely secondary to entrapment of the distal ileum in a peri-appendiceal inflammatory mass\(^3\).
- **Septic seeding of mesenteric vessels**: Ascending infection along the draining mesenteric–portal venous system results in pylephlebitis, pylethrombosis, or hepatic abscess\(^3\).
- **Appendicitis and Pregnancy**: About one in five patients present with complicated appendicitis and a perforation rate of 15-20% which is reportedly highest in the first and second trimesters. Appendicitis in pregnancy is associated with up to a 5% risk of fetal loss which increases to 20% if perforation occurs\(^1\).
Management of Appendicitis

A meta-analysis of post-intervention complications in suspected uncomplicated appendicitis was lower in patients who were managed with appendicectomy (0.8%) than in patients managed with antibiotic therapy (10%)\(^6\).

A meta-analysis of comparison of the management of complicated appendicitis showed that conservative management with or without Interval appendicectomy, is associated with a decreased complication and re-operation rate compared with appendicectomy. Conservative treatment is associated with less wound infections, abdominal/pelvic abscesses, and ileus/bowel obstructions\(^7\).

Timing of Appendicectomy

A prospective study of 723 patients (83% had Open appendectomy and 17% had Laparoscopic appendectomy) showed that delaying appendectomies by 12–24 h had no impact on the perforation rate, operative time, complication rate or length of hospital stay. Delaying appendectomy more than 24 h after presentation had a significantly increased complications rate (18.5%). The number of gangrenous appendices was significantly higher and the histology showed a worse grade of inflammation\(^5\). Refer to Figure 2.

Figure 1: Distribution of organisms isolated from peritoneal cultures stratified by diagnosis\(^4\).

Figure 2: Comparison of the complication rate in the early appendectomy (EA)(< 12hrs from admission), early-delayed appendectomy (EDA)(12-24hrs from admission) and delayed
appendectomy (DA)(>24hrs after admission) groups.

**Interval Appendicectomy**

Inflammation associated with abscess formation or phlegmon may complicate an immediate appendectomy, leading to injury of adjacent loops of small bowel. Some authors prefer an immediate appendectomy and insist on a low morbidity rate, the reduction of the hospitalization stay, and also on the early diagnosis and treatment of unexpected pathologies. Due to the associated morbidity some surgeons will treat this situation conservatively in the face of abscess or phlegmon and return 6-12 weeks later to perform an interval appendectomy. Interval appendicectomy is controversial as the risk of recurrent appendicitis is low compared to the risk of complications from interval appendectomy.

Patients that undergo an interval appendicectomy, 16% will have a normal or obliterated appendix on pathologic evaluation and will likely not benefit from interval appendectomy. 84% of patients will have persistent acute appendicitis, chronic appendicitis, evidence of inflammatory bowel disease, or neoplasm identified, and likely benefit from surgical appendectomy. However, patients with appendicoliths are most likely to benefit from interval appendectomy as this becomes an environment for future infection.

**Laparoscopic versus Open Appendicectomy**

A decreased incidence of wound infections has been considered to be a major advantage of laparoscopic appendectomy. Randomization resulted in 82 patients with open and 87 patients with laparoscopic appendectomy. 6% had wound infections after Laparoscopic appendicectomy—all of them at the umbilical incision—and 7% after Open appendicectomy. Refer to Table 1.

**Table 1:**

<table>
<thead>
<tr>
<th>Postoperative Course and Complications</th>
<th>LA (n=82)</th>
<th>OA (n=87)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of operation, median (range)</td>
<td>35 (10-140) min</td>
<td>31 (15-107) min</td>
<td>NS</td>
</tr>
<tr>
<td>Number of complications, total (%)</td>
<td>16 (16%)</td>
<td>11 (11%)</td>
<td>NS</td>
</tr>
<tr>
<td>Wound infections (%)</td>
<td>5 (6%)</td>
<td>6 (7%)</td>
<td>NS</td>
</tr>
<tr>
<td>Intra-abdominal abscess (%)</td>
<td>2 (2%)</td>
<td>2 (2%)</td>
<td>NS</td>
</tr>
<tr>
<td>Intra-abdominal hemorrhage (%)</td>
<td>3 (3%)</td>
<td>0 (0%)</td>
<td>NS</td>
</tr>
<tr>
<td>Transient paralytic ileus (%)</td>
<td>3 (3%)</td>
<td>0 (0%)</td>
<td>NS</td>
</tr>
<tr>
<td>Hematoma, Seroma (%)</td>
<td>1 (1%)</td>
<td>1 (1%)</td>
<td>NS</td>
</tr>
<tr>
<td>Postoperative analgesic treatment</td>
<td>46 (56%)</td>
<td>56 (66%)</td>
<td>NS</td>
</tr>
<tr>
<td>Number of patients (%)</td>
<td>2 (1-5) days</td>
<td>2 (1-6) days</td>
<td>NS</td>
</tr>
<tr>
<td>Postoperative antibiotic treatment</td>
<td>14 (17%)</td>
<td>14 (17%)</td>
<td>NS</td>
</tr>
<tr>
<td>Number of patients (%)</td>
<td>4 (1-10) days</td>
<td>3 (1-10) days</td>
<td>NS</td>
</tr>
<tr>
<td>Length of hospital stay, median (range)</td>
<td>3 (2-23) days</td>
<td>4 (1-24) days</td>
<td>0.026</td>
</tr>
<tr>
<td>Duration of surgery related symptoms, median (range)</td>
<td>7 (0-40) days</td>
<td>11 (1-35) days</td>
<td>0.008</td>
</tr>
<tr>
<td>Return to sport activities, median (range)</td>
<td>13 (5-40) days</td>
<td>20 (11-35) days</td>
<td>0.002</td>
</tr>
<tr>
<td>Return to work, median (range)</td>
<td>14 (5-45) days</td>
<td>15 (6-37) days</td>
<td>NS</td>
</tr>
</tbody>
</table>

LA = laparoscopic appendectomy; OA = open appendectomy; NS = not significant.
However, a meta-analysis of all formally randomized prospective trials of Laparoscopic versus Open appendicectomy in adults showed the wound infection rate in the Laparoscopic appendicectomy is less than one half the rate in the Open appendicectomy group. The incidence of intra-abdominal abscess is higher in the Laparoscopic appendicectomy group, but this failed to reach statistical significance.\(^9\)

The management of complicated appendicitis, i.e. gangrenous, perforated appendicitis and appendiceal abscess, remains controversial with regard to Laparoscopic or Open appendicectomy. There is also some concern related to higher technical demand, longer operative time, and reported higher incidence of intra-abdominal collections in Laparoscopic appendicectomy. A retrospective study was conducted between 1999-2004 to evaluate the feasibility, safety, and efficacy of Laparoscopic appendicectomy when compared with open appendectomy in patients with complicated appendicitis. The incidence of intra-abdominal collection rates for patients who had Laparoscopic appendicectomy or Open appendicectomy, was 5.7% and 4.3%, respectively \(p = 0.473\). The conclusion was that Laparoscopic appendicectomy for complicated appendicitis is feasible and safe and has less risk of wound infection \(p<0.001\).\(^{10}\) Refer to Table 2.

### Table 2: Laparoscopic (LA) Versus Open Appendectomy (OA) for complicated appendicectomy\(^{10}\)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>LA group (n = 175)</th>
<th>OA group (n = 69)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (M/F)</td>
<td>75/100</td>
<td>35/34</td>
<td>0.27</td>
</tr>
<tr>
<td>Age (y), mean ± SD</td>
<td>46.3 ± 18.4</td>
<td>42.7 ± 19.1</td>
<td>0.17</td>
</tr>
<tr>
<td>Operative time (min), median (interquartile range)</td>
<td>55.0 (45–65)</td>
<td>70 (60–80)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Length of hospital stay (d), median (interquartile range)</td>
<td>5 (4–7)</td>
<td>6 (5–9)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Cases of wound infection, n (%)</td>
<td>1 (0.6)</td>
<td>7 (10)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Cases of intra-abdominal collection, n (%)</td>
<td>10 (5.7)</td>
<td>3 (4.3)</td>
<td>0.47</td>
</tr>
</tbody>
</table>

\(F\) female; \(LA\), total laparoscopic appendectomy for complicated appendicitis, including 24 conversion cases; \(M\), male; \(OA\), open appendectomy for complicated appendicitis.

*Statistically significant.

### Drain vs. No drain

Post laparoscopic appendicectomy intra-abdominal abscess formation may be more frequent because of spillage within the abdominal cavity of infectious contents, promoted by the carbon dioxide pneumoperitoneum. Allenmann et al. looked prospectively at 320 consecutive patients that underwent laparoscopic appendectomy for complicated acute appendicitis and found a statistically significant lower overall complication rate in the no-drain group, compared to the drained group \(7.7\% \text{ vs. } 18.5\%, p=0.01\), mainly due to low-grade complications. Abdominal wall abscesses were significantly more frequent in the drain group. Therefore the advantage of the laparoscopic over the open approach, i.e., the reduced wound infection rate, is eliminated by routinely inserted drains.\(^{11}\) Refer to Figure 3.
NOTES (natural orifice transluminal surgery)

NOTES procedures, especially transvaginal access, still remain controversial from patients’ and physicians’ point of view.

A prospective study was done on 13 patients that underwent transvaginal-hybrid NOTES appendectomy. One patient developed an intra-abdominal abscess (with a vaginal fungal infection) and another infected hematoma, both occurred after a perforated appendicitis. There is no impairment of sexual function for up to 2 years or child-delivery following this operative access. Intraoperative and early postoperative data seem comparable to that of Laparoscopic appendicectomy. NOTES also prevents abdominal wall trauma like trocar hernias. NOTES appendectomy is therefore an alternative to Laparoscopic appendicectomy.

Summary of Post-operative complications

Complications may be classified as early and late based on their time of presentation postoperatively.

Early complications (within the first week postoperatively), the most common early complication following appendicectomy for perforated appendicitis is wound infection. Patients undergoing primary appendicectomy with antibiotic prophylaxis have a surgical wound infection rate of 2.8% compared with 11.8% without the use of antibiotic prophylaxis. The most common causative organisms are Gram negative bacilli and anaerobes. Wound drainage may be indicated based on clinical findings and antibiotics given pre- and intra-operatively can reduce the rate of wound infection.

Intra-abdominal abscesses are twice as likely to occur in gangrenous or perforated appendicitis and is responsible for the majority of reported deaths after Laparoscopic appendicectomy. The most common sites for this include between loops of small bowel (interloop), pelvic, subphrenic and paracolic sites. Postoperative ileus should
be managed conservatively by keeping the patient nil by mouth, IV fluids and the insertion of a nasogastric tube. Port site haematoma for laparoscopic appendicectomy can be avoided by removing ports under vision¹.

**Postoperative leak** following appendectomy<48 hours the patient should be taken back to the exploration. Leaks that are suspected > 48 hours after a laparoscopic appendectomy, should be assessed in the context of the patient’s clinical status. If the patient is stable and does not show signs of diffuse peritonitis, a CT scan is recommended to assess the presence of intra-abdominal abscesses.

Late complications (more than 1 week post appendicectomy) include adhesional obstruction, faecal fistula, and incisional hernias. They are rare and may occur up to a few months to years after the surgery and more frequently occur in females, diabetics and those with perforation or peritonitis at the time of surgery. Incisional hernias following the open technique occurs in less than 0.12% of patients¹. **Urinary tract disorders** (retention and infection) accounted for 25% of all complications and is slightly more common in patients with perforated appendicitis. More than half of postoperative pneumonias are associated with the presence of perforation⁴.

**Miscellaneous complications** encountered include pseudomembranous enterocolitis, pulmonary embolus, acute renal failure, myocardial infarction, deep venous thrombosis, and common iliac artery laceration secondary to laparoscopic trocar insertion⁴.

**References**


COMPLICATIONS OF TOTAL THYROIDECTOMY
Prof LM Nthihe, Department of Surgery, University of Pretoria and Steve Biko Academic Hospital

INTRODUCTION

Palpable thyroid nodules occur in 3-7% of the population and ultrasound shows that the actual prevalence is up to 50%.

The number of thyroidectomies in the USA is 118,000 – 166,000 per annum, having trebled in the last three decades.

The US Agency for Healthcare Research and Quality Statistics (AHRQ) conclude that the incidence of thyroidectomies in the US to be 37.4/100,000 population.

Indications for total thyroidectomy (TT) are Cancer, Pressure symptoms, Cosmesis and fear of malignancy

COMPLICATIONS ASSOCIATED WITH TT:

1) Recurrent (RLN) and Superior Laryngeal Nerve injury (SLN)
2) Hypocalcaemia due to Parathyroid Gland (PG) injury
3) Haemorrhage
4) Endotracheal Intubation and Extubation Risks
5) Other rare complications:
   - Thyroid Storm
   - Tracheomalacia
   - Pneumothorax
   - Thoracic duct / Right Lymphatic trunks injury and chyle leak
   - Brachial plexus injury
   - Oesophageal ,Laryngeal and tracheal injury
   - Cervical Sympathetic damage…Horner’s Syndrome
   - Phrenic nerve injury
7) Recurrence

SURGICAL Techniques:

   a) Standard Open method
   b) Minimally Invasive Video-Assisted Thyroidectomy (MIVAT)

PRE-OPERATIVE ASSESSMENT

- A thorough History and Physical examination to evaluate and manage any comorbidities
- Thyroid assessment including its functional status, patients should be rendered euthyroid
-ENT assessment including Voice evaluation, may need baseline audio-recording when indicted and laryngoscopy.

- FNA Biopsy

-Imaging:
  
  CXRay, Inlet Xray; CT or MRI when indicated.

INTRA- OPERATIVE MANAGEMENT

Anaesthetic:

Collaboration with the anaesthetist is mandatory

Need for advanced intubation techniques

Surgeon may have to perform an emergency tracheostomy

Valsalva maneuver at TT completion to assess bleeding

Extubation in OT or in ICU or High care.

Surgical technique:

The surgical anatomy is of paramount importance.

Relationships of the to be removed gland to the recurrent laryngeal nerves (RLNs), superior laryngeal nerves (SLN) (Internal and External branches: - IB-SLN and EB-SLN), parathyroid glands (PGs) superior and inferior pairs, carotid sheath,laryngo-tracheal axis, oesophagus and other structures.

1) RECURRENT LARYNGEAL NERVE:

On the right side it recurs around the 4th aortic arch (Subclavian artery). It is 5-6cm long reaching the Cricothyroid joint.

On the left side it is 12cm long reaching the cricothyroid joint.

Both nerves ascend to the tracheo-oesophageal groove. At the ligament of Berry(posterior position) the nerve divides into smaller sensory branches and the main motor branch which enters the larynx below the inferior constrictor muscle.

The RLNs are supplied by vasanervora from the Inferior Thyroid Artery (ITA) therefore excessive stretching may render them ischaemic.

ANATOMICAL VARIATIONS of the RLNs:

1) Non- RLN:
   Emerges at a right angle from the cervical vagus, often associated with dysphagia lusoria on the right side, incidence ( 0.3-1.6%)and 0.04% on the left side.

2) Variation in branching; extralaryngeal branching with the main motor branch at risk (motor to 4 intrinsic muscles) and sensory to glottic mucosa

3) Variable relation to ITA & Berry ligament:
   a) May take a medial course to ligament
   b) May penetrate the ligament especially the motor fibers and thus endangered at the end of TT
Soustelle’s Classification of Non-RLN:

Type 1: arise from the cervical vagus at 90° and enters the larynx at the upper pole with the Superior Thyroid Artery (STA) and vein.

Type 2a: Arize near origin of STA and enters the larynx above the STA
Type 2b: Arize near the origin of the ITA and enters the larynx below the ITA

Danger in type 1…mobilizing the superior pole
Danger in type2….nerve may be mistaken for ITA and ligated.

Pre-Op CT scan: Dysphagia Luzoria picture alerts the surgeon
US scan : Absence of horizontal Y figure of the brachiocephalic artery predictor of a Non- RLN.

Intra-operative Nerve Monitoring (IONM) is valuable in repeat surgery or irradiated necks.

There is a lack of high level evidence showing the effect of IONM, it also does not monitor sensory function.

Some use methylene blue spray to stain the RLNs and PGs.

NB: Active search and visualization is the cornerstone of RLN protection.

Simon’s triangle: RLN is usually found in this space
-oesophagus medially
-carotid artery laterally
-ITA superiorly

RLN lesions

Transient partial – segmental demyelination or focal conduction block

Transient or Permanent total RLN – severe myelin sheath and axonal damage and neural degeneration

Transient palsies are due to Neuropraxia or axonotmesis depending on:

a. Local myelin damage by: oedema. Poor perfusion or blood clot
b. Loss of axon continuity

Clinical Features

- Hoarseness in unilateral lesions
- Acute stridor and airway obstruction with bilateral lesions
- Dysphagia due to injury to posterior branches of RLN to cricopharyngeus muscle and oesophagus, esp for liquids
• Professional singers and public speakers suffer voice incapacity
• 33% are asymptomatic

**Treatment** – difficult

• Should be symptom focused

Once diagnosis is made – management belongs to the domain of the ENT Surgeon therefore refer early (multidisciplinary team including voice specialists)

**Management**

**Bilateral cord paralysis**

- Both the intrinsic muscles are paralysed and both vocal cords adopt a median or paramedian position due to the unopposed actions of cricothyroid muscle

**Clinical features:**

- Acute dyspnoea at extubation
- Voice change

**Treatment:**

1) Re-initubate and maintain ventilation
   - Some people use corticosteroid therapy to reduce laryngeal oedema
   - 20-60 mg solumedrol IVI over 30 minutes – 8 hourly
   - Dexamethasone 4-10 mg /day
   - Add broad spectrum antibiotic and PPI cover
2) Persistent paralysis – tracheostomy
   Recovery is unusual in these cases – may take upto 12 months
3) Surgical treatment
   - Lateralization of the cords by
     - Arytenoidectomy with posterior cord resection to restore air passage.
     - Implantation of sternohyoid
   - Thyroplasty

**Disadvantage:** voice quality is poor, transient aspiration and dysphagia but patients tend to tolerate this trade off.

**Unilateral RLN paralysis**

All intrinsic muscles except the cricothyroid are paralysed on that side

Asymptomatic in 33% of patients

Voice change which improves with speech therapy

Evaluate the patients’ tolerance, QOL, phonation and respiratory function

**Surgical Corrective Procedures**

1) Medialization of the vocal cord by intra-cordial antologous material injection (fat) via endoscopic or transcutaneously route
2) Thyroplasty – implantation via thyrotomy of various materials into the paralysed cord
3) Traction suture – Adduction of the arytenoids muscle
4) Repair of RLN by primary suture with 6 – 0 prolene or nerve graft

2) **SUPERIOR LARYNGEAL NERVE (SLN)**

Galli Curci nerve – named after the Italian opera singer whose career plummeted post thyroidectomy and “it was ultimately said to be due to physiological aging of the singer”.

The SLN originates just below the dorsal ganglion of the vagus nerve below the jugular foramen.

Divides into the Internal Branch (IBSL) which pierces the thyrohyoid membrane accompanied by the STA provides sensory function to the laryngeal mucosa and the EB-SLN about 1.5 cm from its origin.

EB-SLN nerve is 0.8 m wide and 8-9 cm long. It courses antero-inferiorly along the inferior constrictor muscle with branches of the STA, it then curves anteriorly and medially in the Space of Reeve: Steno thyroid muscle insertion superiorly, inferior constrictor and cricothyroid muscle medially and the superior pole of the thyroid inferio-laterally.

Risk of injury is significant when dissecting the superior thyroid pole vessels.

**Cerneaa's classification** of the EB-SLN in relation to the distance between intersection of EB-SLN and STA and the superior pole of the thyroid gland.

Type 1 (60%) – crosses STA 1cm **above** superior pole to gland.

Type 2 (a) (17%) – crosses STA < 1cm **above** superior pole of gland

Type 2 (b) (20%) – crosses STA **below** the superior pole of gland

Type N, (3%) – EB-SLN **not** identified :– sub fascial; intra muscular route

Both type 2a and 2b are usually at high risk.

EB-SLN supplies motor to the cricothyroid muscle – tilts thyroid cartilage and tenses the vocal cords by modifying the distance between the cricoid and thyroid cartilages

Vocal cord tension and thickness affects the frequency of vibration and therefore affects the characteristic timber of one’s voice.

Injury rates: 0-58% (no protocols available to guide diagnosis and management)

Injury to EB-SLN results in weakness of the cricothyroid muscle

- Decreased voice quality
- Decreased voice projection
- Decrease in pitch

Risk of injury at superior pole

**Landmark** – nerve lies on the inferior constrictor, therefore one may have to divide the sternothyroid muscle and elevate it, especially for large goiters, to make superior pole dissection easier in order that the SLN could be identified.
Gentle caudo-lateral traction of thyroid gland gives good exposure of the superior pole vascular pedicle. Avoid excessive traction and diathermy

Identification of the SLN is by gentle blunt (peanut) dissection of the superior pole from the medial aspect in the avascular space between superior pole and cricothyroid muscle.

Very few (3-10%) superior laryngeal nerves are actually visualized at surgery

Intra-op aids:
- Endoscopic visualization
- Intra-op neuro monitoring

**Injury to the SLN:**

Voice fatigue, reduction in high pitch voice, dysphagia, dyspnoea, worse for singers.

Post op aids: Video stroboscopy – mucosal waves

Electromyography–cricothyroid action potentials

Uncomfortable to patients

**Unilateral, combined RLN and SLN**

All the muscles on one side are paralysed. Vocal cord is in cadaveric position, 3.5 mm from the midline.

**Clinical features:**
- Hoarseness of voice
- Aspiration through ineffective glottis
- Ineffective cough

**Treatment:**
- Speech therapy. (Commonly healthy opposite cord moves to the opposite side of the midline so as to compensate for the paralysis of the opposite side)
- Injection of Teflon to the paralysed cord
- Muscle or cartilage implant into the paralysed cord
- Arthrodesis of cricoarytenoid joint

**3) HYPOCALCAEMIA:**

**Definition**
- Total serum calcium < 2mm/L (< 8.0 mg/dl)
  Normal range (8.5-10.2mg/dl)
- Ionized calcium < 1.1 mm/L (< 0.275 mg/dl)
  Normal range (4.5-5mg/dl)

**Incidence – 2 – 83% depending on definition**
- Some include only symptomatic patients, others do not in management decision

**Long term low calcium occurs in <3%**
Cause – decreased PTH secretion resulting in low calcium levels and usually associated with increased phosphate levels.

Risk of post operative hypocalcaemia – transient or permanent is due to:

1) Venous flow from the superior parathyroid gland is through the thyroid gland therefore reduction in circulating PTH
2) Sub capsular or intra-thyroid parathyroid glands especially inferior glands results in 6-21% accidental parathyroidectomy and transient hypocalcimia – 50% cases
3) Massive goiter, Grave Disease, Thyroid Ca with lymph node dissection risks removal of lower PTGs
4) Devascularization during surgery especially with repeat thyroidectomy

NB – post operative PTH level is a better predictor of hypocalcaemia compared to a single value of low ionized serum calcium and Wand et al propose no supplementation if PTH level ≥5 pg/ml

- Measure PTH 6 hours post op (Grodski Australian meta-analysis)
- Noordzi showed that a decrease of > 65% basal PTH level at 6 hours post op predicts hypocalcaemia (sensitivity 96.4 %, specificity 91.4%)

Visualization of at least 2 glands is enough at operation

Careful protection of PTG blood supply

Devascularized parathyroid glands should be auto-transplanted into sternocleiodomastoid or forearm muscle.

Pre-op detection of Vit D (25OH–Vit D3) below 20ng/ml. Treat with 100, 000U of oral cholecalciferol

Management of Hypoparathyroidism:

Diagnosis: Low PTH and decreased calcium ideally 6 hours post op

Treatment combined calcium and Vit D therapy

Symptomatic patients – especially with tetany

Neuromuscular excitability, parasthesia, carpo-pedal spasm.

Calcium <1.75 mM/l (<7 mg/dl) with Tetany etc., with a risk of cardiac decompensation

1) IVI – calcium Gluconate (1-2 ampoules of 10% solution for 24-48 hours plus,
2) Oral calcium (3g) – carbonate
   - Citrate
   - Chloride plus,
3) Dehydrocholecalciferol 2μg plus,
4) Magnesium 1-2g if there is hypomagnesenaemia

Calcium gluconate IVI: 1 ampoule IV bolus followed by infusion of 1-3mg in 500ml DW over 12 hours for 10 days (to avoid arrhythmic CCF) or until symptoms abate.

Long term treatment dose caution: avoid hypercalciurea and renal stones.

Therefore Aim:
1) Calcium and Phosphate product of 4.44 mM/L
2) 24 hour urine calcium < 300mg (7.5 mM/L)

Parathyroid gland recovery may take months to recover and if so start to wean the patient.

4) **POST OPERATIVE HAEMORRHAGE**

Incidence: 0-6.5%

Causes: Slipped ligature, parenchymal bleed etc.

Clinical Presentation:
- Overt bleed
- Haematoma – compressive/non-compressive with respiratory distress
- Pain
- Can occur beyond 6 hours post op – therefore monitoring in the ward for 24 hours
- Respiratory compromise

Leyre *et al.*, 70 patients of 6744 did bleed and 10% after 48 hours
- NB anticoagulant and antiplatelet therapy do not seem to be risk factors as long as the surgery is meticulous.
- Haemorrhage due to IJV injury may also cause air-metabolism

Management

1) Prevention
- Control hyperthyroidism
- Intra operative haemostasis
- Valsalva maneuvre at completion of TT

2) Treatment
- Urgent re-exploration, evacuation and drain
- Ligate bleeders (rarely found)

5) **OTHER COMPLICATIONS**

1) Thyroid storm – rare
   Mx - pre-op care is excellent these days
2) Tracheomalacia – rare, large goiter,
   Mx – Tracheopexy
   - Silastic rings
3) Horner’s syndrome – rare, ptosis, enophthalmic
   Rx – Conservative
   - Cosmetic surgical correction

Infection

Pneumothorax

Thoracic duct injury and chyle leak

Branchial Plexus injury

Oesophagus
- repair and adequate drainage
- Antibiotics
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SERIOUS COMPLICATIONS AFTER COMMON SURGICAL PROCEDURES: PREVENTION AND MANAGEMENT OF THYROID CRISIS
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Abstract

Diseases of the thyroid gland are common and usually present as goiter. Rarely, patients present due to abnormal function which may not necessarily be associated with enlargement. Majority of thyroid diseases do not require involvement of a surgeon. However surgeons get involved when there is hyperthyroidism, need to exclude thyroid malignancy and for management of compression symptoms. Crisis associated with management may result from consequences of extreme derangement of thyroid function, mass effect or complications of treatment such as thyroidecmy. Thyrotoxicosis crisis is extremely rare. Crisis situations which a surgeon is likely to encounter are airway obstruction, bleeding, bilateral recurrent laryngeal nerve damage and hypocalcaemia.

Introduction

Goiter is common but involvement by a surgeon is necessary only if there is abnormal thyroid function, fear of malignancy, compression symptoms, retrosternal extension and rarely for cosmesis. Irrespective of how a patient would have presented clinical evaluation is standardized. Similarly, the first step in diagnostic investigation is determination of serum thyroid stimulating hormone level. Probability of thyroid malignancy is based on a combination of personal, family and environmental risk assessment on history. Compression symptoms are likely in patients who have large goiter, asymmetric goiter with tracheal shift and goiter extending retrosternally. Diagnosis of retrosternal extension is clinical. Chest and neck CT scan is mandatory for patients who have retrosternal extension to enable grading and thyroid malignancy with extensive lymphadenopathy.

Thyrotoxic crisis

A surgeon is likely to encounter hyperthyroidism than hypothyroidism and may therefore occasionally have to deal with cases of thyrotoxic crisis or so called thyroid storm (Kearney and Dang, 2007; Ross, 2011; Jit et al, 2015). Causes of hyperthyroidism which can lead to thyrotoxic crisis are Graves’ disease, toxic multinodular goiter and toxic adenoma. It is however relatively more likely in Graves’ disease generally and in elderly, patients with underlying cardiovascular disease or have severe hyperthyroidism i.e. hyperthyroidism with deranged cardiac function or marked elevation of thyroid hormones (T4 or T3 more than twice above the upper limit of normal range) irrespective of the underlying cause.

Any stress can precipitate thyrotoxicosis crisis. Such stress include infections, surgical procedures (not necessarily thyroidecmy), following I-131 ablation. Almost every organ-system in the body is affected during a thyrotoxic crisis and the manifestations are mainly due to sympathetic hyperactivity. The following systems are most frequently involved: central nervous system, cardiovascular system and gastrointestinal system. Common manifestations include deranged level of consciousness, cardiac arrhythmias, cardiac failure, malignant hyperthermia, etc. Rarely, apathetic thyrotoxic storm may occur in elderly patients.
As it was mentioned previously thyrotoxic crisis is unusual in patients diagnosed with hyperthyroidism who are adequately prepared medically for thyroidectomy or radioablation. Because of a relatively long half-life of thyroid hormones (especially T4), it is advisable to extend treatment with antithyroid drugs beyond the day of definitive treatment to prevent a crisis which may be induced by a surge of hormones released during surgery or radiation driven destruction of the gland. Although pre-procedure preparation with beta blocker alone may be adequate for patients who have mild hyperthyroidism the same is not true for moderate and severe thyrotoxicosis. Clinical and biochemical euthyroidism has to be secured before intervention such as thyroidectomy is implemented. Furthermore, perhaps the major preventative strategy if for surgeons to stop doing thyroidectomy for hyperthyroidism except in less than 1% of patients presenting with hyperthyroidism irrespective of the cause.

Patients in thyrotoxic crisis should be treated in ICU and treatment package include general supportive measures such as fluid therapy and lowering of body temperature, strategies to blunt sympathetic over activity such as using intravenous beta blockage, blocking peripheral conversion of T4 to T3 (propranolol, corticosteroids and propylthiouracil), blunting/stabilizing of receptors (corticosteroids), blocking hormogenesis (antithyroid drugs and iodine) and blocking the release of pre-formed thyroid hormone (iodine containing intravenous contrast or lithium). Diuretics such as furosemide are contraindicated. It is also important to treat the precipitating cause. Predicted mortality in patients who has thyrotoxicosis crisis is 30%.

**Myxoedema coma**

Myxoedema coma following thyroidectomy is rare. Elderly patients are most at risk (Bajwa and Jidal, 2015). It is only expected after more than 4-6 months following total thyroidectomy if a patient is not given replacement therapy. However hypothyroidism leading to myxedema coma may occur even after subtotal thyroidectomy if the remaining thyroid tissue is functionally insufficient such as in MNG or the procedure was done for Graves’ disease and a patient has high levels of destructive autoantibodies.

With adequate dosing and attention to pharmacodynamics of thyroxine severe hypothyroidism should not occur while a patient is on treatment. Acute hypothyroidism may be induced by a critical illness in patients who seemingly were in a euthyroid state. Predominant manifestations involve same organ-systems i.e. central nervous system, cardiovascular and gastrointestinal systems which are affected a thyrotoxic crisis. Recombinant TSH or T3 can be used to shorten the duration a patient is left off thyroid hormone replacement and suppression in preparation for whole body scan during follow up of differentiated cancer of follicular cell origin following total thyroidectomy.

A relatively early consent following treatment of hyperthyroidism is potential of worsening of Graves’ ophthalmopathy if a patient who has severe eye-disease is allowed to become hypothyroid. Treatment of myxedema is mainly supportive and background adrenal insufficiency should always be suspected. Therefore thyroid hormone replacement should be preceded by infusion of glucocorticoids. Precipitating cause must also be sought and managed.
Airway obstruction/compromise

Airway challenges may occur pre-, intra- or post-operatively. Actually, acute airway obstruction might have been the indication for emergency thyroidectomy. Patients at risk of airway obstruction include individuals who have large nodular goiter and goiter with retrosternal extension (Agarwal et al, 2007). Difficult airway intubation is to be expected. Pre-operative CT scan should be done routinely. Consulting a team of anesthetists and ensuring that resources necessary for management of difficult airway in theatre is advisable. Invariably it usually end up as an anti-climax.

Post-operative causes of airway compromise include bilateral recurrent laryngeal nerve damage, tracheomalacia and haematoma. The risk for bilateral recurrent laryngeal nerve damage is higher during thyroidectomy for large nodular goiter, retrosternal goiter and malignant goiter associated with extensive lymph node enlargement as the course of the nerve might have been distorted and in re-do thyroid surgery. Unilateral nerve damage should always be assumed in patients who have had prior thyroid operation even if the voice is normal as the contralateral vocal cord could have undergone compensatory hypertrophy. Routine identification of the RLN throughout its course including of its branches is mandatory. Nerve monitoring is not helpful. Tracheostomy is absolutely necessary in patients who have suffered bilateral RLN damage.

Tracheomalacia is likely in patients who have huge goiter (Agarwal et al, 2007). Key treatment options include leaving a patient intubated (endotracheal tube) for 3-5 days or prophylactic tracheostomy. Strategies to prevent peri-operative bleeding following thyroidectomy include measures to reduce vascularity in thyrotoxic goiter (beta blocker therapy and iodine), meticulous dissection, ligation in continuity of inferior thyroid artery and using drains for complicated thyroidectomy or if thyroidectomy leaves a large potential space. Options for treatment of airway obstruction due to a haematoma include removal of sutures, re-operation or aspiration. Prophylactic drains placed during the operation are useless as the drainage amount often does not correlate with symptoms.

Hypocalcaemic crisis

Hypocalcaemia following thyroidectomy may be due to “hungry bone syndrome” or hypoparathyroidism. The hungry bone syndrome is due increased oestoclastic activity in patients with hyperthyroidism whereas hypoparathyroidism is mostly due to devascularization of parathyroid glands (Ciftci et al). Only in rare occasions are all parathyroid glands are removed. Preventive strategies include high index of suspicion, meticulous dissection, ligation of branches of the inferior thyroid artery on the gland, appropriate selection of energy device, avoiding of energy device when dissecting within 3mm of parathyroid glands, intra-operative intact parathyroid hormone and parathyroid gland auto-transplantation.

Conclusion

Severe complication following thyroid procedures include bleeding, airway obstruction/compromise resulting from bilateral RLN damage, tracheomalacia or haematoma, hypocalcaemia and extreme hypo- or hyperfunction. Crisis resulting from hypo-or hyperfunction are extremely rare and elderly individuals are most at risk.
References

MANAGEMENT OF COMPLICATIONS OF PAROTIDECTOMY
Dr J K Jekel – Dept surgery University of Pretoria

The intimate relationship of the parotid gland and the facial nerve dictates that the facial nerve continuity be sacrificed occasionally for certain neoplastic lesions. This devastating insult to emotional expression is never entirely overcome after the facial nerve is transected. Although patients are told that facial paralysis will follow such operations, it is difficult to communicate to them the full impact of facial paralysis.

Parotid surgery is mostly undertaken in an attempt to remove benign tumours and thus complications should be minimized at all costs.

When the surgery is done to remove malignant tumours the histology should be known and the extent of the tumour evaluated before any attempt at removing the tumour is undertaken. This is important as the possibility of any complication can be pre-empted and fully discussed with the patient prior to the surgery. This is especially important if the facial nerve is possibly involved and decisions about sacrificing the nerve have to be made. Pre-operative discussions are a key factor in the patients understanding and acceptance of the surgical treatment.

Salivary complications of parotid surgery must also be discussed pre-operatively as some patients will have decreased saliva production.

Cosmetic deformities of the face – especially if extensive surgery combined with mandibulectomy and neck dissections are undertaken can be expected. If the patient is to receive post-operative radiotherapy these deformities can be extensive.

The complications of parotid surgery will be divided into:

1. Local
2. Nerve damage
3. Salivary
4. Frey’s syndrome
5. Local cosmetic effects

Local complications

The local complications mostly consist of wound complications and must be treated if and when they occur. The best treatment of local complications is prevention and pre-operative planning should prevent most complications.

1. Wound infection occurs in patients where infective conditions are associated with the indication for surgical intervention. These should be treated pre-operatively with antibiotics or with antibiotic prophylaxis. Leakage of saliva into the wound causes severe septic complications and this can be prevented by proper drainage with suction drains.
2. Wound breakdown can be disastrous as this will usually expose the facial nerve and can cause damage to the nerve. If the tumour is close to the skin or the skin flaps are large with tedious blood supply it might be wise to use flaps (rotation or free tissue transfer) in an attempt to close the surgical defect. The skin must never be sutured under tension as this compromises blood supply to the skin flap and will lead to skin necrosis. Skin grafts are not indicated to cover the facial nerve.

3. Salivary fistulas occur in some cases – especially if a large portion of the parotid remains after the surgery. This can be prevented by removing most, if not all of the parotid tissue. To remove the deep lobe does however increase the possibility of nerve damage dramatically. Division and ligation of the ducts as they are encountered during the surgery as well as proper suction drainage decrease the possibility of fistulation. The treatment of fistulas by surgery is difficult and nerve damage as complication is very high. A short course of superficial radiotherapy with electron radiation usually stops saliva production and thus solves the problem.

Nerve complications

The most devastating aspect of salivary gland surgery is certainly Facial nerve damage. Other nerves can however be damaged and this must be discussed preoperatively

1. Greater Auricular nerve. The greater auricular nerve is the sensory nerve to the ear and lateral part of the face. It exits behind sternocleidomastoid muscle and has a superficial course to the ear. Its branches are frequently damaged as the skin flaps are lifted. Most of the branches run through the parotid substance but can be dissected out if the nerve is not close to the tumour.

2. Facial nerve damage. The course of the facial nerve through the substance of the parotid makes facial nerve damage an ever possible complication. Exploration of the main trunk with visualisation of the facial nerve and dissection on the plane of the nerve is the only way to prevent nerve damage. Nerve stimulators may be of value to detect the nerve. If the main trunk cannot be identified it is essential that the distal branches be dissected out and the nerve followed back to the main trunk.

A) If a planned resection of the nerve is done immediate reconstruction must be attempted. The surreal nerve is usually harvested from the leg and used as a cable graft

B) Nerve transposition can be attempted but should be used only if cable grafts are not possible. Hypoglossal-Facial anastomosis is the most successful of these anastomoses. Other nerves used include spinal accessory and phrenic nerve but these cause more disability than the Hypoglossal.

C) Facial reanimation by muscle transfer is used widely to attempt to minimize complications to the eye and prevent drooling. This should only be attempted if nerve
reconstruction cannot be done or has failed. The muscle used most commonly and successfully is the Temporalis muscle. The Masseter has also been used.

D) Lateral Tarsorrhaphy is widely used to help protect the eye and prevent corneal ulceration.

The lateral parts of the upper and lower eyelids are sutured together after the skin has been denuded in an attempt to prevent ectropion and make closure of the eyes easier. The use of Gold weights in the upper eyelid was used but this is expensive and not used any more.

**Salivary complications**

Parotidectomy done for benign disease usually does not lead to a dry mouth as the other salivary glands function normally. Elderly patients do however frequently complain of dry mouth after parotid surgery. If radiation is planned as part of the treatment this aggravates Xerostomia. The use of newer radiotherapy modalities like IMRT can decrease xerostomia.

There is no treatment for xerostomia but these patients must be encouraged to drink water frequently and the use of chewing gums may help. Transient loss of taste is associated with xerostomia but this can be permanent.

**Frey’s Syndrome or Gustatory sweating.**

This is extremely common after parotid surgery and is characterized by flushing and sweating on the ipsilateral side of the face – especially during eating. The pathophysiology is the misdirected regeneration of sympathetic and parasympathetic fibres to the cholinergic receptors of the skins sweat glands. The treatment is directed to blocking the abnormal pathway.

Surgical procedures include resection of the nerve pathway, resection of the skin with grafting of the defect or destruction of the nerve fibres by alcohol injection.

The best treatment currently is injection of Botox.

**Local cosmetic effects.**

Most patients will accept a cutaneous concavity in the parotid fossa but in certain individuals the defect is less acceptable. If radiotherapy is not part of the treatment the surrounding skin and hair are normal and the defect can easily be hidden. Radiotherapy does however also make free grafts of skin and muscle difficult and most reconstructions must be done after radiotherapy effects have subsided.

**The effect of radiotherapy**

Radiotherapy is frequently used as adjuvant treatment in the management of malignant parotid tumours.

As discussed above this usually aggravates the complications encountered during parotid surgery. If radiation is planned the decision is usually made pre operatively and the surgery and reconstruction modified to minimize these effects.
It is also important to look at oral hygiene and to extract teeth if necessary in order to prevent the problems associated with loss of salivary production and osteoradionecrosis.
FAILURE OF BREAST RECONSTRUCTION AFTER BREAST CANCER SURGERY
Dr S. Sehlale, Department Plastic Surgery

There is increased demand for post mastectomy breast reconstruction but the reconstruction options are rapidly evolving

The choice of reconstructive options is dependent on:

PATIENT FACTORS
- Lifestyle of the patient/patient’s wishes
- Desire to have babies
- Body habitus
- Down time of procedure
- Risk factors
- Cancer prognosis

BREAST FACTORS
- Type of mastectomy +axillary procedure
- DXT
- Failed reconstruction
- Timing of reconstruction
- Status of opposite breast

GOALS OF RECONSTRUCTION:
1. Volume (size of projection)
2. Envelope (shape & position)
3. Symmetry with contralateral breast
4. NAC

TECHNIQUES OF RECONSTRUCTION
- Alloplastic (Expander/implant)
- Alloplastic & Autologous (flap & implant)
- Autologous (flaps)

RECONSTRUCTIVE FAILURE – FAILURE TO ATTAIN RECONSTRUCTIVE GOALS
- Technical failure
- Procedural complications
- Unsatisfactory results

RECONSTRUCTIVE OUTCOME ANALYSIS
1. Complications Revision after prosthetic reconstruction is generally higher than for autologous.
2. Lower revision rates for autologous reconstruction
   - Lower overall major complications.
   - Better aesthetic outcome.
   - Long lasting acceptable results.
CONCLUSION

“It is important to have an open mind and to rethink the treatment strategy … If the situation calls for a change in strategy and if the surgeon is flexible enough to pursue a new course, the reward will often be vastly improved result and a satisfied patient” Kroll & Freeman.
COMPLICATIONS OF CAROTID STENOSIS SURGERY
T.V. Mulaudzi, Vascular Surgeon

Introduction

The improvements in medical and surgical management in carotid artery stenosis have led to the steady decline in morbidity and mortality from ischemic stroke in the past several decades. However, this has not translated to a decline in the incidence of ischemic stroke. Stroke is a truly debilitating condition and impacts significantly on the patient’s wellbeing, the family structure and is a burden on the social and healthcare systems. The role of surgical intervention on extra cranial carotid artery disease has had a telling effect on improving the outcomes of patients with atherosclerotic carotid artery disease.

Atherosclerotic disease of the carotid artery is seen to be a result of intimal injury followed by a process of inflammation, smooth muscle proliferation finally followed by calcium deposition. The resultant stenosis causes changes in flow and induces the phenomenon of flow separation. Furthermore, and probably more importantly, the process leads to plaque rapture, haemorrhage into the lesion and thrombotic events which subsequently may cause embolization to the cerebral circulation. Currently there is ongoing debate concerning the management option for carotid stenosis. Management of high grade stenosis in asymptomatic patients was until recently considered appropriate with pendulum swinging toward medical treatment for all asymptomatic patients without clear unstable plaque characteristics. Intervention is clearly indicated in the symptomatic patients with appropriate stenosis grades and in these patients surgical intervention in form of carotid endarterectomy is the procedure of choice. The debate concerning the management options is beyond the scope of this article, however, and this article will focus on the complications associated with carotid endarterectomy surgery.

Patients tolerate an uneventful carotid endarterectomy very well and hospital care does not have to continue beyond a 48 hours. However, complications of a carotid endarterectomy can be devastating and sometimes irreversible. Fortunately death is infrequent and often secondary to myocardial infarction. The benefits of carotid endarterectomy are largely based on the premise that complications are kept to a minimum. In instances where the complication rate of a centre is higher than the acceptable threshold the benefit of carotid endarterectomy may no longer be warranted.

Complications associated with carotid endarterectomy include neurological, cardiac and, as with any surgical procedure, generic complications occur in form of: anaesthetic related, respiratory, bleeding and infection. The specific complications of nerve injury, cerebral hyper perfusion syndromes and carotid restenosis will be discussed in more detail.

Myocardial & Haemodynamic complications

Coronary complications are the leading culprit of all carotid endarterectomy complications. It is believed that myocardial infarction (MI) may occur between a quarter to half of all the perioperative deaths and is the most common cause for late deaths. Upto half of the patients undergoing carotid endarterectomy have symptomatic coronary artery disease (CAD). Despite these findings the perioperative cardiovascular event rates are on a decline. This change in status quo appears to be driven by improved medical management of these patients.

Haemodynamic instability may be considered a ‘symptom’ of surgery rather than a complication, but because postoperative haemodynamic instability after carotid endarterectomy may be linked to the surgery itself it is prudent to discuss it as a complication. The carotid sinus baroreceptors inhibit sympathetic activity when stimulated.
Evidence suggests that endarterectomy of the atherosclerotic plaque that is found at the bifurcation of the carotid artery creates a heightened sensitivity to baroreceptor mechanism that leads to decreased central nervous system sympathetic activity and results in bradycardia or hypotension or a combination of these. The hypotension mediated through these mechanisms usually occurs within 2 hours of the operation. Intraoperative infiltration at the carotid sinus has been shown to reduce the occurrence of this complication. On the other side of the spectrum post-operative hypertension (HPT) is associated with preoperatively elevated blood pressure. A hypothesis implicates denervation of the carotid sinus as the cause of this post-operative HPT. Recently the validity of the hypothesis has been questioned and other mechanisms, including cerebral noradrenaline and renin release have been looked at. This HPT response normalises in up to 80% of individuals within 24 hours postoperatively.

In contrast to this early haemodynamic changes is the cerebral hyper perfusion syndrome, which occurs within several days postoperatively. The reported incidence ranges between 0.4 – 7.7% after a carotid endarterectomy and is often preceded by a severe headache and is hallmarked by significant HPT and acute neurological deficit. The most devastating consequence is an intracerebral haemorrhagic stroke and mortality rates of the HPS reach 75-100% for the severe cases. The aetiology appears to be ineffective cerebral blood flow calibration secondary to a carotid stenosis which has been relieved with the carotid endarterectomy. HPS appear to be more frequent in patients with high grade contralateral carotid stenosis and in patients who undergo surgery for contralateral high grade stenosis within 3 months of the index carotid endarterectomy for the ipsilateral stenosis. SVS guidelines recommend a strict blood pressure control in the postoperative period, not exceeding SBP 140mmHg and DBP 80 mmHg.

Neurological Complications

Neurological complications can be divided into ischemic strokes and local nerve injuries, nerve injuries being the more frequent of the two. The rate of 30 day stroke/ death in the original symptomatic trials was 5.8-6.7% and 7.5% in the NASCET 13 and ECST trials respectively. These stroke rates, of course, were lower in the asymptomatic patients, as is seen in the ACAS (2.3%) and ACST (3.1%) trials. In the more recent CREST trial the 30 day post-operative strike rate in the carotid endarterectomy group was 3.2%. 215 The strokes which occur intraoperatively or in the immediate postop period may be diagnosed promptly and have the potential to be treated with catheter directed thrombolytic agents. Flow must be restored within 1-2 hours to ensure success. Riles et al examined the rate of perioperative strokes in their series and divided them into categories. The overall perioperative stroke rate was 2.2% and six categories were identified:

1. Ischemia during clamping,
2. Postoperative thrombosis and embolization,
3. Intracranial haemorrhage,
4. Stroke from other mechanisms related to surgery,
5. Strokes unrelated to reconstructed artery,
6. Unknown.

Cranial nerve dysfunction incidence varies considerably, namely 5-20% as seen in retrospective studies. Unfortunately the different series have heterogeneous groups and direct comparison is difficult. The description of dysfunction is also not standardised in these studies. CREST trial cites an incidence of 4.7% of cranial nerve injury. This dysfunction is often transient and resolves at different time points. In one study all injuries resolved within 5 months and in another almost all deficits were gone within 12 months of the carotid endarterectomy. Cunningham et al, investigator for the ECST trial cited a rate of 0.5%
permanent cranial nerve damage in the trial. In a paper by Schauber et al. the permanent cranial nerve damage was 1.1% of patients.

Cranial nerve injury is rarely from the structure being severed but most often related to blunt injury related to the dissection. Stretching and retraction of the tissues inadvertently stretches a nerve and may result in dysfunction. An increased incidence of CN injuries has also been shown in cases where the carotid endarterectomy takes longer than 2 hours. Injuries to branches of the cervical plexus are frequently unavoidable and occur with the skin incision. The transverse cervical nerve (C2 and C3) exits near the anterior border of the sternocleidomastoid (SCM) muscle and supplies cutaneous sensation to the anterolateral neck. Damage to this branch results in an insensate area medial to the incision. The greater auricular (C2 and C3) nerve exits the SCM muscle, then ascends the upper neck to supply sensation to the skin overlying the inferior parotid gland and the lower earlobe.

Facial nerve is almost never damaged unless extensive manoeuvres for a high bifurcation have to be used. However, branches of the facial nerve are at risk. The first branch, cervical, gives motor innervation to the platysma muscle. This nerve is routinely sacrificed without postoperative disability. A second branch, the marginal mandibular nerve, courses inferior and parallel to the edge of the mandible to supply facial muscles responsible for maintaining proper function of the lateral mouth. Injury results in a lower lip droop on the ipsilateral side and usually occur from retraction. Recovery of neuropraxia in almost universal.

**Cranial Nerve IX: Glossopharyngeus**
The glossopharyngeal nerve is found high in the neck, running between the internal and external carotid arteries. It is injured during high dissection of the internal carotid artery. The nerves of Herring (or DeCastro), which supply the carotid sinus, are branches of the ninth cranial nerve.

**Vagus (CN X)**
The vagus nerve usually descends the neck posterolateral to the carotid artery and posteromedial to the internal jugular vein. Injury to the vagus nerve and its branches are the most commonly injured clinically important nerves. Most injuries to the main vagus trunk are clamp related. A branch of the vagus nerve, the superior laryngeal nerve, travels with the superior thyroid artery. The nerve is frequently adherent to the posterior portion of this artery. This nerve supplies motor innervation to the cricothyroid muscle, denervation results in difficulty singing and talking, with limitations on high notes. A second branch of the vagus nerve, the recurrent laryngeal nerve, is rarely injured during a carotid endarterectomy. However, the possibility of a "nonrecurrent" recurrent laryngeal nerve must be kept in mind.

**Spinal accessory nerve (CN XI)**
The spinal accessor nerve exits the base of the skull through the jugular foramen and courses beneath the posterior belly of the digastric and SCM muscles to supply innervation to the SCM and the trapezius. Injury results in complete paralysis of the SCM muscle and partial paralysis of the trapezius muscle. Deficits are noted by the presence of a winged scapula and difficulty in raising the shoulder.

**Hypoglossus nerve (CN XII)**
The hypoglossal nerve should be identified during a carotid endarterectomy dissection. Its course is usually above the bifurcation and anterior to the internal and external carotid arteries. Frequently it is tethered down by an artery and vein to the SCM muscle. Division of the hypoglossal nerve produces ipsilateral deviation of the tongue and can be quite debilitating causing trouble with speech, eating, and drinking. Fibers of the hypoglossal
nerve descend joining fibers of CI to form the anasa cervicalis. Division of this nerve is done with impunity leaving no obvious loss of function.

**Bleeding and Infection**

Complications associated with infection are uncommon. Reported incidence of wound infection ranges from 0.09 – 0.15%. This is probably attributed to the generous blood supply in the neck. Increased infection with prosthetic patch, while theoretically proposed, has not been shown. Many surgeons are changing over to the use of bovine pericardium patches and these are thought to be very resistant to infection.

Postoperative bleeding is mostly attributed to capillary oozing and associated with perioperative antiplatelet use and heparin administration intraoperatively. Haemorrhage is uncommon and reported incidence ranges between 0.7-3%

**Restenosis**

Late complications in form of carotid artery restenosis are known to occur in 5-22% of patients. Importantly, it appears that only 3 % of these are symptomatic. Recurrent stenosis develop more frequently in female patients, patients who persist with smoking, poorly controlled HPT, high cholesterol and diabetes mellitus. Restenosis may be divided into early, < 2 years and late more than 2-3 years. The early restenosis is secondary to neointimal hyperplasia (NIH) and has less likelihood of ulceration or thrombosis as it does not exhibit the typical atherosclerotic plaque surface characteristics. NIH has also been shown to regress in up to 35% of cases. A surgeon should have a high threshold for reintervention for restenosis and should be reserved for very high grade lesions or unstable lesions. Treatment modalities include CAS or carotid endarterectomy with weak data to support each modality of treatment. Albeit both carry higher risks than the initial carotid surgery.

**Conclusion**

Current literature supports the use of endarterectomy for carotid stenosis in patients with the appropriate indications. Because of the fact that most perioperative strokes occur as a result of technical imperfections, meticulous care must be given to all aspects of the procedure and medical perioperative management optimised in order to avoid potentially devastating complications. Detailed knowledge of anatomy is required to prevent surgical ‘mishaps’ and especially inadvertent local nerve injury. Restenosis is often clinically asymptomatic and repeat corrective procedures must be carefully considered on individual patient basis.
References

ARE SURGEONS VIGILANT ENOUGH FOR THROMBO-EMBOLIC DISEASE? Y-N
S.C Tsoetseri: Vascular Surgeon

Introduction

Acute DVT is a common disease with potentially life-threatening consequences and long-term life-altering complications. It imposes huge economic burden on the society. The disease is difficult to diagnose because of the lack of sensitivity and specificity of its clinical manifestations. Diagnostic algorithms are needed for safe and effective diagnosis. The treatment is standard anticoagulation therapy which reduces significantly the mortality risk and recurrence of the disease but does a poorer job when it comes to the long-term morbidity of the disease. Active management of acute of acute DVT, especially proximal iliofemoral DVT, using catheter directed thrombolysis with or without mechanical thrombectomy modalities is gaining momentum and may prove to be the standard therapy of the future as it adds to the standard therapy the significant reduction of the long term morbidity specifically post thrombotic syndrome.

Etio-pathogenesis

In 1860, Rudolf Virchow described the famous triad of factors that are still considered the main factors in the pathogenesis of venous thrombosis.

Natural History

The relative balance between organization, thrombolysis, propagation and rethrombosis determines the outcome of human acute DVT. Venous duplex ultrasound which permits individual venous segments to be observed over time has documented that recanalization does occur in most patients after an episode of acute DVT. Van Ramshorst and associates found that most recanalizations occur within the first 6 weeks, with flow re-established in 87% of cases. Killewiand colleagues reported a linear decrease in thrombus load, and by 24 – 36 weeks, only 26% of the original thrombus remained. However, recanalization may continue albeit at a slower rate for months to years after an acute event. Rethrombosis has also been reported in the affected segments. The incidence of recurrent thrombosis is 5.2% of those who were treated with standard anticoagulation for 3 months compared with 47% in those who were inadequately treated.

Complications

Pulmonary embolism (PE) is the most devastating complication of acute DVT. The majority of these incidents are clinically silent. Recent studies have shown that in patients with symptomatic acute DVT of the lower extremity, 50 – 80% develops asymptomatic PE. It was also found that symptomatic PE complicates 17% of patients with proximal upper extremity acute DVT. Patients with symptomatic PE have 18 times the mortality rate compared with patients with acute DVT alone. Patients who survive the acute PE event are still at risk of development of chronic thromboembolic pulmonary hypertension.

Post thrombotic Syndrome (PTS) is a group of clinical manifestations in patients who have history of acute DVT due to ambulatory venous hypertension in their affected limbs. It is the most common late complication of acute DVT and is responsible for a greater degree of
greater of chronic socioeconomic and quality of life morbidity. The cause of ambulatory venous hypertension is a combination of venous reflux secondary to valvular incompetence and residual luminal obstruction.

**Diagnostic strategy**

Although duplex ultrasound is extremely accurate in the diagnosis of acute DVT, its overuse in the evaluation of patients with suspected acute DVT imposes intense burden on the hospital resources. The suspicion of acute DVT in hospitalised patients requires confirmatory testing, usually duplex ultrasound due to the high pre-test probability of the disease in that population.

The situation is different in the outpatient setting where the prevalence of the disease and hence the pre-test probability are much lower. In those patients, the first step is to stratify their pre-test probability for the presence of acute DVT using one of the available risk stratification scores, commonly the Wells score. Patients with low and intermediate probability are exposed to D-dimer testing. In case it is negative, that essentially excludes the diagnosis of acute DVT. On the other hand, patients with high probability for DVT will require duplex ultrasound.

**Treatment**

The therapeutic goals of treatment of acute DVT of the lower extremity include prevention of clot extension and possible resultant PE and relief of lower extremity pain and swelling as a consequence of venous obstruction. The long term goals include preservation of venous valvular function and patency to prevent the development of PTS.

The contemporary management of acute lower extremity DVT includes these alternatives:

1. Standard anticoagulation alone
2. The use of thrombolytic therapy in conjunction with anticoagulation
3. Endovenous thrombectomy using designed to mechanically remove the thrombus combined with thrombolytic therapy in addition to anticoagulation
4. The use of iliac stenting in patients with iliac stenosis
5. Inferior vena cava filters in patients with a contraindication to anticoagulation estimate for recurrent VTE was imprecise. The American College of Chest Physicians current guidelines recommend the placement of an inferior vena cava filter in patients with documented VTE or PE and a contraindication to anticoagulation, complications of bleeding as a result of anticoagulation, failure of anticoagulation, or VTE despite therapeutic anticoagulation.
6. Compression stockings applying an ankle pressure of 30 to 40 mm Hg and a lower pressure higher up the leg (i.e., graduated pressure) should be started as soon as feasible after starting anticoagulant therapy.
7. Thromboprophylaxis, either mechanical or pharmacologic is recommended according to the patient’s risk
References

This presentation will deal briefly with (a) the present situation in public health sector regarding treatment for deep vein thrombosis to prevent pulmonary embolism; (b) the problems of procurement in the public sector; (c) general principles regarding medical practice in a resource-starved environment; (d) when healthcare practitioners will be liable for negligence or malpractice; (e) when healthcare administrators will be liable for negligence; (f) vicarious liability; and (g) when healthcare practitioners can be absolved. The participants will then be given a scenario to discuss.

The present situation in public health sector regarding treatment for deep vein thrombosis (DVT) to prevent pulmonary embolism (PE).

Research indicates that PE arising from DVT accounts for 10% of all hospital deaths. VTE prophylaxis is used to prevent DVT and subsequent PE. Patients are assessed using patient-related and procedure-related risk factors. Patient’s undergoing low-risk procedures with no patient-related risk-factors, require no specific prophylaxis. Patient’s undergoing low-risk procedures with additional patient-risk factors, or those undergoing higher-risk procedures with or without patient-related risks, are put on a LMWH according to the risk classification in the protocol. (see generally, BF Jacobson et al ‘Venous thromboembolism – prophylactic and therapeutic practice guideline’ (2009) 99(6) SAMJ 467-473)). For very high-risk procedures attempts are made to arrange intermittent compression devices (IVD) to be applied in theatre and used until the patient is mobile – but IVDs are not always available. The use of IVDs is based on surgeon preference and is not in the protocol.

Problems of procurement in the public sector

IVDS and the necessary consumables are ordered and supplied by the procurement department of the hospital.

Stock controllers are supposed to check and ensure the availability of consumables and other relevant stock such as LMWHS and IVDS. Usually LMWHS are ‘readily available and used frequently’. IVDS are not readily available. Often there is a stock problem and the correct consumables are not available because the items have not been ordered or the budget has been overspent.

General principles regarding medical practice in a resource-starved environment

In a resource-deficient environment, liability for medical malpractice depends on whether there was intentional or negligent wrongful conduct by the parties concerned. Where the department of health is involved, vicarious liability will depend on whether its employees were acting in the course and scope of their employment when they committed the wrongful acts or omissions. Departments of health and public sector hospitals may be liable for the
wrongful conduct of their healthcare service staff as well as their administrators and managers.

Departments of health and public hospitals will be vicariously liable for the intentional or negligent wrongful acts or omissions of their clinical health care and support staff acting in the course and scope of their employment. The department of health and public hospitals will be liable for harm caused to patients arising from a shortage of resources due to intentional or negligence conduct by their administrative employees.

**When healthcare practitioners will be liable for negligence or malpractice**

Healthcare practitioners are expected to exercise the degree of skill and care of reasonably competent practitioner in their particular branch of profession. (*Castell v De Greef* 1993 (3) SA 501 (C)). The more complicated the procedures the greater the skill and care that must be exercised - within available resources. (*Collins v Administrator, Cape* 1995 (4) SA 73 (C)). The test for negligence is whether a reasonably competent practitioner in the position of the defendant ought to have foreseen the likelihood of harm and guarded against it. A failure to exercise the required degree of skill and care resulting in injury and damage to a patient will result in a claim for professional negligence. An intentional wrongful act against a patient will result in a claim for malpractice (e.g. breach of confidence; failure to obtain an informed consent).

**When healthcare administrators may be held liable for negligence or maladministration**

Healthcare administrators are expected to exercise the degree of skill and care of reasonably competent people in their position in the organisation. The test for negligence is whether a reasonably competent person in the position of the administrator ought to have foreseen the likelihood of harm and guard against it. Thus administrators who intentionally overspend their budgets due to engagement in unlawful tender transactions; wasteful expenditure on travel, entertainment and study tours; or the overuse of consultants etc. May be guilty of maladministration. Other examples are negligently failing to repair or replace medical equipment, or to order medical items and drugs when funds are available, or diverting funds from health care services to the detriment of patients. (D McQuoid-Mason ‘Establishing liability for harm caused to patients in a resource-deficient environment’ (2010) 100(9) SAMJ 573-575).

**Vicarious liability**

Vicarious liability means that one person is liable for another person’s wrong act even though the first person is not at fault. Vicarious liability applies where a person employs another as a ‘servant’ and the latter unlawfully harms a third person while acting ‘within the course and scope of their employment’. Subject to the above, departments of health may be liable for wrongs committed by people employed by them whether they act negligently or intentionally. The employees – whether health care service practitioners or administrators may also be held liable personally. Health care practitioners and administrators are not liable
for the acts of independent contractors contracted by them – unless they negligently failed to prevent the harm. *(cf S v Kramer 1987 (1) SA 887 (W)).*

**When health care practitioners can be absolved**

Health care practitioners can be absolved from liability:

- Where they did not negligently or intentionally cause harm to the patient or anyone else;
- Where they are faced with an emergency situation and have acted reasonably outside their speciality because nobody else is available to assist – they will be judged by the standard of how a reasonably competent practitioner with their skill and experience would have acted in the same situation;
- Where they are faced with a shortage of resources such as drugs or equipment, if they acted in the way a reasonably competent person in their branch of the profession would have acted in the same situation;
- Where a ‘material risk’ against which a patient was warned manifests itself and there was no negligence by the health care practitioner.

*[Note: Both health care practitioners and administrators they may be held jointly and severally liable to the injured persons in which case damages will be apportioned].*

**Scenario**

A healthy, active, 40 year patient presents at a State hospital emergency room complaining of shortness of breath on exertion and chest pain. He is subjected to (a) a non-stress test (during which he fainted); (b) a CT scan without contrast (‘because it would be too hard on his kidneys’); (c) a D-Dimer blood test (which was abnormal and elevated); (d) a V/Q perfusion lung scan (in which part of his lungs were hidden by his arms and the radiologist interpreted as ‘low probability of PE’); and (e) an echocardiogram (indicating massive enlargement of the right ventricle and right atrium, pulmonary hypertension, and moderate Tricusbid regurgitation). A cardiologist and pulmonologist are consulted.

The cardiologist after seeing the other test results, relies soley on the V/Q scan and diagnoses obstructive sleep apnea. The pulmonologist does not examine the echocardiogram and diagnoses asthma and obstructive sleep apnea and like the cardiologist indicates that the results of the V/Q scan are sufficient to rule out pulmonary embolism. The patient’s discomfort continues and at another hospital he is diagnosed as having a ‘massive’ PE and has to have an interior vena cava filter inserted. He continues to suffer from shortness of breath and can no longer work. Were the cardiologist and pulmonologist negligent? Why or why not? If they were, would the State be vicariously liable for their negligence?