The General Organization For
Teaching Hospitals and Institutes
Technical Affairs

الهيئة العامة للمعاهد
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EMERGENCY PROTOCOLS

2024

ID	Topic	Speciality
1	Acute Liver Failure (ALF)	Gastroenterology, Hepatology and Infectious Diseases.
2	Approach to acute upper gastrointestinal bleeding in adults	Gastroenterology, Hepatology and Infectious Diseases.
3	Approach to acute lower gastrointestinal bleeding in adults	Gastroenterology, Hepatology and Infectious Diseases.
4	Gastrointestinal Foreign Body Ingestion	Gastroenterology, Hepatology and Infectious Diseases.
5	Hepatic Encephalopathy	Gastroenterology, Hepatology and Infectious Diseases.
6	Fever	Gastroenterology, Hepatology and Infectious Diseases.
7	Heat Stroke and Heat Exhaustion	Gastroenterology, Hepatology and Infectious Diseases.
8	Drug-related Hyperthermias	Gastroenterology, Hepatology and Infectious Diseases.
9	The Septic Patient	Gastroenterology, Hepatology and Infectious Diseases.
10	Clinical Practice Protocol of Primary Care of Non-traumatic Acute Abdomen in Adults	General Surgery
11	Asthma	Pediatrics
12	Treatment of Acute asthma exacerbation in children	Pediatrics
13	Polytrauma Protocol	Orthpedics
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16	Acute Compartment Syndrome Clinical Pathway	Orthpedics
17	CRUSHED LOWER LIMB	Plastic surgery
18	Guidelines for management of crush injuries of the hand	Plastic Surgery
19	Postpartum Hemorrhage	gynaecology
20	ANTEPARTUM HEMORRHAGE	gynaecology
21	ENT Critical Condition Curriculum for residence	ear, nose, and throat
22	Epilepsy - Stroke Management	Neurology
23	Protocol of Management of Peripheral Nerve Injury	Neurosurgery
24	Angle Closure Glaucoma	Ophthalmology
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26	Extraocular foreign body removal	Ophthalmology
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ID	Topic	Speciality
28	Urinary Tract infection Management protocol. - Cystitis - Pyelonephritis - Prostatitis - Bacteriuria	Nephrology.
29	Plasmapheresis protocol	Nephrology.
30	Hyper kalemia management protocol	Nephrology.
31	Intravascular catheter infection	Nephrology.

Acute Liver Failure (ALF)

Bachground:

Acute Liver failure is an uncommon condition in which rapid deterioration of the liver function results in coagulopathy and alteration in the mental status of a previously healthy individual. Acute liver failure often affects young people and carries a very high mortality.

The term acute liver failure is used to describe the development of coagulopathy, usually with an international normalized ratio (INR) of greater than 1.5, and any degree of mental alteration (encephalopathy) in a patient without preexisting cirrhosis and with an illness of less than 26 weeks duration.

The outcome of acute liver failure is related to the etiology, the degree of encephalopathy, and related complications . Although mortality from FHF remains significantly high, improved intensive care measures and the use of orthotopic

liver transplantation have improved survival from less than 20% to approximately 60%

Etiology:

1-Viral hepatitis

Viral hepatitis may lead to hepatic failure. Hepatitis A and B account for most of these cases.

Hepatitis C: rarely causes acute liver failure.

Hepatitis D, as a coinfection or superinfection with hepatitis B virus, can lead to fulminant hepatic failure.

Hepatitis E (often observed in pregnant women) in endemic areas is an important cause of fulminant hepatic failure

Atypical causes of viral hepatitis and fulminant hepatic failure include the following:

Cytomegalovirus

Hemorrhagic fever viruses

Herpes simplex virus

Paramyxovirus

Epstein-Barr virus

Dengue virus

2-Autoimmunehepatitis

may also result in hepatic failure

3-Hepatic failure in pregnancy:

Acute fatty liver of pregnancy (AFLP) frequently culminates in fulminant hepatic failure. AFLP typically occurs in the third trimester; preeclampsia develops in approximately 50% of these patients. AFLP has been estimated to occur in 0.008% of pregnancies.

The most common cause of acute jaundice in pregnancy is acute viral hepatitis, and most of these patients do not develop fulminant hepatic failure. The one major exception to this is the pregnant patient whodevelops hepatitis E virus infection, in whom progression to fulminant hepatic failure is unfortunately common and often fatal.

4-Drug-related hepatotoxicity:

DILI is the primary cause of acute liver failure in adults

-Acetaminophen (also known as paracetamol and N-acetyl-p-aminophenol [APAP]) may lead to liver failure as a result of intentional or accidental overdose

-Prescription medications that have been associated with idiosyncratic hypersensitivity reactions include the following:

-Antibiotics (ampicillin-clavulanate, ciprofloxacin, doxycycline, erythromycin, isoniazid, nitrofurantoin, tetracycline)

-Antidepressants (amitriptyline, nortriptyline)

-Antiepileptics (phenytoin, valproa

-Anesthetic agents (halothane)

-Lipid-lowering medications (atorvastatin, lovastatin, simvastatin)

-Immunosuppressive agents (cyclophosphamide, methotrexate)

--Nonsteroidal anti-inflammatory agents (NSAIDs)

-Salicylates (ingestion of these agents may result in Reye syndrome)

-Others (disulfiram, flutamide, gold, propylthiouracil)

5-Vascular causes:

The following are vascular causes of hepatic failure:

-Ischemic hepatitis (consider especially in the setting of severe hypotension or recent hepatic tumor chemoembolization)

-Hepatic vein thrombosis (Budd-Chiari syndrome)--

-Hepatic veno-occlusive disease

-Portal vein thrombosis

-Hepatic arterial thrombosis (consider posttransplant)

6-Metabolic causes:

-Wilson disease-

-Alpha1-antitrypsin deficiency

-Galactosemia

-Tyrosinemia

-Reye syndrome

7- Malignancies:

-Primary liver tumor (usually hepatocellular carcinoma, rarely cholangiocarcinoma)

-Secondary tumor (extensive hepatic metastases or infiltration from adenocarcinoma, such as breast and lung, and melanoma primaries [common]; lymphoma; leukemia)

8-Miscellaneous:

Miscellaneous causes of hepatic failure include adult-onset Still disease, heatstroke, and primary graft nonfunction in liver transplant recipients

Signs and Symptoms:

Signs and symptoms of acute failure may include the following

- Encephalopathy

- Cerebral edema: May lead to signs of increased intracranial pressure (ICP) (eg, papilledema, hypertension, bradycardia)

- Jaundice: Often present but not always

- Ascites: Potential for hepatic vein thrombosis with rapid development in the presence of fulminant hepatic failure accompanied by abdominal pain

- Right upper quadrant tenderness: Variably present

-Change in liver span: May be small due to hepatic necrosis or may be enlarged due to heart failure, viral hepatitis, or Budd-Chiari syndrome

- Hematemesis or melena: Due to upper gastrointestinal (GI) bleeding

- Hypotension and tachycardia: Due to reduced systemic vascular resiatance.

Diagnosis:

The most important step in the assessment of patients with acute liver failure is to identify the cause, because certain conditions necessitate immediate and specific treatment and affect prognosis. All patients with clinical or laboratory evidence of moderate or severe acute hepatitis should have immediate measurement of prothrombin time (PT) and careful evaluation of the mental status. The presence of PT prolongation or mental status changes is grounds for hospital admission.

Lab testing:

--CBC: may reveal thrombocytopenia

--Coagulation studies: PTT, INR

-Liver function tests: often elevated levels of ALT,AST,ALK PH

-Serum bilirubin level: elevated

-Sserum ammonia level :may be dramatically elevated (arterial > Venous)

-Serum glucose level :may be dangerously low

-Serum arterial lactate level :often elevated

-Arterial blood gases :may reveal hypoxemia

-Serum creat.level : may be elevated

-Serum free copper, and ceruloplasmin levels: low levels in Wilson disease

-Serum phosphate level :maybe low

-Acetaminophen and acetaminophen-protein adducts level-

-Drug screening: consider in patience who are intravenous drug abusers

-Blood cultures: for suspected infections

-Viral serologies: Consider for Hepatitis A Immunoglobulin M (IgM), hepatitis B surface antigen (HBsAg), hepatitis B virus anticore IgM; hepatitis C viral load testing; hepatitis D virus

IgM if HBsAg is positive; in posttransplantation or immunosuppressed setting, consider studies for cytomegalovirus viremia, cytomegalovirus antigenemia, and herpes simplex virus

-Autoimmune markers (for autoimmune hepatitis diagnosis): Antinuclear antibody (ANA), anti-smooth muscle antibody (ASMA), and immunoglobulin levels

Other studies may include the following:

-Electroencephalography

-Intracranial pressure monitoring

-Percutaneous (contraindicated in the presence of coagulopathy) or transjugular liver biopsy

Imaging studies:

-Hepatic Doppler ultrasonography

-Abdominal computed tomography (CT) scanning or magnetic resonance imaging without contrast

-Cranial CT scanning

Management:

The most important aspect of treatment for acute liver failure is to provide good intensive care support, including the protection of the airways.

Specific therapy is also dependent on the cause of the patient's liver failure and the presence of any complications.

Pay careful attention to the patient's fluid management and hemodynamics. It is crucial to monitor their metabolic parameters, assess for infection, maintain nutrition, and promptly recognize GI bleeding.

Pharmacotherapy:

Various medications may be necessary because of the variety of complications that may occur from fulminant hepatic failure. In specific cases, antidotes that effectively bind or eliminate toxins are essential.

The following medications may be used in the management of acute liver failure:

.Antidotes (eg, penicillin G, silibinin, activated charcoal, N-acetylcysteine)

.Osmotic diuretics (eg, mannitol)

.Barbiturate agents (eg, pentobarbital, thiopental)

.Benzodiazepines (eg, midazolam)

.Anesthetic agents (eg, propofol)

Surgery:

Liver transplantation is the definitive treatment for acute liver failure. In selected patients for whom no allograft is immediately available, consider support with a bioartificial liver. This is a short-term measure that only leads to survival if the liver spontaneously recovers or is replaced

Nonbiologic extracorporeal liver support systems, such as hemodialysis, hemofiltration, charcoal hemoperfusion, plasmapheresis, and exchange transfusions permit temporary liver support until a suitable donor liver is found. However, no controlled study has shown long-term benefit.

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Approach to acute upper gastrointestinal bleeding in adults

INITIAL EVALUATION:

Past medical history:

-Prior episodes of upper GI bleeding.

-Comorbid conditions.

Drug history:

-NSAIDs.

-Anticoagulants.

General examination:

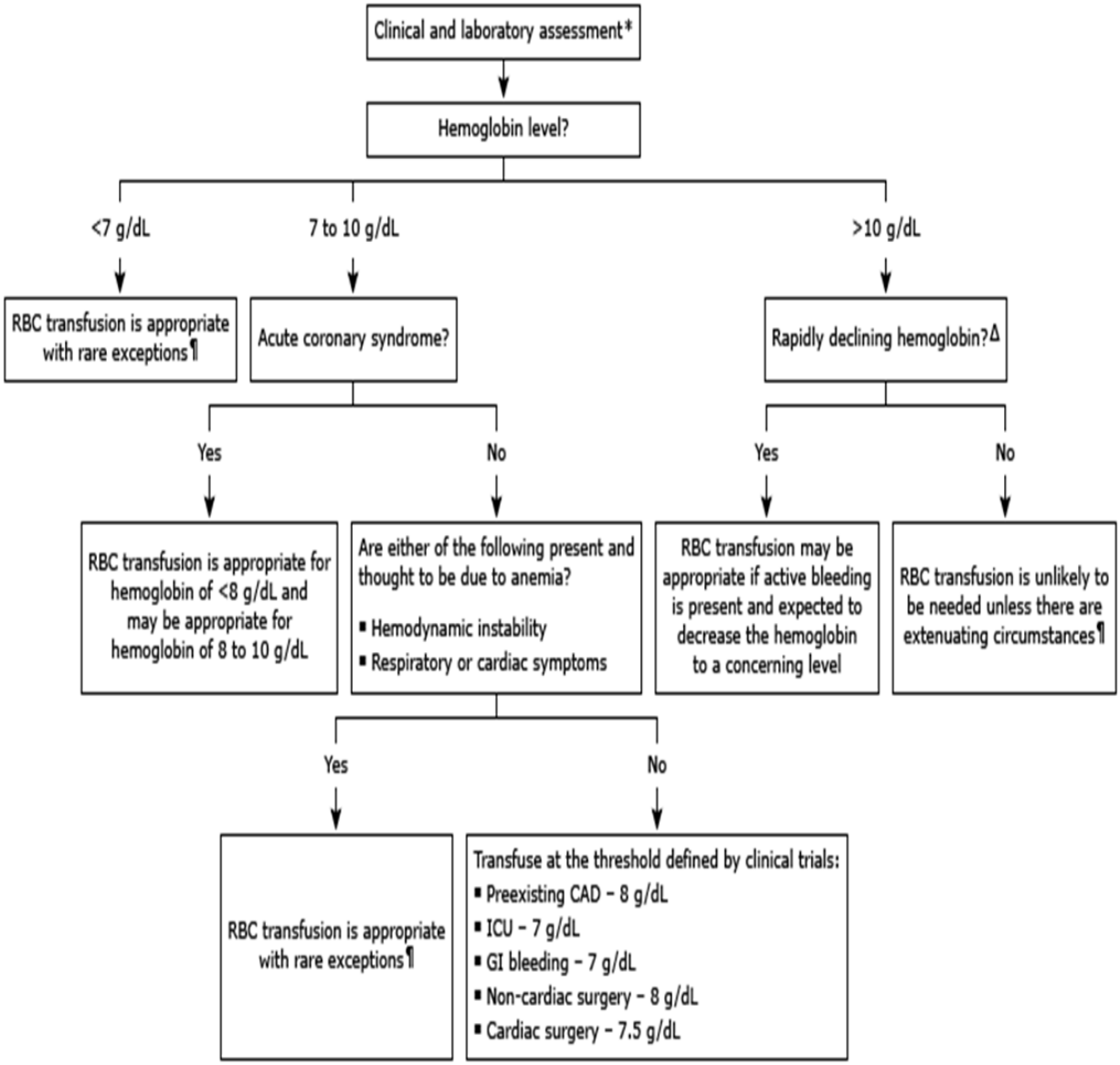
Laboratory data:

-Complete blood count, liver and kidneyfunction tests, coagulation studies (PT, INR).

-The hemoglobin level should initially be monitored every two to eight hours, depending upon the severity of the bleed.

-Nasogastric lavage:

-NGT lavage may be used when it is unclear if a patient has ongoing bleeding and thus might benefit from an early endoscopy.



General management:

-Closely monitor airway, clinical status, vital signs, cardiac rhythm, urine output, nasogastric output (if nasogastric tube in place).

-Do **NOT** give patient anything by mouth.

-Establish two large bore IV lines (16 gauge or larger).

-Provide supplemental oxygen (goal oxygen saturation ≥94% for patients without COPD).

-Treat hypotension initially with rapid, bolus infusions of isotonic crystalloid (eg, 500 to 1000 mL per bolus; use smaller boluses and lower total volumes for patients with compromised cardiac function).

- Blood transfusion:

- Platelet transfusion:Patients with critical or life-threatening bleeding and a low platelet count (<50,000/microL) should be transfused with platelets, an upper endoscopy can be performed if the platelet count is >20,000/microL, though if the patient is suspected to have active bleeding, we attempt to raise the platelet count to >50,000/microL prior to endoscopy.

- Managing anticoagulants, antiplatelet agents

Emergency reversal of anticoagulation from warfarin for life-threatening hemorrhage in adults:

A. If 4-factor prothrombin complex concentrate (4F PCC) is available:
   - Give 4F PCC 1500 to 2000 units IV over 10 minutes. Check INR 15 minutes after completion of the infusion. If INR is not ≤1.5, give additional 4F PCC.
   - Give vitamin K 10 mg IV over 10 to 20 minutes.

B. If 3-factor prothrombin complex concentrate (3F PCC) is available but 4F PCC is not available:
   - Give 3F PCC* 1500 to 2000 units IV over 10 minutes. Check INR 15 minutes after completion of the infusion. If INR is not ≤1.5, give additional 3F PCC.
   - Give Factor VIIa 20 mcg/kg IV **OR** give FFP 2 units IV by rapid infusion. Factor VIIa may be preferred if volume overload is a concern.
   - Give vitamin K 10 mg IV over 10 to 20 minutes.

C. If neither 3F PCC nor 4F PCC is available:

- . Give FFP 2 units IV by rapid infusion. Check INR 15 minutes after completion of infusion. If INR ≥1.5, administer 2 additional units of FFP IV rapid infusion. Repeat process until INR ≤1.5. May wish to administer loop diuretic between FFP infusions if volume overload is a concern.
- Give vitamin K 10 mg IV over 10 to 20 minutes.

Direct oral anticoagulant-associated bleeding reversal strategies:

Type of bleeding	**Agent**	**Possible interventions**
Life-threatening bleeding (eg, intracranial, retroperitoneal, compartment syndrome, massive gastrointestinal)	Dabigatran (Pradaxa)	-Idarucizumab. -Antifibrinolytic agent (eg, tranexamic acid, epsilon-aminocaproic acid) -Anticoagulant discontinuation -Oral activated charcoal (if last dose within prior two hours). -**Hemodialysis.** -RBC transfusions if needed for anemia. -Platelet transfusions if needed for thrombocytopenia or impaired platelet function (eg, due to aspirin). -Surgical/endoscopic intervention if appropriate.
	Rivaroxaban (Xarelto), Apixaban (Eliquis), Edoxaban (Lixiana)	-Antifibrinolytic agent (eg, tranexamic acid, epsilon-aminocaproic acid) -Anticoagulant discontinuation -Oral activated charcoal (if last dose recent enough). -RBC transfusions if needed for anemia -Platelet transfusions if needed for thrombocytopenia or impaired platelet function (eg, due to aspirin). -Surgical/endoscopic intervention if appropriate.
Minor bleeding	Dabigatran (Pradaxa)	-Local hemostatic measures -Possible anticoagulant discontinuation. ▪ Half-life (normal renal function): 12 to 17

(eg, epistaxis, uncomplicated soft tissue bleeding, minor [slow] gastrointestinal bleeding)		hours -Possible antifibrinolytic agent: (eg, tranexamic acid, epsilon-aminocaproic acid).
	Rivaroxaban (Xarelto), Apixaban (Eliquis), Edoxaban (Lixiana)	-Local hemostatic measures -Possible anticoagulant discontinuation. -Half-lives (normal renal function): ▪ Rivaroxaban 5 to 9 hours ▪ Apixaban 8 to 15 hours ▪ Edoxaban 6 to 11 hours -Possible antifibrinolytic agent (eg, tranexamic acid, epsilon-aminocaproic acid)

Medications:

-PPI: 80 mg bolus followed by infusion of 8 mg/h for 72 hours.

-Vasoactive medications: Octreotide,analogue of somatostatin,is given as an intravenous bolus of 50 mcg, followed by a continuous infusion at a rate of 50 mcg per hour for 2-5 days.

-Antibiotics for patients with cirrhosis, eg, ceftriaxone.

-Early endoscopy: Perform upper endoscopy within 24 hours for most patients with upper GI bleeding, but only after adequate resuscitation has been provided. For patients with suspected variceal bleeding, we perform endoscopy within 12 hours of presentation.

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Approach to acute lower gastrointestinal bleeding in adults

INITIAL EVALUATION:

Past medical history:

-Prior episodes of lower GI bleeding.
-Comorbid conditions.

Drug history:

-Anticoagulants.

General examination:

Laboratory data:

-Complete blood count, liver and kidney function tests, coagulation studies (PT, INR).

-The hemoglobin level should initially be monitored every two to twelve hours, depending upon the severity of the bleed.

Consider an upper GI bleeding source:

-10 to 15 % of patients with severe hematochezia will have an upper GI source. Suggested by hemodynamic instability, orthostatic hypotension, and an elevated blood urea nitrogen (BUN)-to-creatinine or urea-to-creatinine ratio (>20 to 30:1 or >100:1, respectively.

General management:

-Monitoring of vital signs, cardiac rhythm, urine output.

-Do not give patient anything by mouth if an urgent upper is needed.

-Establish two large bore IV lines (18 gauge or larger).

-Provide supplemental oxygen by nasal cannula.

-Fluid resuscitation: Patients with active bleeding should receive intravenous fluids (eg, 500 mL of normal saline or lactated Ringer's solution over 30 minutes).

-Blood transfusion

-Hemodynamically stable patients without comorbid illness may not require transfusion until the hemoglobin falls below 7 g/dL.

-Older patients and those who have severe comorbid illnesses, such as active coronary disease, require packed RBC transfusions to maintain a higher hemoglobin level (eg, 9 to 10 g/dL).

-Management of coagulopathies, anticoagulants, and antiplatelet agents:

-In patients with life-threatening bleeding and a coagulopathy (prolonged PT with INR greater than 1.5) warfarin and direct-acting anticoagulants (DOACs) should be withheld.

-Fresh frozen plasma (FFP) should be considered in patients on warfarin with life-threatening lower GI bleeding and an INR >2.5.

-Platelets should be transfused in patients with a low platelet count to maintain a platelet count of >30,000/micro L in patients with severe lower GI bleeding and >50,000/micro L in patients who require endoscopic management.

-In patients with life-threatening lower GI bleeding on DOACs, reversal agents may be needed for the small subset of patients that do not respond to initial resuscitation and cessation of the anticoagulant. Targeted reversalagent(idarucizumab for dabigatran and andexantalfa for apixaban and rivaroxaban).

-Platelet and plasma transfusions should also be considered in patients who receive massive RBC transfusions (>3 units of packed RBCs within one hour).

-In patients with an INR of 1.5 to 2.5, endoscopic hemostasis may be performed before or concomitant with the administration of reversal agents.

-In patients with an INR >2.5, reversal agents should generally be administered before endoscopy.

-Aspirin should be continued for secondary prophylaxis in patients with high-risk cardiovascular disease.

-Dual antiplatelet therapy should not be discontinued without cardiology consultation in patients with an acute coronary syndrome within the past 90 days or with a bare-metal stent placed within the preceding six weeks or drug-eluting stents within the preceding six months.

-If low-dose aspirin or antiplatelet agents are withheld, they should be resumed, preferably within five days, or earlier if hemostasis has been achieved or there is no further evidence of bleeding.

-Colonoscopy: no difference in identification of stigmata of recent hemorrhage, rebleeding, or blood transfusions in patients undergoing colonoscopy in less than 24 hours of presentation versus within 24 to 96 hours.

-Bowel preparation: four to six liters of polyethylene glycol over three to four hours until the rectal effluent is clear.

-CT angiography: In patients with severe bleeding who cannot be stabilized for colonoscopy or with severe ongoing bleeding despite colonoscopy.

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Gastrointestinal Foreign Body Ingestion

Introduction:

Foreign body (FB) and food impaction are one of the most common gastrointestinal complaints seen in the emergency department.Encountered in both the adult and pediatric populations, foreign bodies tend to pass spontaneously without intervention in most instances (80 to 90%).

In the setting of a FB, the role of the healthcare worker is to identify those patients that have a high risk for complications, and that will require prompt intervention.

Objectives:

- Review the appropriate evaluation of gastrointestinal foreign bodies.
- Outline the management options available for gastrointestinal foreign bodies.
- Summarize inter-professional team strategies for improving care coordination and communication to improve the outcomes of patients with a gastrointestinal foreign body.

Etiology:

The majority of foreign body ingestions are unwitnessed and resolve without ever involving a healthcare professional.

Adults usually are symptomatic at the time of presentation to the clinic or the emergency department for a foreign body.

Children presenting with foreign bodies usually do so because the event was witnessed by a parent or family member.

The most common complication seen is impaction, which is most frequently within the esophagus.

There are three sections of the esophagus where foreign bodies are at higher risk of becoming impacted due to narrowing.

1. The First, and most commonly, is at the thoracic inlet where the crico-pharyngeus muscle is located.
2. The second section is at the aortic arch,
3. The third section is at the gastro-esophageal junction.

Objects impacted in these locations can be seen on X-rays, at the level of clavicles, carina, and just above the gastric bubble, respectively.

Pathophysiology:

The foreign body, when ingested, can lodge in the GI tract resulting in complete or partial obstruction. Furthermore, it can erode the GI mucosal wall and lead to migration and perforation.

History:

- Children with foreign body ingestion can present with:
  1. Commonly patients are asymptomatic
  2. Sore throat.
  3. Chest discomfort.
  4. Globus sensation.
  5. Symptoms of complications. Dysphagia or odynophagia.
- Adults tend to present with food impactions more commonly than foreign body ingestion.

The physical exam in the majority of these patients does not offer any additional information since patients are usually asymptomatic or with minimal symptoms.

Differential diagnosis:

A patient presenting with a witnessed foreign body ingestion or Globus sensation may seem straightforward; however, other pathologies should be considered as well. Some of these other conditions are the following:

1. Esophagitis
2. Pharyngitis

3. Laryngitis
4. Peri-tonsillar or retropharyngeal abscess
5. GERD
6. Acute coronary syndrome in adults
7. Gastritis
8. Gastroparesis
9. Pyloric stenosis

Evaluation:

1. It is essential first to evaluate the patient's airway and breathing.
2. Radiography is the first-line modality for evaluating patients with a possible foreign body. 83% of ingested foreign bodies are radiopaque. However, it is essential to note that smaller objects might not be visible in thicker body parts. Frontal and lateral neck and chest X-rays, as well as an abdominal X-ray, should be obtained.
3. Radiolucent foreign bodies will be seen as edges or irregularities on the radiograph. X-rays show one-third of foreign bodies.
4. If patients present with suspected acute esophageal obstruction, imaging is not necessary to localize and should not delay urgent endoscopy.

Management:

When a foreign body is identified and localized in the GI tract, the management plan can then be decided upon.

1. Once an object that is not high-risk reaches the stomach, it will likely continue through the GI tract and be expelled without any intervention. These patients can be discharged with parental instructions to inspect the stool for the object.
2. A button battery, in an asymptomatic patient that has already passed into the stomach, can also be managed from home. Parents should be instructed to watch for signs of obstruction or GI injury. The patient should maintain a regular diet, participate in physical activity, and their stool should be inspected for the battery. If the battery doesn't pass within 10 to 14 days, repeat imaging may be required.
3. If the battery is within the esophagus at the time of presentation, prompt endoscopy is required since damage can occur as early as 2 hours of ingestion.

4. Ingestion of an individual magnet can usually be managed at home similarly as described above.
5. For adults presenting with possible food impaction or esophageal FBs should be removed as quickly as possible endoscopy is required.
6. Patients may be required to go to the OR if they are presenting with signs of obstruction, peritonitis, or perforation.

Treatment planning:

Foreign bodies that seem to be dangerous because of their shape and size should be effectively removed. There are many options available, including:

1. Endoscopy
2. Laparoscopy
3. Laparo-endoscopic removal of foreign bodies.

Prognosis:

Most foreign bodies pass spontaneously without any intervention, and only 1% can cause perforation. A high risk of perforation is present when the foreign body has sharp ends. The common sites of perforation are at points of narrowing in the GI tract.

Early diagnosis and immediate treatment are essential to improve the prognosis of foreign bodies lodging in unusual locations. Up to a 10% mortality rate has been reported because of missed or delayed diagnosis.

Complications:

Complications and their severity are usually related to the object ingested, its location, and the amount of time that has passed since the ingestion.

- Bleeding and perforation.
- Button batteries, which are not chemically inert, impacted in the esophagus, can cause severe tissue damage and burns caused by the build-up of sodium hydroxide.
- Fistulization into major blood vessels can occur.
- Ingestion of magnets can lead to obstruction, volvulus, and fistula formation.
- Tissue necrosis and perforation can occur, leading to peritonitis.
- Deep pressure ulcerations can occur within the first 8-24 hours following ingestion.

Consultations:

The following consultations are required in cases of GI foreign bodies:

- Surgery
- Gastroenterology
- Radiology
- Nutrition
- Otolaryngology
- Psychiatry
- Pediatric

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Hepatic Encephalopathy

Definition

Hepatic encephalopathy (HE) describes a broad range of neuropsychiatric abnormalities caused by advanced hepatic insufficiency or portosystemic

Grading Westhaven criteria

Grade 1
- Trivial lack of awareness
- Euphoria or anxiety
- Shortened attention span
- Impaired performance of addition

Grade 2
- Lethargy or apathy
- Minimal disorientation for time or place
- Subtle personality change
- Inappropriate behavior
- Impaired performance of subtraction

Grade 3
- Somnolence to semi-stupor, but responsive to verbal stimuli
  Confusion
- Gross disorientation

Add Glasgow coma scale.

Grade 4
- Coma (unresponsive to verbal or noxious stimu

Add Glasgow coma scale.

Incidence & pathophysiology

Overt HE occurs in **30% to 45%** of patients **with cirrhosis**, withan **incidence of20% per year**, and it reaches as high as **50% after TIPS**. Although its exact pathophysiology remains controversial, evidence has established a combined role of hyperammonemia and systemic inflammation.

Classification of HE

According to the underlying disease. Type A in patients with acute liver failure, **Type B** in thosewith portosystemic shuntand **Type C** in those with cirrhosis.

- **Covert** (minor or no signs/symptoms but abnormalitieson neuropsychological and/or neurophysiological tests) or**Overt** (grades II or over according to the West Haven criteria).

Overt HE is classified as

Recurrent if -->2 bouts occur within 6 months

Persistent if -->the patient does not return to her/his baseline performance between bouts.

Precipitating factors

- Gastrointestinal (GI) bleeding—increases the protein in the bowel and rapidly precipitates hepatic encephalopathy;
- Constipation.
- Metabolic alkalosis.
- Potassium deficiency induced by diuretics.
- Opioids, hypnotics, and sedatives.
- Medications containing ammonium or amino compounds.
- Paracentesis with consequent hypovolemia.
- Hepatic or systemic infection.
- Portosystemic shunts (including transjugular intrahepatic portosystemic shunts).

Diagnosis

General principles

- Diagnosis of hepatic encephalopathy is largely clinical and based on ruling out alternate explanations for altered mental status.
- It is critical to identify the precipitating factor (e.g., GI bleed).
- The diagnosis of covert hepatic encephalopathy requires psychometric testing, which is

usually carried out by a specialist.
- Minimal HE requires specialised psychometric or neurophysiological testing.
- Overt HE is diagnosed clinically utilizing the West-Haven criteria.
- Ammonia levels: if normal, unlikely to be HE.
-Serum ammonia levels are usually elevated in hepatic encephalopathy. However, elevated levels are not diagnostic and the magnitude of elevation does not correlate with the degree ofencephalopathy.

Differential Diagnosis of HE

- Intracranial bleeding and stroke.
- Brain lesions (traumatic, neoplasms, normal pressure hydrocephalus).
- Metabolic: uraemia, hypercapnia, inherited metabolic disorders (especially urea cycle defects)
- Diabetic (hypoglycaemia, ketoacidosis, hyperosmolar, lactate acidosis).
- Alcohol (intoxication, withdrawal, Gayet-Wernicke's encephalopathy).
- Drugs (benzodiazepines, neuroleptics, opioids).
- CNS infections.
- Electrolyte disorders (hyponatremia and hypercalcemia).
- Nonconvulsive epilepsy.
- Psychiatric disorders.
- Severe medical stress (organ failure and inflammation),

Investigations

Laboratory studies:

- CBC, liver/kidney function, electrolytes.
- Ammonia, TSH, CRP, glucose level.
- urine analysis, culture & blood culture .

- alcohol level, drugs screening.
- Lumbar puncture, EEG.

Imaging studies :

- Chest X-ray to rule out infection.
- Abdominal US to assess for ascites and PVT.
- No cerebral imaging prove the diagnosis of HE.
- CT or MRI brain should be performed if suspecting central cause or no response to treatment.

Treatment

Precipitating factors should be sought and managed.

- In patients with covert HE, anti-HE treatment should be considered for the purposes of differential diagnosis and to prevent overt HE.
- Reduce nitrogenous load in the gut.
- Due to the unpredictable clinical course and risk of aspiration of patients with overt HE grades 3and 4, treatment should occur in the ICU.
- Nonabsorbable disaccharides (lactulose, lactitol) decreasing the absorption of ammonia in the bowel. Initial dose is 30 mL three or four times daily orally, titrateso that the patient produces two or three bowel movement per day.
- Rifaximinoral antimicrobial, broad-spectrum, nonabsorbable (550mg twice daily).
- Routine zinc supplementation is not recommended due to the conflicting evidence of zinc supplementation in HE patients.
- In patients with HE, demonstrated or suspected vitamin/ micronutrient deficiencies should be treated, as they can compound HE.
- In patients with recurrent/persistent HE, replacement of animal protein with vegetable and dairy protein can be considered, provided that overall protein intake is not compromised and that patient's tolerance is considered.
- Patients should be provided with information about driving safety, including potential risks with vehicle driving and overt HE episodes.
- Patients scheduled for non-urgent TIPS should be thoroughly assessed for the presence or history of HE.
- Patients with end-stage liver disease and recurrent or persistent HE not responding to other treatments should be assessed for liver transplantation.

Secondary prophylaxis

Initiate secondary prophylaxis against hepatic encephalopathy after an episode of overt HE.

- Lactulose is recommended as secondary prophylaxis and should be titrated to obtain 2-3 bowel movements per day.
- Rifaximin as an adjunct to lactulose is recommended as secondary prophylaxis following>−1 additional episodes of overt HE within 6 months of the first one.

Summary Points

- Overt hepatic encephalopathy consists of neurological and psychiatric abnormalities that can be detected by bedside clinical tests, whereas minimal hepatic encephalopathy can only be distinguished by specific psychometric tests.
- There are many grading scales available for hepatic encephalopathy, including the long-standing West Haven Criteria, which is the most commonly used system.
- Diagnosis of overt hepatic encephalopathy requires the exclusion of alternate causes of altered mental status. Serum ammonia levels should not be used as a diagnostic tool or as a means of monitoring response to treatment.
- Treatment of acute overt hepatic encephalopathy should include (1) supportive care, (2) identifying and treating any precipitating factors, (3) reduction of nitrogenous load in the gut, and (4) assessment of need for long-term therapy and liver transplant evaluation.
- Lactulose should be used as initial drug therapy for the treatment of acute hepatic encephalopathy.
- Rifaximin can be added for those individuals who do not have an adequate response to lactulose.
- Prevention of recurrent hepatic encephalopathy or treatment of persistent hepatic encephalopathy includes drug therapy as well as prevention or avoidance of precipitating factors, including potentially sedating medications.
- Protein restriction should be avoided as a general rule, as it can actually lead to worsening of hepatic encephalopathy. Persons with cirrhosis are advised to consume 1.2 to 1.5 g/kg protein daily.
- Liver transplant evaluation should be considered in appropriate candidates once a diagnosis of overt hepatic encephalopathy is made. Liver transplantation is indicated in persons with liver failure and recurrent intractable overt hepatic encephalopathy.

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Fever

Introduction

There are different definitions regarding fever (pyrexia) and hyperthermia. These involve the distinction between increased temperature due to the body's thermoregulatory Centre increasing the set level required (for a variety of reasons) and increased temperature due to the body's thermoregulatory centre losing control (again due to a number of possible causes).
For the purposes of this algorithm we will ignore these distinctions and consider patients presenting with a markedly increased temperature as one group. Body temperature ≥40°C is generally considered to be dangerous and is the cut- off used for the purposes of this algorithm.

Signs and symptoms

Possible signs and symptoms of markedly elevated temperature include:
Weakness or fatigue, headache and/or impaired judgement, thirst, nausea and/or vomiting, dizziness/stumbling gait, anxiety/agitation or hysteria, tachypnoea and tachycardia, paraesthesia, myalgia, cramps, or tetany, diarrhea, excessive salivation and swallowing dysfunction, collapse, seizures, or coma Any of these can occur at any time and in any combination.

Elements suggestive of a thyroid storm

History of thyroid dysfunction or symptoms of hyperthyroidism, tachycardia and palpitations, chest pain or shortness of breath, anxiety and irritability or disorientation, heart failure, increased sweating or high temperature, weakness.

Evaporative cooling

Strip patient to underwear, sponge or spray the patient with cool water, and apply fans to encourage evaporation.

Malignant hyperthermia

This is a particular presentation with markedly increased temperature (among other signs). Here patients with the genetic predisposition when exposed to suitable triggers, e.g. volatile anaesthetic agents or succinylcholine, can develop an uncontrolled and extreme increase in skeletal muscle oxidative metabolism. This usually occurs when patients receive anaesthetics for surgical procedures within the theatre area; however, there is a possibility that itmay occur in the Emergency Department if a patient receives an RSI for the first time. The AAGBI has produced a safety guideline for use when malignant hyperthermia occurs.

Neuroleptic malignant syndrome

This is another particular type of presentation where, along with increased temperature, muscle rigidity and autonomic hyperactivity predominate. This is caused by various antipsychotics (including haloperidol, olanzapine, and
risperidone) and some antiemetics (including droperidol, metoclopramide, and prochlorperazine). These lists are by no means exhaustive; it is vital that any history of the patient having taken antipsychotics or antiemetics is sought.

Serotonin syndrome

A very similar picture is seen with serotonin syndrome; this is due to an accumulation of serotonin, which can be caused by a wide variety of drugs including SSRIs, serotonin- norepinephine reuptake inhibitors, tricyclic antidepressants, monoamine oxidase inhibitors, some migraine medications, antiemetics and analgesics, illegal substances (lysergic acid diethylamide (LSD), 3,4-methyl enedioxy- methamphetamine (MDMA, commonly known as ecstasy),
cocaine, amphetamines), and even some herbal remedies. Again this list is not exhaustive but emphasizes the need for a very careful and thorough drug history.

Algorithm for fever

Patient presents with temperature≥40°C

Full set of observations including Glasgow Coma Scale & core temperature

Does patient ful•l two or more Systemic In•ammatory Response Syndrome criteria with suspicion of infection?

- NO
- Yes → Follow sepsis

Give antipyretics (paracetamol or ibuprofen (if not contraindicated)) and if core temperature >40°Cinstitute evaporative cooling ,& apply ice packs - Take thorough history and perform full examination, paying particular attention to signs andsymptoms listed in the text and prescribed, over-the-counter and illegal drug history

Has patient just received anaesthetic drugs (volatile agents or succinylcholine)?

- NO
- Yes

Ensure appropriate investigations completed including FullBlood Count, clotting, Urea & electrolytes, Magnesium, boneprofile, lactate, Liver function tests Creatinine kinase, glucose,cross match, Arterial blood gases and urine analysis

Activate Association ofAnesthetists of Great Britainand Ireland (AAGBI) malignanthyperthermia safety guideline

Are there signs/symptoms/history to suggest athyroid storm ?

- Yes → Discuss with endocrinologist/senior medic regardingfurther management andadmit. Management will, depend on cause of thyroidtoxicity but beta-blockers may be useful I
- No → Is patient GlasgowComa Scale ≥13?
  - Yes
  - No → Is Glasgow Coma Scale≤8, i.e, airway protection required?
    - No → If safe to do so supply patient with cold fluids to drink; if any problems with hypersalivation or swallowing problems provide cooled intravenous fluids (eg Normal saline). If patient requires sedation use benzodiazepines
      - Has temperature reduced and patient's condition improved?
        - Yes → Continue to monitor and provide cold intravenous fluids, being wary of • fluid overload
        - No → Discuss with anaesthetic colleagues regarding need for intubation and ventilation and further drug treatment including Dantrolene
    - Yes → Organize immediate intubation& ventilation of patient

Treat with cooled intravenous fluids and request urgent anaesthetic review. Consider alkalization of urine. Discuss with senior emergency physicians & anaesthetic colleagues regarding use of drugs such as Dantrolene

Heat Stroke and Heat Exhaustion

There is a spectrum of heat- related disease, from rashes and cramps through to heat stroke. Heat exhaustion ischaracterized by non- specific symptoms such as malaise, headache, and nausea and may progress to heat strokewhich involves the central nervous system (CNS) and results in altered conscious state.

Heat stroke is defined as a core temperature of >40.6°C due to environmental exposure and lack of thermoregulation.This differentiates it from fever and has a significant mortality associated with it. It can be classified asexertional or non- exertional.

Exertional heat stroke (EHS) results from strenuous activity in hot conditions. Non- exertional heat stroke(NEHS) describes loss of thermoregulation resulting in overheating and particularly affects infants, children, and theelderly. CNS disorder, medications, and endocrine abnormalities (e.g. thyrotoxicosis) may all contribute to NEHS.

Clinical presentation:-

● Patients may present with a hyperdynamic state, cramps, tachycardia, CNS disturbance including seizures andcoma, and coagulopathy sometimes with GI bleeding

● Blood investigations should include FBC, U&E, liver function, Mg, Ca, phosphate, lactate, creatine kinase (CK),arterial gas, coagulation screen, thyroid function, and glucose

● A CT head scan may be required to exclude intracerebral bleeding

● Sinus tachycardia and non- specific ST and T wave changes may be seen on ECG

● Aggressive reduction of temperature to 39°C can be life- saving

● Benzodiazepines may be required for fits or shivering, but antipyretics have no role in the management of heatstroke.

Cooling methods

● Immersion in a bath of iced water is rapidly effective but largely impractical

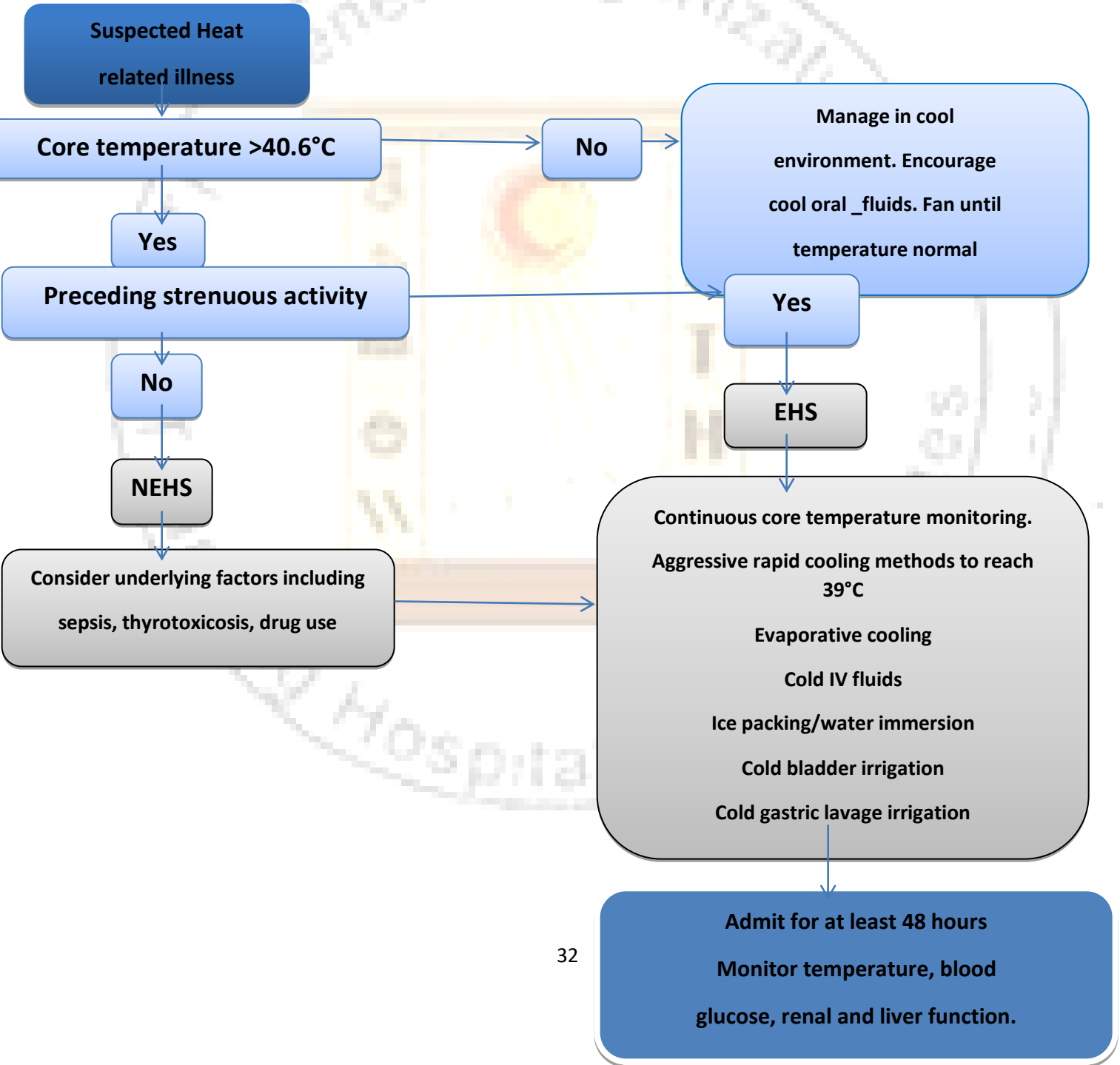
● Evaporative cooling involves removing the patient's clothes and spraying with a fine mist of water at 15°C,followed by fanning

● Ice packing involves placing ice packs in the groin and axillae, and even covering the whole trunk

● Gastric lavage requires intubation with a cuffed ET tube prior to instilling 10 mL/ kg iced water via an NG tube,and suctioning out after 1 minute

- Cardiac bypass or haemodialysis may be considered in refractory cases
- Care should be taken with IV fluids to avoid pulmonary oedema

Algorithm for heat stroke and heat exhaustion



Drug-related Hyperthermias

There are three defined drug- related conditions that include hyperthermia as a feature: malignant hyperthermia,neuroleptic malignant syndrome, and serotonin syndrome. Hyperpyrexia related to stimulant drug use includingamphetamines is also increasingly seen and is similar to serotonin syndrome.

Malignant hyperthermia

This is an inherited disorder resulting in hypermetabolism in skeletal muscle triggered by certain drugs (typically inhaled anaesthetic agents or suxamethonium). Acidosis, rigidity, and hyperkalaemia will be present, and a rapid risein end tidal carbon dioxide in anaesthetized patients is often the first sign of development. Anaesthetic managementguidelines should be followed and dantrolene may be life- saving.

Neuroleptic malignant syndrome

This refers to the combination of hyperthermia, rigidity, and autonomic dysfunction associated with antipsychoticmediation. Bradykinesia or akinesia is typical and delirium is common. The onset may be over a number of days.

Raised CK and/ or WBC count is also commonly found.

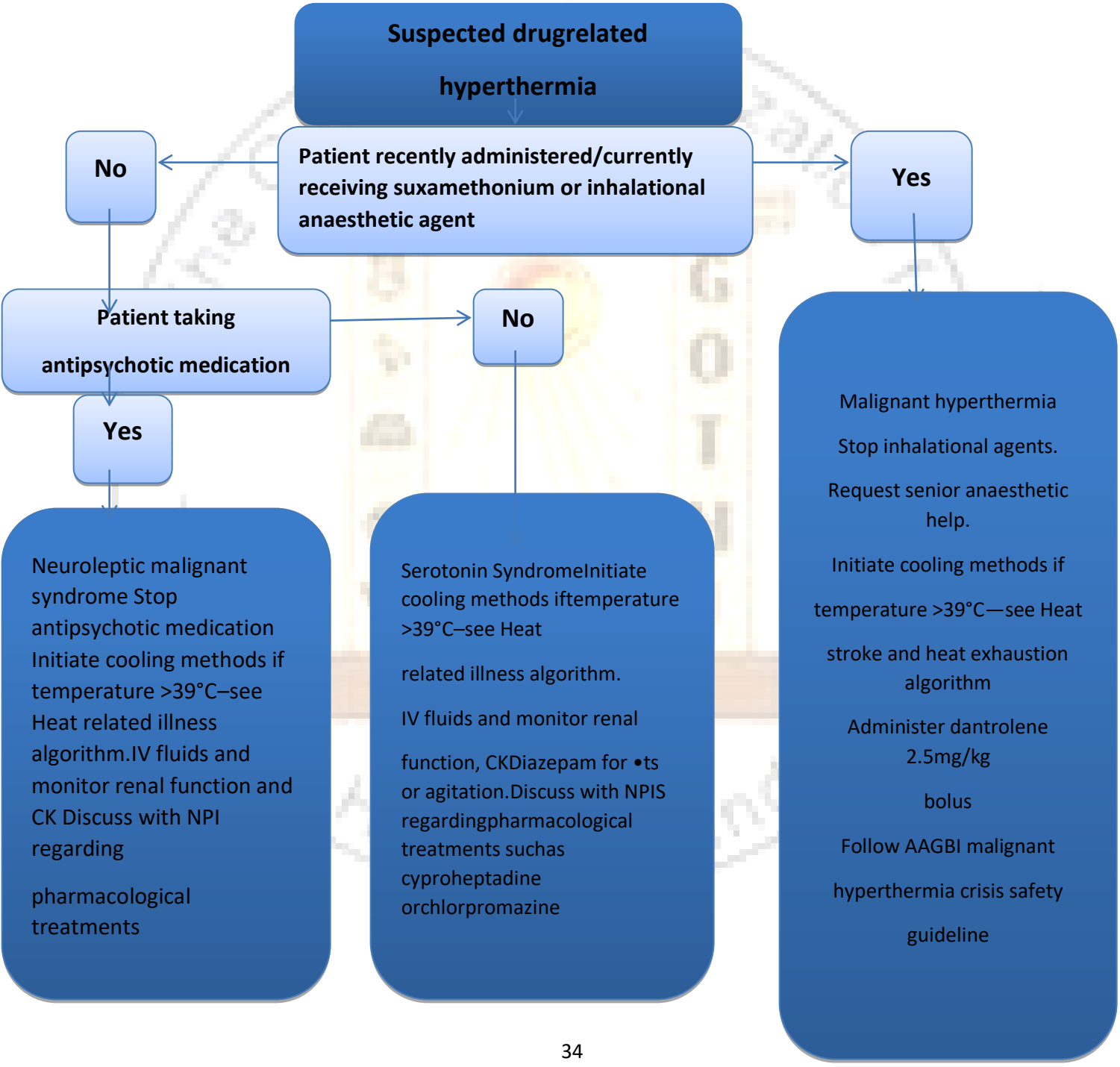
Serotonin syndrome

This has some similar features to neuroleptic malignant syndrome, including altered mental status, autonomicdysfunction, and neuromuscular abnormalities, but may be distinguished by tremor, hyperreflexia, and more rapidonset over a period of hours. Selective serotonin reuptake inhibitor (SSRI) medications are the typical cause.

Blood investigations should include FBC, U&E, liver function, Mg, Ca, lactate, CK, arterial gas, coagulation screen,and glucose. Rhabdomyolysis may be seen with all causes and monitoring of CK and renal function is essential.

The role of pharmacological agents such as dantrolene or bromocriptine is unproven; however, as with anytoxicological presentation, advice can be sought from the NPIS. If indicated, the initial dose of dantrolene is 1 mg/kg given intravenously.

Algorithm for drug-related hyperthermias



The Septic Patient

Introduction

Sepsis is a complex syndrome presenting with systemic manifestations of infection and can be defined as life-threateningorgan dysfunction caused by a dysregulated host response to infection.

Identification

It is identified using a compilation of various signs, symptoms, and investigations, although no definitive diagnostictest currently exists.

There is increasing acknowledgment that previous definitions used in this field, such as systemic inflammatory response syndrome (SIRS) and severe sepsis, are unhelpful: SIRS is neither sensitive nor specific for infectionfor sepsis, and sepsis itself carries a high enough mortality and morbidity to be labelled as a ‘severe’ condition.

The algorithm identifies a place for the SIRS criteria, as this is still used in many hospitals around the world toidentify sepsis; however, there is newer evidence suggesting that the Sequential (sepsis- related) Organ FailureAssessment (SOFA) score has a higher accuracy in identifying early those patients with a higher morbidity andmortality and thereby needing more urgent and thorough medical input. The SOFA score examines six areasof physiology (respiratory, coagulation, liver, cardiovascular, CNS, and renal) and the presence of abnormalities.

A shorter scoring system— Quick SOFA (qSOFA)— looks at three aspects of physiology and can bemeasured rapidly and easily at the bedside.

Septic shock

Septic shock (previously known as ‘severe sepsis’) is a state of acute circulatory failure associated with a significantlyhigher mortality. Its definition is not exact; however, two tests point towards this: a lactate >2 despite adequatefluid resuscitation and the need for vasopressor support to maintain a mean arterial pressure (MAP) >65 mmHg.

Including the initial investigations, further investigations may be needed, depending on the clinical picture,including radiological (CXR, abdominal US, CT) and microbiological (urine dip/ mid-stream urine (MSU)/ catheterspecimen urine (CSU), sputum and wound samples, diagnostic pleural or ascetic or joint aspirations).

SIRS criteria:-

SIRS criteria	(≥2 = SIRS)
Temperature >38°C or <36°C	1 point
Respiratory rate >20	1 point
Acutely altered mental state	1 point
Blood glucose >7.7 mmol/ L in absence of diabetes	1 point
WBC count >□2 × □09/ L or <4 × □09/ L	1 point
Heart rate >90/ minute	1 point

QSOFA score:-

qSOFA score	1 point
Respiratory rate ≥22/ min	1 point
Altered mentation (GCS <□5)	1 point
Systolic BP <100 mmHg	1 point

Algorithm for the septic patient

INFECTION SUSPECTED OR PROVEN?

1- Full set of observations including BM
2. Calculate qSOFA score
3. Examine for SIRS
4. Call for help.

IF qSOfA≥2 or SIRS≥2 PERFORM SEPSIS 6

GIVE OXYGEN	IV ANTIBIOTICS	TAKE BLOODS	MEASURE	IV FLUID	FLUID
Target O2 sats: • 96–98% (88–92% if at risk of hypercapnia)	Follow local trust guidelines	Blood cultures, FBC, U&E, CRP,LFT, clotting, glucoee, ESR, VBG, or ABG	Aim trepeat After adequate Fluidresuscitation	0.9% saline 500ml stat. Then reassess. May need up to 2–3 L	Consider catheter OR strict input/ output measurements

FIND SOURCE OF SEPSIS

- ENT/maxillofacial
- Abdominal/GI
- Foreign body/implanted device
- tract/Perineal/Genital
- Recent operation/trauma
- Endocarditis
- Biliary tract
- Skin or wound

CNS
Intravascular catheter
Female reproductive

Pneumonia
Urinary tract
Bone or joint infection

Consider other investigations

Continual reassessment is vital.
Consider further fluid boluses up to 30ml/kg
Urgent referral needed to senior in the relevant specialty

Ifdespite adequate fluid resuscitation: Mean arterial pressure <65 AND – Lactate ≥2 , the patient is in SEPTIC SHOCK
Consider discussion with Critical care, microbiology, senior registrar
May need vasopressor support

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Clinical Practice Protocol of Primary Care of Non-traumatic Acute Abdomen in Adults

Purpose

The purpose of thisprotocol is to provide practical medical guidelines to clinicians for improving the quality and efficacy of treatment and advancing the prognosis and quality of life of patients with acute abdomen. However, the subjects of thisprotocol are limited to adults with nontraumatic acute abdomen.

Thisprotocolis designed for all clinicians who treat acute abdomen, ranging from general clinicians to the ones specializing in acute abdomen.

Definition

Acute abdomen refers to intra-abdominal pathology, including extra-abdominal, thoracic and systematic pathology, withan onset of less than one week that may require urgent interventions, such as surgery.

<Comment> Abdominal disease with acute onset of abdominal pain requiring immediate intervention including emergency surgery is termed acute abdomen

Epidemiology

No exact numbers are available, but between 7% and 10% of emergency department visits are for abdominal pain,15% of all emergency hospitalization is due to acute abdominal pain, and about 40% is due to surgical causes. About one-third of abdominal pain patients are diagnosed with non-specific abdominal pain. Another 30% have acute renal colic.However, the most common cause of surgical acute abdomen is acute appendicitis.

Prognostic factors:

*Poor prognostic factors (with high mortality and morbidity rates):

- Cardiovascular diseases (myocardial infarction, mesenteric arterial occlusion, non-obstructive mesenterium ischemia, and aortic aneurysmrupture);

- Poor overall status and abnormal vital signs (e.g.,perforative large

bowel peritonitisand intestinal necrosis due to ileal strangulation);

- Generalized peritonitis (postoperative30-day and in-hospital mortality

rates are about 8.8% and 14.1%, respectively).

- Presence of comorbidities;
- Advanced age;
- Steroid use;
-Respiratory dysfunction;
-High APACHE II, SOFA, POSSUM, or E-PASS scores;
- ASA classification class ≥ 3;
-Organ failure; &
- Recent invasive surgery.

History-Taking:

*The following should be included in history-taking of acute abdomen:

- Abdominal pain(onset,course, duration, character,severity, location, radiation/referral, palliatives& provocative.
- Associatedsymptoms (e.g., nausea, anorexia, vomiting, hematemesis, hematochezia, diarrhea, constipation, weight loss,dysuria, hematuria, vaginal dischargeetc.)
- Age, for complaints of pain are often obscure in elderly patients and also for age-related illnesses.
- Sex, for sex-specific diseases such as testicular torsion in males and ruptured ovarian cyst in females).

- Allergies,
- Medications history [In particular, the type and quantity of NSAIDs(for possible pepticulcers), antimicrobial agents(for possible pseudomembranous enterocolitis)andsteroid therapies (commonly mask pain)] should be determined.
- Comorbidities (e.g., diabetes, hypertension, cardiopulmonary disease etc.)

- Detailed surgical history (even aminor abdominal surgerymay cause adhesions and astrangulated bowel obstruction or an abdominal incisional hernia),

- History of abdominal disease (a history of ureteral calculi, cholecystolithiasis, gastroduodenal ulcer, or whether thepatient had a similar pain previously,since there are many recurring cases).

- Diet (e.g., fatty diet and high-fiber diet for gallstones and diverticular disease respectively),

-Menstrual history in females (for differentiation of gynecological causes of acute abdomen, suchas ovarian hemorrhage, endometriosis, or painful functional menses),

- Pregnancy in women of reproductive age (for differentiation ofpregnancy-related causes of acute abdomen, such as extrauterine pregnancy or miscarriage.

<Comment> In patients with intense pain and unstable vitalsigns, sufficient time may not be available for careful takingof complete history. In these cases, a brief medicalhistory should be taken to differentiate cases requiring emergencytreatment. The following acronyms have been proposed as a method of obtaining useful informationin a timelymanner:

- "**SAMPLE**":[**S** (signs &symptoms),**A** (allergies), **M** (medications), **P** (relevant past medical history),**L** (last oral intake) and **E**(events leading up to the present illness)]

- "**OPQRST**": [**O** (onset), **P** (palliative/provocative), **Q** (quality/quantity), **R** (region/radiation), **S** (associated symptom), **T**(time course)].

-The most common intra-abdominal causesof nontraumatic acute abdomen in adults include (but not limited to)(Table 1):

- Acute appendicitis,
- Cholelithiasis,
- Small intestinal obstruction,
- Ureteral stone,
- Gastritis,
- Peptic ulcer perforation,
- Gastroenteritis,
- Acute pancreatitis,
-Diverticulitis,
-Obstetrics and gynecological diseases in women (e.g., ruptured Graafian follicles,pelvic inflammatory disease(PID), tubo-ovarian abscess, ovarian torsion, ovarian hemorrhage,& fibroid degeneration)

Furthermore, intra-abdominal causesshould be differentiatedfromextra-abdominal, thoracic and systematic pathology (e.g., myocardial infarction, torsion of the spermatic cord,and other systemic diseases).

Table 1:Common intraabdominal causes of acute abdomen in adults

Causes	Onset	Location	Charateristics	Intensity
Appendicitis	Gradual	Periumbilical early; RLQ late	Diffuse early, localized late	++
Cholecystitis	Acute	RUQ	Localized	++
Pancreatitis	Acute	Epigastric, back	Localized	++ to +++
Diverticulitis	Gradual	LLQ	Localized	
Perforated peptic ulcer	Sudden	Epigastric	Localized early, diffuse late	+++
Small bowel obstruction	Gradual	Periumbilical	Diffuse	++
Ruptured abdominal aortic aneurysm	Sudden	Abdominal, back, flank	Diffuse	+++
Mesenteric ischemia/infraction	Sudden	Periumbilical	Diffuse	+++
Gastroenteritis	Gradual	Periumbilical	Diffuse	+ to ++
Pelvic inflammation	Gradual	LQ, pelvic	Localized	++
Ruptured ectopic	Sudden	LQ, pelvic	Localized	++

Abbreviations: LLQ: left lower qudrant; RLQ: right lower quadrant; RUQ; right upper quadrant; +: mild; ++: moderate; +++:severe.

Physical examination:

- The evaluation of patient appearance, vital signs, and an estimate of pain degree/severity should be performed to assess the requirement for emergency surgery.
- **Generalappearance** (expression, complexion, position etc.)
- The **conjunctiva** should be examined for the presence of jaundice or anemia. Jaundice may also be indicated in the **skin**.
- Regarding **posture**, patients who refuse to change posture are likely to have peritonitis. Patient contortion may indicate gallstones and ureteral stone.
- **Vitalsigns** (pulse, blood pressure, respiratory rate, temperature, consciousness level). Vital signs should always be measured in patients with acute abdomen:

-Tachypnea typically indicates intrathoracic disease and metabolic ketoacidosis. It raises the probability of pneumonia, cardiorespiratory failure, and bacteremia.

- Tachycardia, hypotension, and body temperature are correlated

withseverity and prognosis.

-The assessment of orthostatic changes in blood pressure and pulse rate has utility in the assessment of patients withacute abdominal pain since the pulse rate and blood pressure may be normal withoutdevelopment of intravascular dehydration during

the earlystage of bowel obstruction, peritonitis, and intestinal

ischemia,

- Estimate of **pain** (degree/severity) should be performed to assess the requirement for emergency surgery.

Abdominal examination:

- The abdominal medical examination comprises inspection, auscultation, percussion, and palpation.
- The chest, lower back, rectum, and urogenital apparatus should be examined as required.
- When examining the abdomen with the right hand, it is recommended that the examiner stand on the right side of the patient. It is important to confirm where the abdominal pain begins and if there are any changes in pain location.
- Patients should be adequately exposed from the xiphoid process to the groin.

- **Inspection:**

- abdominal surgery scarsvery likely indicate bowel obstruction,
- abdominal wall movement with respirations,
- herpes zoster infection,stigmata of cirrhosis, skinchangessuch as ecchymosis,
- abdominaldistension (localized or diffuse),abdominal beat, massand intestinalperistalsis
- hernias.

- **Auscultation:** peristaltic sound, abnormal peristaltic murmurs, bruit (+/-).
  - Auscultation is a mandatory component of the abdominal medical examination.
  - If the conduction of peristaltic sounds is good, auscultation at a singlelocation is considered adequate.
  - it is notrecommended that multiple auscultations be performed at differentlocations or for increased durationsin patients withacute abdomen, in whom peristaltic sounds cannot be heard, since the clinical significance of auscultation is limited in these patients.
- The identification ofabnormal peristaltic murmurs has utility in the diagnosis of bowelobstruction.The significance of abdominal bruits is currentlyunknownin cases of acute abdomen.

- **Percussion:**

- Quiet percussion has utility in detecting acute peritonitis.
- If percussion pain (percussion tenderness) is positive, it is not necessary for additional examination of rebound tenderness.
- Abdominal tension should be removed to identifytrue muscle guarding.
- Percussion has utility inidentifying the presence of ascites and measuring the size of the liver, bladder, and spleen.
-Tympanic sounds on percussion indicate expanded bowel and dullness indicates the presence of ascites or abdominal masses.

- **Palpation:**

- **Superficial (light) palpation:**

-Muscle guarding, muscle rigidity, and rebound tenderness.

- Muscle guarding is an early peritoneal stimulation sign and may develop on one side in cases of localized peritonitis.

- **Deep palpation:**

- Localized tenderness is usually reliable evidence of underlying disease in the case of abdominalpain.

- Generalizedtendernessis ofpoor diagnostic significancein the case of abdominalpain.

-Organomegaly, abdominal cavity masses such as those in the gallbladder, liver, spleen, or bladder may be detected .

- If possible, the size, pulsation, and presence of masses in the liver and spleen should be assessed by palpation and percussion.

- Pulsatile, distensible mass associated with tenderness is characteristic of abdominal aortic aneurysm (AAA).

- Observe patient expression whileevaluating the site and degree of abdominal pain. Patient eye closing during palpation (closed eye sign) increases the probability that organic disease is absent (sensitivity 33%, specificity 93.5%).

- **Special tests:**

- When acute appendicitis is suspected, the iliopsoas muscle test and the obturator test are recommended.
- When an obturator hernia is suspected, the Howship–Romberg sign should be confirmed.
- Abdominal wall tenderness test(Carnett's sign and modified Carnett's sign)has utility in diagnosing abdominal wall ache or psychogenic stomachache andexcluding intra-abdominal lesions.

- **Rectal examination(DRE):**

- Rectal examinationisnot recommended for the routine examination in the diagnosis of theacute abdomen because:
  - -the information obtained isextremely limited, and
  - - the examination is sometimes distressingfor the patient.
- Rectal examination may be indicated when:
  - - it is necessary to examine the stool properties
  - - anal diseases, such as hemorrhoidsand anal fistula, digestive tract hemorrhage, rectal cancer, prostatecancer or prostatitis, are suspected.

- **Vaginal examination:**

- No definitive evidence showing the utility of a routine internal examination of women with acute abdomen in the emergency room.
- When extrauterine pregnancy or gynecologic pathology, including pelvicinflammatory disease (PID), is suspected, pain upon movement of thecervical canal and appendicular tenderness on internal examination may be useful in the diagnosis.

- **Laboratory tests:**

- Common blood tests usually performed for acute abdomen
  - CBC: WBC, RBC, Hb, Ht, MCV, MCH, MCHC, Plt
  - Serum electrolytes: Na, K, Cl, Ca
  - Liver function tests: T-Bilirubin, AST, ALT, ALP, LDH (D-Bil)
  - Kidney function tests: BUN, Creatinine
  - Inflammation: CRP, CK
  - Blood sugar
- Blood test item for specific diseases:
  - Pancreas: Lipase, amylase
  - acute coronary syndrome (ACS) (suspected ACS, epigastric pain): cardiac troponin T, H-FABP, CPK-MB, (myoglobin).
  - Heart failure: BNP
  - Coagulation (Suspected coagulation disorder, DIC, Pulmonary

embolism or aortic dissection): PT, APTT, FDP, D-dimer
- Unconsciousness: NH3, (vitamin B12, alcohol)
- Infection: HBs antigen, HBs antibody, HCV antibody, HIV antibody& an examination for syphilis
- Blood gas analysis: pH, PaO2, PaCO2, HCO3, BE, Lactate (evaluation of the overall status, intestinal ischemia).
- Culture: Blood cultures
-Transfusion: Blood type, an irregular antibody

- **Urinalysis:**

-Generally, protein, glucose, pH, urobilinogen, bilirubin, ketones, occult blood, and nitrites are measured by urinalysis
- urinary human chorionic gonadotropin (hCG) levels have utility in the diagnosis of pregnancy.
- urinary porphobilinogen has utility in the diagnosis of acute porphyria.
- Urine qualitative analysis has utility in the diagnosis of ureteral stones, urinary tract infection, and ketoacidosis.

- **Imaging tests:**
  - **Plain abdominal radiography:**

-Its diagnosability is limited. and has novalue as a part of routine examinations.
- It is considered inpatients suspected to have bowel obstruction, ileus, gastrointestinalperforation, urinary calculus, emphysematous lesion, or a foreignbody,in settings without access to US or CT.
- Acute abdomen series is performed and include: a set of abdominal radiographs in the dorsal and standing position and a chest radiograph (if indicated) in the standing position.
- When a standing position isdifficult, a left lateral decubitus position can be substituted.

-**Abdominal US:**

- US is recommended as a screening test(expertise in US is required as this technique is highlyoperator-dependent)
- Particularly useful in:
-AAA rupture or acute cholecystitis
-Pregnant women, young women, or children in whom radioactive exposure is not desirable.

- **CT Abdomen:**

- CT may be indicated in all patients with acute abdomen.
- It can be omitted when a diagnosis ismade by a precedent examinations such as US.
- Radiation exposure should be considered with the use of CT.
- Enhanced CT image useful for detailed evaluation of organ ischemia, vascular lesions, or acute pancreatitis severity

- CT has high diagnosability for intestinal ischemia, gastrointestinal perforation, acute appendicitis, diverticulitis, biliary tract calculus, acute pancreatitis and intra-abdominal freeair among others.

- **MRI Abdomen:**

- MRI should be considered for acute abdomen due to hepatobiliarydisease and gynecological disease for which a diagnosis is notobtained by ultrasonography and CT.
- MRI (usually nonenhanced but enhanced when the benefit sufficiently exceeds equivalenttest) should be considered for a pregnant womanfor whom ultrasonography does notlead to a diagnosis.

- **Differential diagnoses:**

-**Differential diagnosis for RUQ pain:**

- Common**:** esophagus, stomach/duodenal disease, hepato-biliary diseaseis common.
- Gastrointestinal (GI): Cholecystitis, cholelithiasis, cholangitis,colitis, diverticulitis, appendicitis, liverabscess, hepatitis, liver masses, gastric/ duodenal ulcer, pancreatitis
- Vascular**:** Acute coronary syndrome, myocarditis,endocarditis, pericarditis, aortic dissection,superior mesenteric artery occlusion/ dissection
-Urinary: Nephrolithiasis, pyelonephritis, ureteral stone,renal infarction, nephrolithiasis, and others
-Respiratory: Pneumonia, pulmonary embolism, empyema
-Others: Fitz-Hugh-Curtis syndrome.

- **Differential diagnosis for epigastric pain:**

- Common: esophagus, stomach/duodenal disease, hepato-biliary diseaseis common.
- GI: Gastric ulcer, duodenal ulcer, bowelobstruction, colitis, diverticulitis,appendicitis, cholecystitis, cholelithiasis,cholangitis, liver abscess, hepatitis, livermasses, pancreatitis
- Vascular: Acute coronary syndrome, myocarditis,endocarditis, pericarditis, aortic dissection,superior mesenteric artery occlusion/dissection.
- Urinary: Nephrolithiasis, pyelonephritis, ureteral stone,renal infarction, adrenal infarction
- Others: Respiratory disease (pneumonia, pulmonaryembolism, empyema)

-**Differential diagnosis for LUQ pain:**

-GI: Esophageal rupture, esophagitis, esophagealspasm, gastric ulcer, gastritis, splenicinfarction, splenomegaly, splenic rupture, splenic abscess, spleen twisting, splenicaneurysm, diverticulitis, ischemic enteritis,bowel obstruction, left-sided appendicitis, pancreatitis, pancreatic tumor.

-Vascular: Acute coronary syndrome, myocarditis,endocarditis, pericarditis, aortic dissection,superior mesenteric artery dissection/occlusion

-Left kidney/adrenalgland: Renal infarction, adrenal infarction, pyelonephritis, nephrolithiasis, ureteral stone

- Others: The disease in the left chest (left lower lungpneumonia, left pneumothorax, left empyema)

-**Differential diagnosis for RLQ pain:**

- Common: intestinal disease, urinary disease, gynecological disease.
- GI: Appendicitis, colitis, diverticulitis,inflammatory bowel disease, irritable bowelsyndrome, cholecystitis, pancreatitis, inguinalhernia
- Urinary: Prostatitis, epididymitis, ureteral stone,urinary tract infection
- Obstetrics andgynecological disease: Extrauterine pregnancy, endometriosis,ovarian hemorrhage, ovarian cyst rupture,torsion of ovary, uterine myoma, pelvicperitonitis, appendicular abscess (Fallopiantubes/ovarian abscess), adnexitis
- Vascular: Aortic dissection, aneurysm rupture
- Others: Iliopsoas abscess, retroperitoneal hemorrhage

- **Differential diagnosis for subumbilical pain:**

-Common: intestinal disease, urinary disease, gynecological disease.
- GI: Appendicitis, colitis, diverticulitis,inflammatory bowel disease, irritable bowelsyndrome
- Urinary: Cystitis, ureteral stone, pyelonephritis,urinary retention
- Obstetrics and gynecological: Extrauterine pregnancy, endometriosis, uterine myoma, ovarian tumor, torsion ofovary, pelvic peritonitis, ovarian hemorrhage

-**Differential diagnosis for LLQ pain**:

- Common: intestinal disease, urinary disease, gynecological disease.
- GI: Constipation (obstruction by the stool), bowelobstruction (including hernia incarceration)bowel tumor, colitis (infective, ischemic), inflammatory bowel disease, bowel infection,diverticulitis.
- Urinary: Prostatitis, epididymitis, ureteral stone,urinary tract infection
- Obstetrics andgynecological: Extrauterine pregnancy, endometriosis, ovarian hemorrhage, ovarian cyst rapture,torsion of ovary, uterine

myoma, pelvicperitonitis, appendicular abscess (fallopiantubes/ovarian abscess), adnexitis
-Vascular: Aortic dissection, aneurysm ruptured
- Others: Iliopsoas abscess, retroperitoneal hemorrhage

-Differential diagnosis for periumbilical pain:
- Common: esophagus, stomach/duodenal disease, hepato-biliary system
- GI: Acute appendicitis (initial symptoms), smallintestinal obstruction, simple bowel colic, pancreatitis
- Vascular: Mesenteric artery occlusion, acute coronarysyndrome, abdominal aortic aneurysm,splanchnic artery dissection
- Others :Tabes dorsalis, acute glaucoma, urachalremnant

-Differential diagnosis for generalized abdominal pain:
- Vascular: Aortic aneurysm rupture, aortic dissection,mesenteric artery occlusion, mesentericveinthrombosis
- GI: Gastrointestinal perforation/obstruction(strangulated), acute gastritis, acute enteritis, rupture of abdominal organ, pancreatitis
- Endocrine: metabolic Diabetic ketoacidosis, alcoholic ketoacidosis,acute porphyria
- Others:poisoning (lead, arsenic, etc.), IgA vasculitis(Henoch-Schonlein purpura), bilateralpneumonia.

-Differential diagnosis for abdominal and back pain:
- Should be careful about retroperitoneal lesions
- Vascular: Aortic aneurysm rupture, aortic dissection
- GI: Acute pancreatitis (chronic pancreatitis),cholelithiasis, acute cholecystitis, splenicinfarction
- Urinary: Renal/ureteral stone, renal infarction
- Others: Herpes zoster, compression fracture, iliopsoasabscess

-Differential diagnosis for severe central abdominal painwith shock:
- Acute pancreatitis,
- superior mesenteric artery occlusion,
- hemoperitoneum,
-aortic aneurysm rupture,
-aortic dissection,
- gastrointestinal perforation,
- intestinal necrosis,
- acute coronary syndrome,
- extrauterine pregnancy (women in child-bearing age).

-Differential diagnosis of acute abdomen in the elderly patients:

- Gallstone disease
-Acute cholecystitis
-Acute appendicitis
- Peptic ulcer and perforation
- Diverticulitis
-Small bowel obstruction
- Large bowel obstruction
- Acute cholangitis
- Abdominal aortic aneurysms rupture
-Acute mesenteric ischemia
- Superior mesenteric artery embolism (50%)
- Superior mesenteric artery thrombosis (15–20%)
- Non-occlusive ischemia (20%)
-Mesenteric venous thrombosis (5%)
- Atypical causes:

Urinary tract infection, pyelonephritis, myocardial infarction (inferior wall), pulmonary embolism, congestive heart failure with hepatic congestion, pneumonia, constipation, urinary retention, or an abdominalmuscle injury.

-Differential diagnosis of acute abdomen in women:

Themost frequent causes include:

-Gynecological:

-Gravid women:

- ectopic pregnancy
- miscarriage

-Nongravid and gravid women:

- adnexal (ovarian) torsion,
- pelvic inflammatory disease (PID),
- ovarian cyst rupture,
- ovarian hemorrhage,
- Mittelschmerz pain
-uterine fibroids

- endometriosis

-Non-gynecological:

-acute appendicitis

- bowel obstruction,

- acute cholangitis,

- acute cholecystitis,

- ureteralcalculi,

- urinary tract infections,

- peptic ulcer,

- gastrointestinal perforation,

-Differential diseases for acute abdomen other than abdominal and retroperitoneal disease:

-Extra-abdominal causes:

*Cardiovascular: - Acute coronary syndrome,
- endocarditis, pericarditis,
-myocarditis, aortic dissection,
-aortic aneurysm rupture

*Respiratory: -Respiratory Pneumonia, pleuritis,
- empyema, pneumothorax,
- pulmonary thromboembolism

*Esophageal: - Esophageal rupture,
- esophageal spasm,
- esophagitis

*Musculoskeletal:- Radiculopathy,
- spinal cord/peripheral nerve tumor,
- spinalosteoarthritis, herniated disk,diskitis,
- iliopsoas muscleabscess,
- osteomyelitis, costochondritis,
- slippingrib syndrome,
- Mondor disease,
- ACNES(abdominal cutaneous nerve entrapment syndrome.

*Groin, pubic region: -Torsion of spermatic cord,
- epididymitis,
-Inguinal, femoral, obturator hernial (incarceration),
- Hemorrhoids,
- Anal fistula.

-**Systemic disease:**

*Hematological, allergic, connective tissue diseases:

-Acute leukemia, hemolyticanemia, sickle cell disease,

- lymphoma, systemic lupuserythematosus, rheumatoid arthritis, dermatomyositis,

- polyarteritis nodosa, IgAvasculitis (Henoch-Schonlein purpura),

- food allergy,angioedema, eosinophilicenteritis

*Endocrine, metabolic:

- Acute adrenal insufficiency,

- diabetic ketoacidosis,

- hyperthyroidism, porphyria,

- uremia

* Poisoning:

- Drug allergy (insect/spider stab, snake toxin),

- lead poisoning

* Infection:

-Streptococcal sore throat,

- herpes zoster, varicella,

- osteomyelitis, typhoid fever,

- tuberculosis, brucellosis,

- toxicshock syndrome

* Others:

- Acute glaucoma,
- abdominal epilepsy, abdominalmigraine,
-mental disorder, foreign body, heat stroke,
-familial Mediterranean fever,
-gynecological(ovulation) pain.

- Primary care(initial treatment) outline of acute abdomen(Fig.1)

- The initial treatment of acute abdomen in a **two-step method**:

Step 1:

1-Differentiate clinical condition and the life-threatening diseases.
2-Confirm vital signs, airway (A), breathing (B), circulation (C), and consciousness.
3-Emergency treatment is required if any abnormality is identified during ABCD assessment
4-Differentiate super emergent disease and emergent disease while stabilizing the physiologic state of the ABC of patients with abdominal pain and abnormal ABCD
5- Request a hospital transfer to a specialty institution in patients with abnormal vital signs and in whom emergency tests or radical treatments for the causative disease is challenging.Transfershould only be done after stabilizing the patient's condition (securing the airway and starting initial treatment such as establishing intravenous access routes).

Step 2:

1-Evaluation of clinical condition and physical examination.

2-When vital signs are stable, determine whether the cause of the abdominal pain requires surgical intervention according to the findings of a medical history, physical examination,laboratory findings and imaging studies.

3-In patients with sudden onset or increasing pain, emergency surgery is often required.

4-Initial transfusions in case of acute abdomen:

a- It should be initiated immediately when intraabdominal infections are suspected even if circulatory dynamics are stable.

b-Crystalloid solutions, such as Ringer's solutions, should be used.

c- Albumin preparations should be considered in patients in shock and requiring large transfusion volumes or who have hypoalbuminemia.

d-Erythrocyte transfusion should be initiatedat hemoglobin levels 7g/dl with a target of 7-9 g/dl.

5-Analgesic use in case of acute abdomen (Fig.2):

a- Use analgesics early before a definitive diagnosis is made regardless of the cause of acute abdomen since pain control is associated with improved diagnosis and treatment.

b-Acetaminophen 1gm iv is recommended regardless of pain severity.

c-Intravenous narcotic analgesics should be added accordingto pain severity.

c-Antispasmodics (e.g., butylscopolamine bromide) is used as adjuvant therapy rather than first drug choice in cases of colic.

d-NSAIDs should be used in colic of:

- the biliary tract (1st drug of choice)
- ureteral calculi (if not applicable, use opioids).

e-Morphine, opioids such as fentanyl, an antagonistic analgesic such as pentazocine, and buprenorphine should be consideredin cases of acute abdomen.

6-Antimicrobial agents use in caseof acute abdomen:

a-Blood cultures should be taken and antimicrobial agents administered when intraabdominal infections are diagnosed or suspected.

b-In cases of septic shock due to infected acute abdomen, antibiotics should be initiated within 1 h of presentation.

c- In cases without shock, and intra-abdominal infections are diagnosed or suspected antimicrobial agents should be given within 8 h of presentation.

d-When surgeryfor the intra-abdominal infections is necessary, additional antimicrobialagents should be given for less than 1 h (if possible, less than 30 min) prior to the start of surgery to prevent surgical site infection.

Figure 1:Flowchart for the initial treatment of acute abdomen: **a two-step method.**

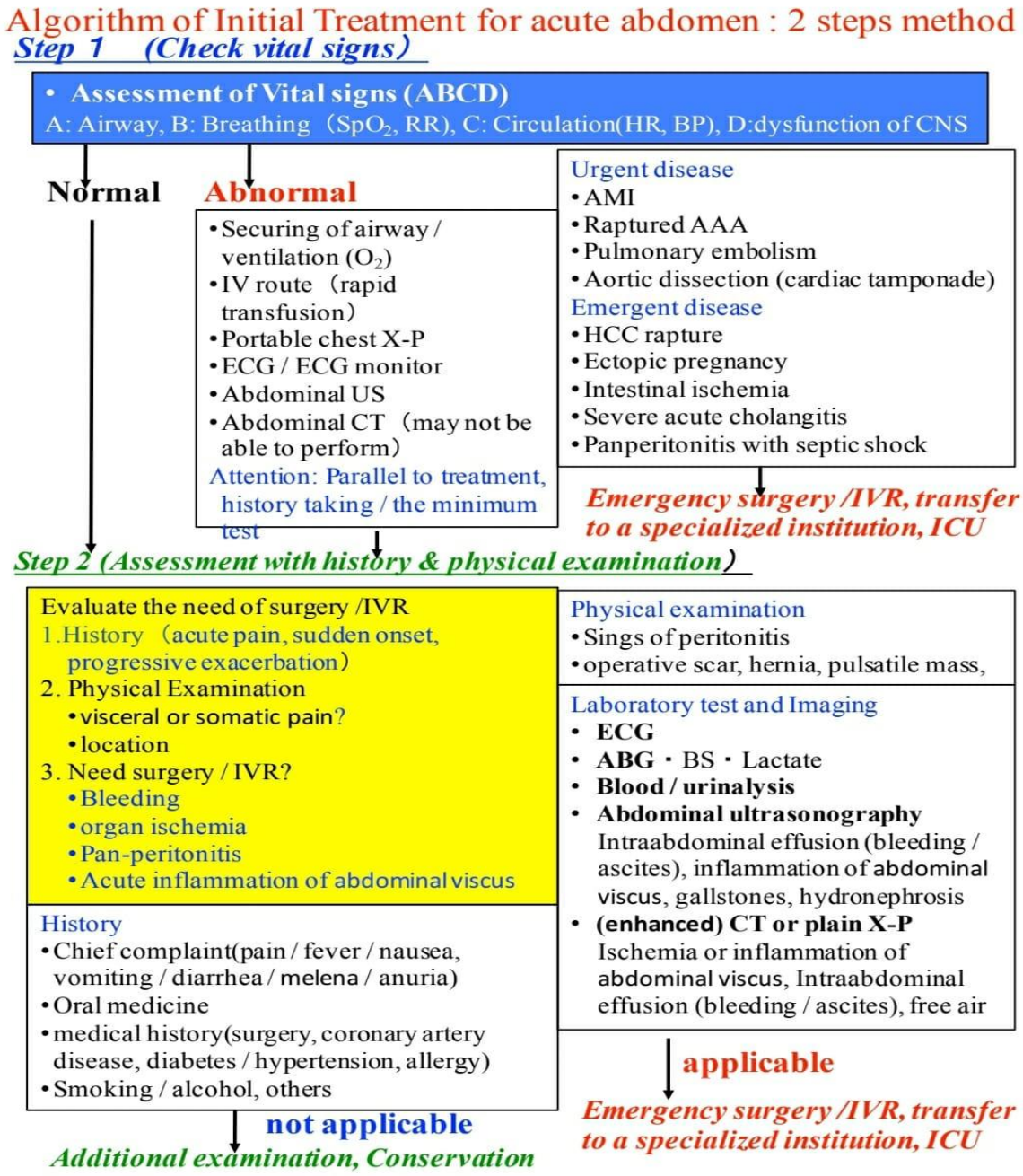
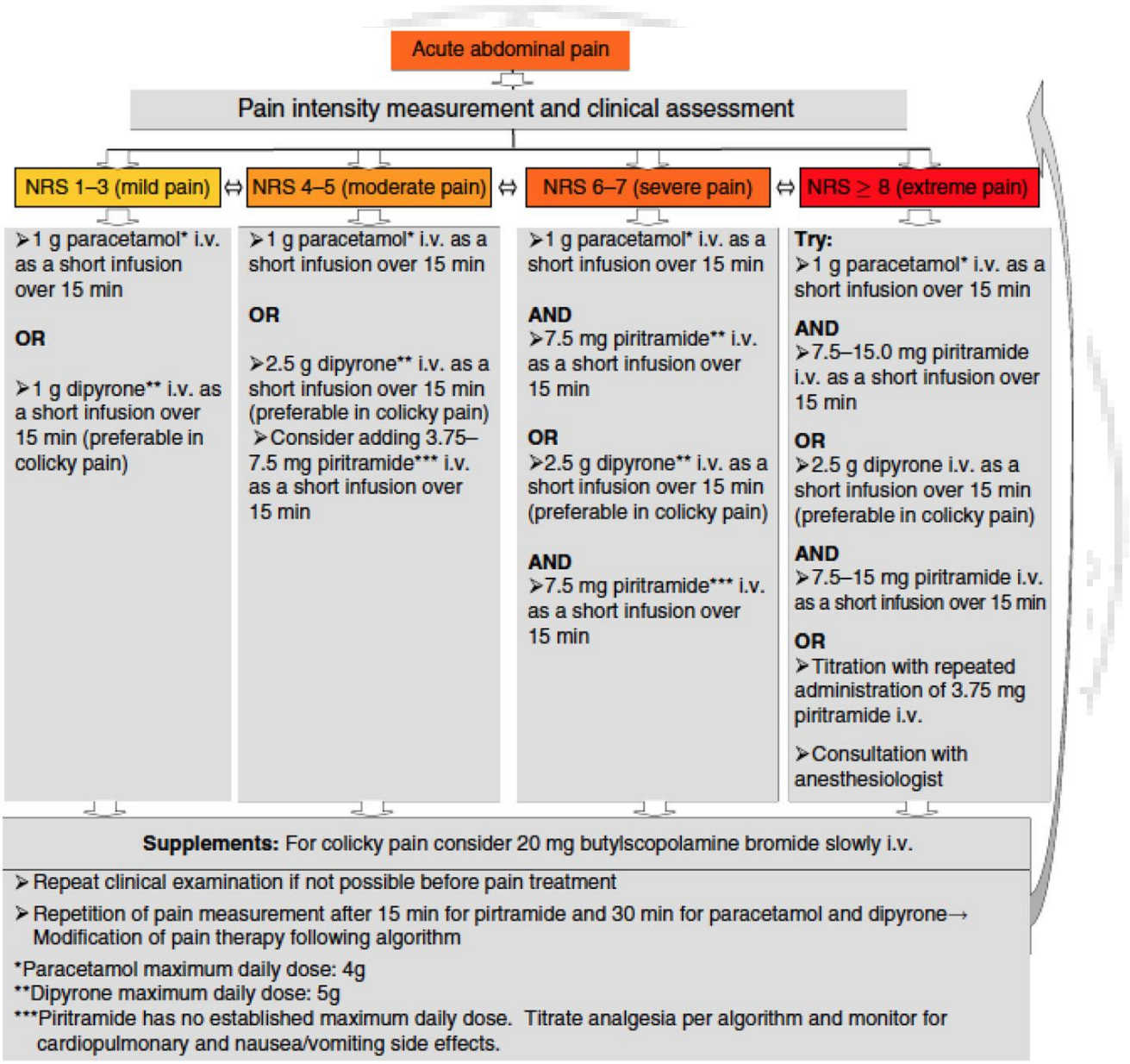


Figure 2:Algorithm for the selection of prediagnostic analgesia for acute abdominal pain in the general adult population.



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Asthma

CPG Source	Recommendation	Level of Evidence
Initial management in the first hour		
GINA **1995-2018**	**Supplementaloxygen** • Oxygen by face mask to achieve and maintain percutaneous oxygen saturation of 94–98%	Evidence A
GINA **1995-2018**	**Short-actingbeta2-agonist(SABA)** **5yearsandyounger** • 2-6puffs of salbutamol by spacer, or 2.5 mg of salbutamol by nebulizer, every 20 minutes for the first hour. **6yearsandolder** • For mild to moderate exacerbations, repeated administration of inhaled SABA (up to 4–10 puffs every 20 minutes for the first hour)	Evidence A
GINA **1995-2018**	**Systemiccorticosteroids** **5yearsandyounger** • **For severe exacerbations:** give oral prednisolone (1–2 mg/kg up to a maximum 20 mg for children <2 years old; 30 mg for children 2–5 years) • OR, intravenous methylprednisolone 1 mg/kg 6-hourly on day one. **6yearsandolder** **Usedinallbutthemildestexacerbations** • 1–2 mg/kg/day oral prednisolone for children 6–11 years up to a maximum of 40mg/day) or 200 mg hydrocortisone in divided doses Duration (short course)	Evidence A Evidence A Evidence B
SIGN **1990- 2016**	**Ipratropiumbromide** • In case of moderate to severe asthma in all age groups, start with ipratropium bromide at a dose of 250ug by nebulization mixed with SABA to be repeated every 20	Evidence A

GINA 1995-2018	Nebulizedisotonicmagnesiumsulfate: • Nebulized isotonic magnesium sulfate 150 mg added to/ mixed with each nebulized salbutamol and ipratroprium bromide can be given in severe asthma exacerbations in the first hour of treatment for children ≥2 years old, particularly those with symptoms lasting <6 hours and in setting where intravenous magnesium sulphate is not suitable.	Evidence B
GINA 1995-2018	Inhaledcorticosteroids 6yearsandolder • High-dose ICS given within the first hour after presentation reduces the need for hospitalization in patients not receiving systemic corticosteroids	Evidence A
Further management after the first hour		
GINA 1995-2018	Short-actingbeta2-agonist(SABA) 5yearsandyounger • If symptoms improved at 1st hour but recurred, give additional 2–3 puffs SABA per hour • Admit to hospital if >10 puffs required in 3–4 hours. • Failure to respond at 1 hour, or earlier deterioration, should prompt urgent admission to hospital and a short-course of oral corticosteroids	Evidence D Evidence D
GINA 1995-2018	6yearsandolder • The dose of SABA required varies from 4–10 puffs every 3–4 hours up to 6–10 puffs every 1–2 hours, or more often. No additional SABA is needed if there is a good response to initial treatment.	Evidence A
GINA 1995-2018	Intravenousmagnesiumsulfate: • For severe asthma exacerbations in children 2 years and above who fail to respond to initial treatment, give slow IV infusion of magnesium sulfate as a single dose of 50 mg/kg/dose (max. 2gm) over 20-60 minutes in the following setting: Emergency department in hospitals and with close monitoring of the vital data.	Evidence A

1- Assessment of Acute Asthma Exacerbation Severity in children ≥ 6 years *:* -

Mild/Moderate	Severe
• Talks in phrases, prefers sitting to lying, not agitated • Respiratory rate increased <30 breaths/minute • Accessory muscles not used • Pulse rate 100-120 bpm • O2 saturation (on air) >92% • PEF >50% predicted or best	• Talks in words, sits hunched forward, agitated • Respiratory rate increased >30 breaths/minute • Accessory muscles in use • Pulse rate >120 bpm • O2 saturation (on air) <92% • PEF ≤50% predicted or best

Copied from GINA Guidelines 2018

2- Initial Assessment of Acute Asthma Exacerbations in children 5 years and younger

Symptoms	Mild	Severe
Altered consciousness	No	Agitated, Confused or Drowsy
Oximetry on presentation (SaO2)	>95%	<92%
Speech	Sentences	Words
Pulse rate	<100 beat/minute	>200 beats/minute (0-3years) >180 beats/ minute (4-5 years)
Central Cyanosis	Absent	Likely to be present
Wheeze intensity	Variable	Chest may be quiet

3 : Factors that increase the risk of Asthma-related death in children 6 years and more:

- A history of near fatal asthma requiring intubation and mechanical ventilation.
- Hospitalization or emergency care visit for asthma in the past year.
- Currently using or having recently stopped using OCS (a marker of event severity).
- Not currently using ICS.
- Over-use of SABAs, especially use of more than one canister of salbutamol (or equivalent) monthly.

***The presence of one or more of these risk factors should be quickly identifiable in the clinical notes, and these patients should be encouraged to seek urgent medical care in the course of exacerbation*.** **GINA Guidelines 2018**

4. : Risk Factors for Asthma Exacerbation:

Risk factors for exacerbations include:
• Uncontrolled asthma symptoms **Additional risk factors, even if the patient has few symptoms:** • High SABA use (≥3 canisters/year) • Having ≥1 exacerbation in last 12 months • Low FEV1; higher bronchodilator reversibility • Incorrect inhaler technique and/or poor adherence • Smoking, or exposures to tobacco smoke; indoor or outdoor air pollution, indoor allergens (e.g. house dust mite, cockroach, pets, mold), • Obesity, chronic rhinosinusitis, blood eosinophilia • Ever intubated for asthma. • Major psychological or socio-economic problem for child or family

5. : Criteria for hospitalization:

Patients' clinical status and lung functions 1 hour after commencement of treatment are more reliable predictors of the need for hospitalizations than patients' status upon arrival.

- If pre-treatment FEV1 or PEF is <25% predicted or personal best, or post-treatment FEV1 or PEF is <40% predicted or personal best, hospitalization is recommended.
- If post-treatment lung function is 40-60% predicted. Discharge may be possible after considering the patients' risk factors and availability of follow-up care.
- If post-treatment lung function is >60% predicted or personal best, discharge is recommended after considering risk factors and availability of follow up care.

Other factors associated with increased likelihood of need for admission include:

- Female sex, non-white age
- Use of more than 8 beta2-agonist puffs in the previous 24 hours
- Severity of exacerbation (e.g. need for resuscitation or raid medical intervention on arrival, respiratory rate >22 breaths/minute, oxygen saturation <95%, final PEF < 50% predicted).
- Past history of severe exacerbation (e.g. intubations, asthma admissions).
- Previous unscheduled office and emergency department visits requiring use of OCS.

Discharge plan for children 6 years and above:

- Prior to discharge from the hospital or emergency department to home, arrangement should be made for a follow-up appointment within one week, and strategies to improve asthma management including medications, inhaler skills and written asthma action plan, should be addressed.

Follow-up after emergency department presentation or hospitalization for asthma:

- Following discharge, the patient should be reviewed by their health care provider regularly over subsequent weeks until good symptoms control is achieved, and personal best lung functions is reached or surpassed. Incentives such as free transport and telephone reminders improve primary health care follow up but have shown no effect on long term outcome.
- Patients discharge following hospitalization or emergency department presentation for asthma should be especially targeted for an asthma educational program, if one is available. Patients who were hospitalized may be particularly receptive information and advice about their illness. Health care provider should take the opportunity to review:

- The patient's understanding of the cause of their asthma exacerbation
- Modifiable risk factors foe exacerbation
- The patient's understanding of the purposes and correct uses of medications
- The actions the patient needs to respond to worsening symptoms or peak flows.

- Referral to exert advice should be considered for patients who have been hospitalized for asthma, or who repeatedly present to an acute care setting despite having a primary care provider. No recent studies are available, but earlier studies suggest that follow-up by a specialist is associate with fewer subsequent emergency department visits, or hospitalization, and asthma control.

6. : Discharge management after hospital or emergency department care for asthma:

Medications
Inhaled Corticosteroids (ICS) • **Initiate ICS prior to discharge, if not previously prescribed. Patients currently prescribed ICS containing medications should generally have their treatment stepped up for 2-4 weeks and should be reminded about the importance of adherence with daily use.** **Oral Corticosteroids (OCS)** • **Prescribe at least 3-5 days course of OCS (1-2 mg/kg/day to a maximum of 40 mg/day). Review progress before ceasing OCS. If OCS is dexamethasone, treatment is only for total 1-2 days. For patients considered at risk of poor adherence, intramuscular corticosteroids maybe considered (Evidence B).** **Reliever medication** • **Transfer patients back to as-needed rather than regular reliver medication use,** • • • **based on symptomatic and objective improvement. If ipratropium bromide was used in emergency department or hospitals, it may be quickly discontinued, as ot is unlikely to provide benefit.**
Risk factors that contributed to the exacerbation

- **Identify factors that may have contributed to the exacerbation and implement strategies to reduce modifiable risk factors. An exacerbation severe enough to require hospitalization may follow irritant or allergen exposure, inadequate long-term treatment, problem with adherence and/or lack of a written asthma action plan, as well as unavoidable factors such as viral respiratory infections.**

Self-management skills and written asthma action plan

- **Review inhaler technique.**
- **Review technique with PEF meter if used.**
- **Provide a written action plan, or review the patient's exiting plan, either at discharge or as soon as possible afterwards. Patients discharged from the emergency department with and action plan and PEF meter have better outcomes than patients discharged without their resources.**
- **Evaluate the patient's response to exacerbation. If it was inadequate, review the action plan and provide written guidance to assist if asthma worsen again.**
- **Review the patient's use of controller treatment before and during the exacerbation. Was it increased promptly and by how much? Were OCS added and if not, why not? Consider providing a short course of OCS to be on hand for subsequent exacerbations.**

Follow-up appointment

- **A follow-up appointment within 2-7 days of discharge should be made with the patient's usual health care provider, to ensure that treatment is continued, that asthma symptoms are well controlled, and that the patient's lung function reaches their personal best (if known).**

Indication for Immediate transfer to the hospital for children 5 years and below:

Immediate transfer to hospital from a primary health care unit is indicated if a child ≤ 5 year with asthma has any of the following:

At initial or subsequent assessment

- Child is unable to speak or drink
- Cyanosis
- Subcostal retraction
- Oxygen saturation <92% when breathing room air
- Silent chest on auscultation

Lack of response to initial bronchodilator treatment

- Lack of response to 6 puffs of inhaled SABA (2 separate puffs, repeated 3 times) over 1- 2 hours.
- Persistent tachypnea* despite three administration of inhaled SABA, even if the child shows other clinical signs of improvement.

Social environment that impairs delivery of acute treatment, or parent/carer unable to manage acute asthma at home.

Normal respiratory rate <60 breaths/minute in children 0-2 months, < 50 breaths/minute in children 1-2 months, <40 breaths/minute in 1-5 years.

GINA Guidelines 2018

Figure (1) Asthma Exacerbation Management in Children Algorithm 1

First: Assess is it asthma?

Risk Factors for Asthma Related Death?

Severity of the exacerbation?

Mild/Moderate

- Talks in phrases, prefers sitting to lying, not agitated
- Respiratory rate increased <30 breaths/minute
- Accessory muscles not used
- Pulse rate 100-120 bpm
- O_2 saturation (on air) >92%
- PEF >50% predicted or best

Severe

- Talks in words, sits hunched forward, agitated
- Respiratory rate increased >30 breaths/minute
- Accessory muscles in use
- Pulse rate >120 bpm
- O_2 saturation (on air) <92%
- PEF ≤50% predicted or best

Oxygen by face mask to achieve and maintain percutaneous oxygen saturation of 94–98%

5yearsandyounger

- 2-6puffs of salbutamol by spacer, or 2.5 mg of salbutamol by nebulizer, every 20 minutes for the first hour.
- **For severe exacerbations:** give oral prednisolone (1–2 mg/kg up to a maximum 20 mg for children <2 years old; 30 mg for children 2–5 years)
- OR, intravenous methylprednisolone 1 mg/kg 6-hourly on day one.

6years and older

- For mild to moderate exacerbations, repeated administration of inhaled SABA (up to 4–10 puffs every 20 minutes for the first hour)
- **Used in all but the mildest exacerbations** 1–2 mg/kg/day oral prednisolone for children 6–11 years up to a maximum of 40mg/day) or 200 mg hydrocortisone in divided doses Duration (short course)

In Moderate to Severe Cases

Ipratropium bromide at a dose of 250ug by nebulization can be mixed with SABA to be repeated every 20 minutes (for 1 hour only).

Figure (2) Asthma Exacerbation Management Algorithm 2

Assess the child as regards Symptoms:
If symptoms still persistent or worsening
If symptoms recur within 4 hours after improvement
Admit

5years and younger

- If symptoms improved at 1st hour but recurred, give additional 2–3 puffs SABA per hour
- Admit to hospital if >10 puffs required in 3–4 hours.
- Failure to respond at 1 hour, or earlier deterioration, should prompt urgent admission to hospital and a short-course of oral prednisolone (1–2 mg/kg up to a maximum 20 mg for children <2 years old; 30 mg for children 2–5 years)
  OR, intravenous methylprednisolone 1 mg/kg 6-hourly on day one
- Continue Oxygen by face mask to achieve and maintain percutaneous oxygen saturation of 94–98%
  (short course).
-

6years and older

- The dose of SABA required varies from 4–10 puffs every 3–4 hours up to 6–10 puffs every 1–2 hours, or more often.
- Continue Oxygen by face mask to achieve and maintain percutaneous oxygen saturation of 94–98%
- Continue or intiate 1–2 mg/kg/day oral prednisolone for children 6–11 years up to a maximum of 40mg/day) or 200 mg hydrocortisone in divided doses Duration (short course)
-

For severe asthma exacerbations in children 2 years and above who fail to respond toinitialtreatment:
give slow IV infusion of magnesium sulfate as a single dose of 50 mg/kg/dose (max. 2gm) over 20-60 minutes in the following setting: Emergency department in hospitals and with close monitoring of the vital data.

If improved:
Discharge home with: home management plan
Follow up plan

If Not Improving Or Deteriorating
consider **PICU ADMISSION**

Summary

Treatment of Acute asthma exacerbation in children

Treatment in the first hour

1. **Oxygen by face mask** (1L /min) to achieve oxygen saturation 94-98%.

2. **Short-acting β 2 agonist Salbutamol (SABA) and Ipratopium bromide inhalation:**

Children 5 years old and younger:

- 2-6 puffs of Salbutamol by pMDI with spacer or 2.5 mg by nebulizer, every 20 minutes for first hour.
- For children with moderate-severe exacerbations, 2 puffs **of ipratropium bromide 80mcg or 250mcg by nebulization** /20 min mixed with SABA.

Children 6 years and older:

- **For mild to moderate exacerbations**: repeated administration of inhaled Short-acting β 2 agonist up to 4-10 puffs every 20 minutes in the first hour.
- **For severe exacerbation:** 2 puffs of ipratropium bromide 80mcg **(250mcg by nebulizer)**/ 20 min mixed with SABA for one hour.

N.B. If inhalation therapy is not possible an I.V. Terbutaline 2 mcg/kg may be given over 5 minutes followed by continuous infusion of 5mcg/kg/hour with monitoring of the vital data.

3. Systemic corticosteroids:

Children 5 years old and younger:

- Initial dose of oral prednisolone (1-2mg/kg up to a maximum **20 mg/day** for children < 2years; **30 mg/day** for children 2-5 years) the duration for 3-5 days course is sufficient and can be stopped abruptly.
- Or I.V. Methylprednisolone 1mg/kg/6 hours in day 1.

Children 6 years and older:

- Used for all but the mildest exacerbations 1-2 mg /kg for children 6-11 years, maximum **40 mg/day**, for children 12 years and adolescents **50mg/day** total dose.

4. Inhaled corticosteroids: Children 6 years and older

High dose ICS "Quadrupled the patient' ICS low dose" (average 500-1600 mcg BDP equivalent) can be given within the first hour reduce the need for hospitalization in patients not receiving systemic corticosteroids.

5. Magnesium Sulphate: Children 2 years and more

- Consider **nebulized isotonic Mg. sulphate** 150mg 3doses mixed with each nebulized salbutamol and ipratropium bromide In the first hour, can be given in severe exacerbation particularly with symptoms lasting less than 6 hours and in setting where I.V. Mg. Sulphate is not suitable.
- When patients fail to respond to initial treatment, **single dose I.V Mg. Sulphate** 50mg/kg infusion over 20-60 min. has been used within the first hour, maximum dose 2gm . It should be given in emergency department in hospitals and with close monitoring of the vital data.

Further management after the first hour

1. **Short-acting β 2 agonist Salbutamol inhalation (SABA):**

Children 5 years old and younger:

- If symptoms improved after one hour, but persist or recurred within 3-4 hour give additional 2-3 puffs SABA per hour.
- . Admit to hospital if **>10 puffs required in 3-4 hours** .

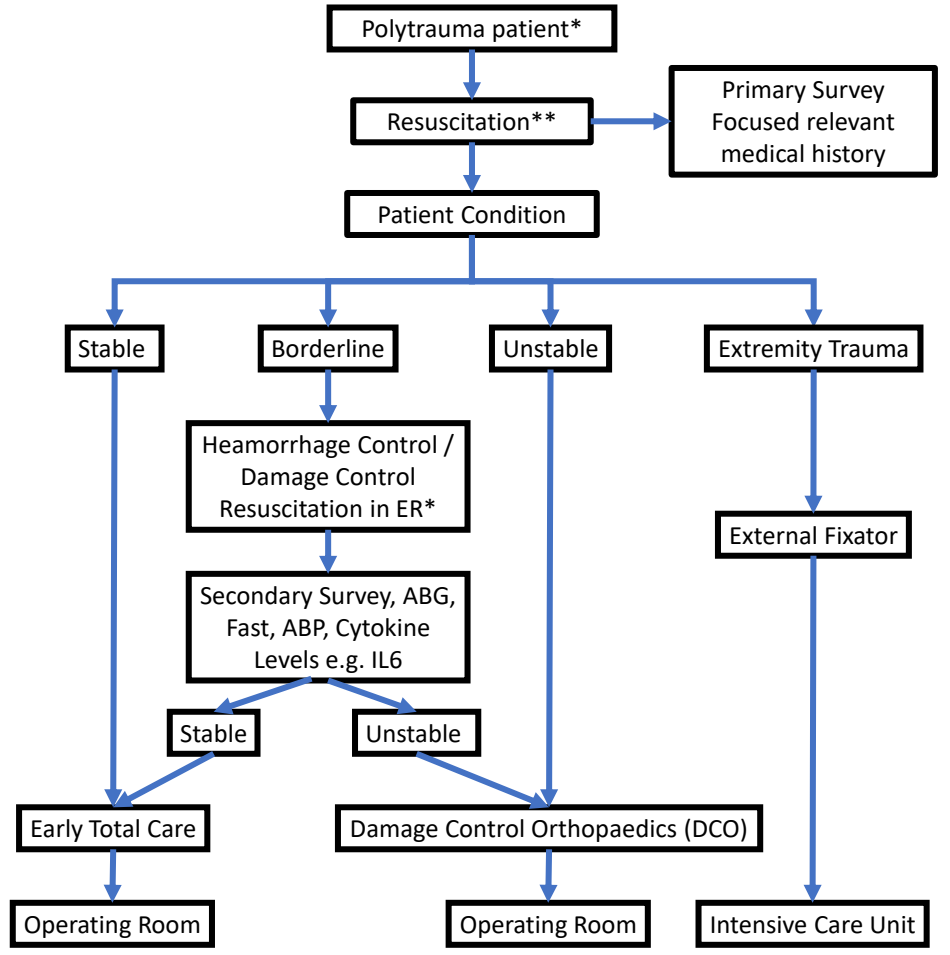
- Failure to respond after one hour, or earlier deterioration should prompt urgent admission to hospital and a short course of oral corticosteroids given.

Children 6 years and older:

- The dose of SABA required varies from 4-10 puffs every 3-4 hours up to 6-10 puffs every 1-2 hours, or more often.
- No additional SABA is needed if there is a good response to initial treatment.

2. **Maintain current controller treatment** for both young and older children if prescribed ICS or LTRA or both. If ICS not prescribed previously give **double the low dose** to be continue few weeks or months.

Polytrauma Protocol



* Ploytrauma Definition: two injuries that are greater or equal to 3 on the AIS and one or more additional diagnoses (pathologic condition), that is, hypotension (systolic blood pressure e 90 mm Hg), unconsciousness (GCS score ≤ 8), acidosis (base deficit ≤ −6.0),coagulopathy (PTT ≥ 40 seconds or INR ≥ 1.4), and age (≥70 years)

** See Heldelberg University Algorithm For Resuscitation details

Abbreviations: ABG: Arterial Blood Gas; ABP: Arterial Blood Pressure; FAST: Focused assessment sonography for trauma; IL6: interleukin-6

Evolving concepts and strategies in the management of polytrauma patients. Upadhyaya, Gaurav K. et al. Journal of Clinical Orthopaedics & Trauma, Volume 12, Issue 1, 58 - 65

Multiple Trauma

Treatment Algorithm for Multiple Trauma

University of Heidelberg - surgical emergency admission

Period Red

Aim: Airway, Breathing, Circulation, Neurology; Cervical immobilization; Evaluation of the vital functions; Evaluation of the need to intubate the trachea; venous access; fluid therapy

Surgery	Anesthesiology	Radiology
• bleeding control • immobilisation • undress the patient	• breathing sounds, check ET • intubation if necessary • blood pressure • venous access, volume replacement • emergency drugs if necessary • ABG, laboratory, crossmatch	• sonography (FAST) • chest film

Patient conditions: stable or unstable ? Operation is indicated, immediately?

Period Yellow

Aim: stabilization of vital functions, emergency diagnosic procedures*
*if patient is intubated and there are clinical hints of multiple trauma (see table) -> Multislice computertompgraphy of the head and the trunk, if not: conventional diagnostics and complemernary computertomography

Surgery	Anesthesiology	Radiology
• whole body check	• fluid therapy • stomach tube • antibiotics, if indicated • catheter in an artery, if indicated • control of ABG	• multislice computer-tomography of the head and the trunk

***assume multiple trauma:** fall from height (> 3 meter), ejection of passenger, dead of another passenger, pedestrian or cyclist crash, high speed motor vehicule accident, strong deformity of vehicule, entrapping, explosion

Patient conditions: stable or unstable ? Operation is indicated, immediately?

Period Blue

Aim: further diagnosis, stabilization

Surgery	Anesthesiology	Radiology
• urinary catheter • tetanus prophylaxis • emergency consults • reevaluation	• fluid therapy • packed blood cells • fresh frozen plasma • emergency drugs • thermocontrol • reevaluation	• control of chest film • radiological examination of spine and extremities, if indicated

Patient conditions: stable or unstable ? Operation is indicated, immediately?

Period Green

Aim: complementary diagnosis, final interventions/operations

Surgery	Anesthesiology	Radiology
• organization: OP • documentation	• central venous line if needed • transesophageal echocardio-graphy, if needed • bronchofiberscopy, if needed • fluid control • organization: ICU • documentation	• CT-diagnosis • control of sonography • documentation

→ documentation contemporary and bedside
"Caution: critical result"

Patient conditions: stable or unstable ? Operation is indicated, immediately?

Open Fracture

Patient with open fracture → ATLS© (ABCDE) Life before limb → Open fracture care D-I-S-C approach

Documentation

Control (Heamorrhage): Direct pressure
Tourniquet last resort
Neurovascular Examination: + Compartment syndrome
Control Pain: Analgesia
Care of the wound: See below
Straighten and Align Limb (Neurovascular Exam)
Photograph the wound
Classify: Gustilo/Anderson* (See next page)

Immobilization

Splint the fracture (Neurovascular Examination again)
Imaging: X-ray: two orthogonal views, two joints

Systemic antibiotics

Antibiotics: ASAP ideally within 3 h of trauma
- Type I : max 48 h
- Type II and III: max of 72h or until soft tissue closure

According to local antimicrobial protocol and guidelines
- **Initially**: co-amoxiclav or cephalosporin / Clindamycin (if allergic)- type III add gentamycin
- **At time of debridement**: + gentamycin
- **At definitive stabilization/soft tissue coverage**: Gentamicin + vancomycin or teicoplanin

Care of the wound

Care of the wound
- Straighten and Align Limb (Neurovascular Exam)
- Exploration and irrigation is not allowed
  - Remove Gross Contamination
  - Moist saline dressing / no antiseptics
- Avoid tight wraps to prevent circulatory compromise

Griffin M, et.al Update on the management of open lower limb fractures. Open Orthop J. 2012;6:571-7

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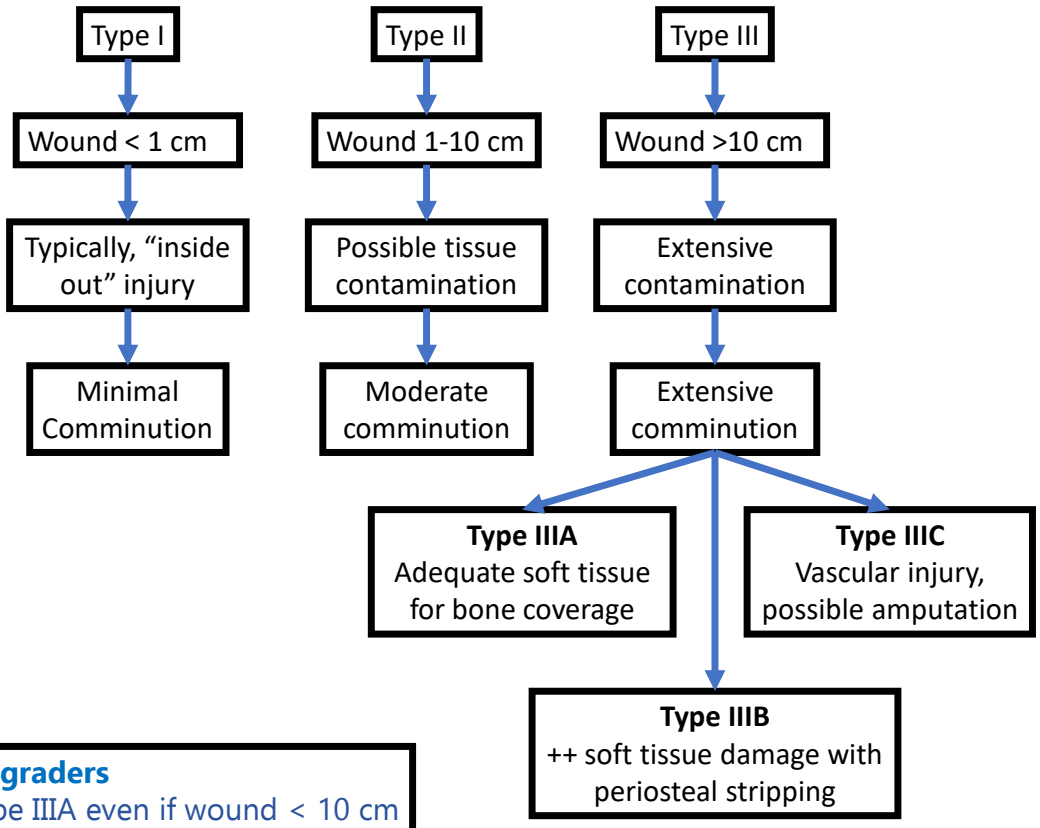
والمستشفيات التعليمية
الأمانة الفنية

Open Fracture Classification Gustilo-Anderson



Upgraders

Type IIIA even if wound < 10 cm

- Gross contamination
- Delayed presentation > 6hrs
- Farmyard injury
- Ballistic /blast injuries
- High energy gun shot

Prepared by the Orthopaedic Advisory Committee, 2023

Gustilo RB. Use of antimicrobials in the management of open fractures. Arch Surg. 1979 Jul;114(7):805-8.
Gustilo RB, Anderson JT. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. J Bone Joint Surg Am. 1976 Jun;58(4):453-8.

Guidelines for management of crush injuries of the hand

The hand is a complex structure comprising of several tissues (skin, nerves, blood vessels, tendons, bones joints and intrinsic muscles) that are closely packed in a small space.

Hand injuries can be complex and difficult to classify. Each injury has a unique pattern and requires a unique plan of management. Crush injuries of the hand involve damage to multiple structures within the hand, loss of tissue, devascularisation and possibly amputation of digits. There are no set procedures that can be described, however fundamental principles can be laid down to guide surgical management.

The common features of these injuries are:

- Severe injury
- Multiple structures are damaged
- Different tissues are damaged to varying degrees
- Tissue loss
- Devascularisation

Optimal management of such injuries requires a clear understanding of principles of management, correct decision making and wide repertoire of surgical techniques.

Due to the varying patterns there is no standard operating procedure, however based on principles and guidelines correct treatment can be formulated for each patient

Optimum management requires a planned and decisive approach. The surgeon or the team must be well versed with techniques necessary for management of all the structures within the hand, microsurgery and free tissue transfer.

The overriding principle of reconstruction is to provide a maximally functional hand in the shortest period of time with minimum number of surgical procedures. Each procedure must be performed with clear goals and should set stage for the next procedure

Midgeley and Entin in 1979[1] provided a succinct list of characteristics that constitute functionality in the hand, These are.

- Strength,
- Position,

- Length,
- Stability,
- Mobility,
- and Sensibility

The essential components of management are, accurate assessment of the injury and creating a reconstructive plan by structures. The essential components for primary surgery are precise and complete debridement, skeletal stabiliation, vascular repair and if vessels are exposed, soft tissue cover. Secondary surgery should include procedures to enhance the function of the hand or to improve the aesthetics of the hand. These include bone grafting, fusion, tendon and nerve reconstruction, flap de-bulking and toe to hand transfers.

Assessment: A complete assessment is crucial. Accurate assessment leads to better planning

History should include the following:

When (Time of injury): indicates ischemia time and risk of infection.

How (Mechanism): Indicates severity of injury.

Where (Environment): indicates the possible infective agent, for example anaerobes in barnyard injuries, and marine organisms in injuries involving sea water.

Age of the patient and co-morbidities: such as associated diabetes, cardiac and renal conditions which may compromise the safety of surgical procedures.

If the patient is found to have life threatening comorbidities attempts should be made to optimise the patient for anaesthesia or the surgery may be considered under regional anaesthesia if possible.

1.1. Examination of the patient: life before limb

It is easy to be focussed on the injured hand, however in any trauma an overall examination of the patient must be performed in order to rule out any **associated life threatening trauma.**

Crush injuries of the hand may be associated with head injuries, thoracic injuries or abdomino-pelvic injuries causing the patient to collapse or decompensate during surgery.

This should be followed by lifesaving procedures if necessary which includes fluid resuscitation, blood transfusions, chest tube etc.

Amputated Parts: Amputated parts should be cleaned with saline, wrapped in a moist gauze and placed in a dry plastic bag. The bag should be placed in a container with ice. The container should be labelled clearly with patient's name and identification number. The parts should never be discarded. They can either be replanted or used for spare parts.

1.2. Examination of the hand

A conventional examination of the hand may not be possible. Light touch sensation may be carried out to assess sensation in the digits; formal muscle testing may be impossible due to pain, bleeding and anxiety. However synthesis of anatomical knowledge, the posture of the hand, the colour of digits and visible location and depth of trauma can provide a fairly accurate diagnosis of the injury.

1. Skin: Skin flaps with a narrow base, The nature of skin separation and the length-breadth ratio of skin flaps will indicate whether the skin flaps will survive or will need to be excised and primary or secondary skin cover may be needed.
2. Loss of cascade: indicates injury to tendons or proximal innervation. Extended posture of digits indicates flexor tendon injuries, whereas finger drop indicates extensor injuries.
3. Pale or cyanosed digits: indicate devascularisation. Since arteries and nerves are within the same sheath, concomitant nerve injury should be anticipated.
4. Rotation or deviation of fingers: indicates fractures or joint dislocations.

Other procedures to be done

1. **Antibiotics:** Intravenous antibiotic therapy should be commenced in the emergency department itself. In general broad spectrum coverage for gram positive, gram negative and anaerobic organisms is necessary. Specific antibiotics may be needed in special circumstances such as exposure to biological contaminants (agricultural or barnyard injuries) or sea water.
2. **Sterile dressing**: should be applied in order to send the patient to radiology
3. **X-rays:** x-rays are essential in all cases. However it may be impossible to obtain good X-ray views particularly when the hand has been bandaged. Hence it may be necessary to perform X-Rays preoperatively on table when the patient is under anaesthesia and the wounded hand can be positioned without pain. X-ray images of the amputated parts should also be obtained.
4. **Recording:** Once the patient has been resuscitated the hand may be inspected in the emergency department. Photographs should be taken which can be reviewed without having to repeatedly open the wounds. These photographs also serve as records for future reference. The photographs should be stored in secure electronic medical records or secure servers and not shared on social media.
5. **Planning:** Once a visual examination has been performed, a systematic record of injuries should be made in structure by structure manner. This is essential for creating a reconstructive plan.

The list should include.

- Palmar skin
- First dorsal interossei, Thenar and hypothenar muscles
- Palmar/Digital arteries
- Palmar/digital nerves
- Metacarpals/phalanges of individual digits and joints
- Dorsal skin
- Vascularity of individual digits (highlighting devascularised or congested digits)
- Each structural loss should be complemented with a reconstructive plan. For example.
- Palmar skin: groin flap
- First dorsal interossei, Thenar and hypothenar muscles: debridement
- Palmar/Digital arteries: vein grafts from ipsilateral forearm
- Palmar/digital nerves: sural nerve graft
- Metacarpals/phalanges of individual digits and joints: K wire fixation& cerclage wiring
- Dorsal skin: groin flap may need a combined superficial epigastric flap
- Vascularity of individual digits (highlighting devascularised or congested digits): devascularised Middle finger (arterial injury in the palmar region)

Preparation: The above plan guides preemptive surgical positioning and preparation and avoids setbacks during surgery.

1.3. Surgical preparation

The prepared parts will be chosen according to the needs as follows

- Sural nerve graft, saphenous vein graft: Prepare the leg or use spare-parts
- Smaller vein grafts: Prepare the Forearm
- Tendon: Prepare the forearm Palmaris longus, sacrifice FDS, use spare-parts
- Flap: Prepare the Groin/abdomen and prepare the ipsilateral thigh for skin grafting the abdominal donor defect.

Goals of surgical management

- Restoration of maximal function
- In shortest possible time
- Through minimum number of procedures
- Each procedure should be performed with clear goals and should set stage for the next procedure

Primary (Emergency) surgery: A well performed primary surgery is critical for optimum outcomes. It should accomplish the following goals

1. Excisional debridement
2. Skeletal stabilisation
3. Revascularisation
4. Skin cover

NB. May or may not include tendon and nerve reconstruction/bone grafts.

1. **Excisional debridement:** Excisional debridement is performed in layer by layer fashion. Skin flaps are excised to healthy bleeding skin. Contused or de-vascularised fat and muscles should be completely excised as they will form nidus for infections that may compromise the vascular repairs and the flaps. Contaminated fracture ends should be shortened using saw and medullary cavity should be curetted.

- Longitudinal structures: intact arteries and nerves are preserved. Contamination should be removed through excision of the adventitia under magnification. Cut or crushed arteries and nerves should be debrided under magnification until healthy ends are seen. Intact tendons should be preserved and paratenon or partial longitudinal excision may be carried out to remove contamination. Cut tendon ends should be debrided.

NB. Repeated and incomplete debridement should be avoided. This potentially results in secondary infection of non-viable tissue and delays reconstructive procedures. However in certain circumstances such as biological or faecal contamination a cautious approach of staged debridement may have to be taken.

Intraoperative 'triage' and decision making: Decide Early What is salvageable, what is not[3, 4, 5]

- Thumb ray reconstruction is the first priority, if distal stump is available, any suitable digital stump can be used to restore the thumb
- The **least injured fingers are then salvaged** irrespective of the position.
- The **best amputated segment** is transferred **to the best available stump.**
- **Severely traumatized digits** may be **used as a source of spare parts**

2. **Skeletal stabilisation:** Forms the foundation for the entire reconstruction. Decision regarding the method of stabilisation is governed by the nature of contamination, adequacy of debridement, and presence of bone loss. If contamination is minimal plate and screw fixation can be performed. Bridge plates are used for maintaining length. However in cases of significant contamination K-wire fixation or external fixation is preferred. The placement of implants especially external fixation should be planned such that it does not interfere with the placement of the flap. Bone grafts represent non-viable tissue and are preferably performed as secondary procedures in open contaminated injuries.

3. **Revascularisation:** The next essential step is that of revascularisation. For successful revascularisation the vessels should be adequately debrided and healthy ends approximated. Whenever necessary reversed vein grafts should be used for arteries. For circumferential wounds it is essential to anastomose veins or venae commitans.

4. **Skin cover:** Flap cover is essential if blood vessels are exposed. Skin cover can be performed as a secondary procedure if vessels have a viable skin or muscle cover. In situations of

major vascular injuries abdominal or groin flaps are performed as they do not sacrifice donor vessels from the injured limb.[6] However if the hand requires revascularisation, anterolateral thigh flap[7] or a radial artery forearm flap from the contralateral upper limb may be used a flow through flaps. Some composite flaps such as the dorsalis pedis flap can be used to provide simultaneous reconstruction of tendons and skin.[8,9]

If vessels are not exposed the wound can be sealed using a sterile dressing and a flap cover may be planned electively within next 48 h. Undue delay may result in tissue desiccation and microbial colonisation. Vacuum assisted dressing (V.A.C) may assist in providing a sterile closure.

Rehabilitation: Rehabilitation in crush injuries may be complex and needs to be tailored to each injury. It can be affected by several factors such as strength and stability of skeletal fixation, combined flexor and extensor injuries in the same digit, and repaired ligaments such as collateral ligaments.

The aim should be to allow wound healing, maintain joint mobility, prevent adhesions and contractures and enhance scar maturation.

Secondary surgery: secondary surgery is performed after complete healing of the initial surgery. It is usually timed beyond 6 weeks from the initial trauma. During this period joints are mobilised to prevent stiffness, and soft tissue massage is performed to reduce oedema which may compromise the results of final reconstruction. The aim of secondary surgery is to enhance function and the appearance of the hand.

Secondary surgery is used to address the following:

1. Nerve reconstruction using nerve grafts
2. Tendon grafts or tendon transfers
3. Bone grafting
4. Joint fusion

Other reconstructive procedures such as **toe transfer** are planned either in the secondary stage or tertiary stage. Flap de-bulking is performed as a secondary or as the final procedure to improve the appearance of the hand as well as to enhance the function.

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CRUSHED LOWER LIMB

1. A multidisciplinary team, including orthopedic and plastic surgeons with appropriate experience, is required for the treatment of complex open fracture. If not available arrange for immediate referral to the nearest specialist center.

2. The primary surgical treatment (wound debridement/excision and skeletal stabilization and soft tissuereconstruction) of these complex injuries takes place at the specialist center.Rapid access to theatres is essential to avoid delay in management where right surgeon with the right facilities and with minimal delay so get union rates similar to those of closed fractures

Primary management in the emergency department

1. Airway with spinal control, Breathing and Circulation managed according to Advanced Trauma Life Support principles.

2. Stop external hemorrhage by direct pressure or, as a final resort, application of a tourniquet (record the time).

3. Neurovascular examination of the limb.

4. Analgesia if appropriate.

6. Repeat neurovascular examination.

7. Remove gross contaminants from the wound. Leave wound undisturbed until patient reaches the operating theatre. Wounds are handled only to:

(a) Remove gross contaminants

(b) Photograph for record

(c) Seal from the environment

- A ‘mini debridement’ of the open fracture in the emergency room does not aid treatment .Digital exploration of the wound is unnecessary, reveals little real information and should be avoided. Lavage through the open wound serves to drive particulate debris further in.

8. Photograph wound.

9. Cover wound with sterile, moist (saline) dressing and adhesive film or light bandage. Antiseptics inthe dressing should not be used .

11. Splint fracture (if not done prehospital).

12. Repeat neurovascular examination after any intervention.

13. IV antibiotics: co-amoxiclav (1.2 g) or cefuroxime (1.5 g) 8 hourly, or clindamycin 600 mg if the patient is allergic to penicillin.

14. Check tetanus status and administer prophylaxis if required.

15. X-ray: two orthogonal views, two joints – knee and ankle.

16. Immediate referral to the orthoplastic team

- **Examine circulation** by capillary refill, dorsalispedis and posterior tibial pulses palpation or by Doppler.

If major blood vessel injury is suspected, immediately refer to vascular surgeon. Muscle death starts to occur within 3-4 h of warm ischemia. Repeated examinations are done

- Compartment syndrome may not be evident at first check or there may be difficulties in a satisfactory clinical assessment owing to patient's general condition. Severe pain increased by active or passive movement of the toes or ankle or local pressure raises the possibility of compartment syndrome. Compartment pressures should be measured if clinical suspicion is aroused. Do not wait till full picture of pain, pallor, parethesia, loss of function is established.

- **Neurological examination** is done motor and sensory

Toes and ankle dorsiflexion (common peroneal nerve) and plantar flexion (posterior tibial nerve) should be tested and the possibility of more proximal injury (to the sciatic nerve, nerve roots or spinal cord) considered. Muscle paralysis is also seen with prolonged ischemia after arterial injury. Appreciation of light touch should be tested on the sole of the foot (posterior tibial nerve) and in the first dorsal web space (deep peroneal nerve).

Antibiotic Prophylaxis

1. Antibiotics should be administered as soon as possible after the injury and certainly within 3 h.

2. The antibiotic of choice is co-amoxiclav (1.2 g 8 hourly) or a cephalosporin

(e.g. cefuroxime 1.5 g 8 hourly), and this should be continued until soft tissue closure or

for a maximum of 72 h, whichever is sooner.

4. Gentamicin 1.5 mg/kg and either vancomycin 1 g or teicoplanin 800 mg should be given on induction of anesthesia at the time of skeletal stabilization and definitive soft tissue closure. These should not be continued post operatively. The vancomycin infusion should be started at least 90 min prior to surgery.

5. Patients with anaphylaxis to penicillin should receive clindamycin (600 mg IV 6 hourly preoperatively) in place of co-amoxiclav/cephalosporin. For those with lesser allergic reactions, a cephalosporin is considered to be safe

Wound Debridement

1. The only reasons for immediate surgical exploration are the presence of: (a) Gross contamination of the wound (b) Compartment syndrome (c) A devascularized limb (d) A multiply injured patient.

2. In the absence of these criteria, the wound, soft tissue and bone debridement is performed by senior plastic and orthopedic surgeons working together on scheduled trauma operating lists within normal working hours and within 24 hours of the injury unless there is marine, agricultural or sewage contamination. The 6 hour rule does not apply for solitary open fractures.

3. Traumatic wounds are excised comprehensively and systematically as follow

(a) Initially, the limb is washed with a soapy solution and a tourniquet is applied

(b) The limb is then 'prepped' with an alcoholic chlorhexidine solution, avoiding contact of the antiseptic with the open wound and pooling under the tourniquet

(c) Soft tissue debridement/excision is safely performed under tourniquet control, especially in cases of extensive degloving. This allows identification of key structures such as neurovascular bundles, which may be displaced, and permits accurate examination of tissues by avoiding blood-staining

(d) Visualization of the deeper structures is facilitated by wound extensions along the fasciotomy lines.

(e) The tissues are assessed systematically in turn, from superficial to deep (skin, fat, muscle, bone) and from the periphery to the center of the wound. Non-viable skin, fat, muscle and bone are excised

Skin is relatively resilient but is vulnerable to torsion/avulsion injuries, which lead to degloving in a plane superficial to the deep fascia and disruption of the septocutaneous and musculocutaneous perforating vessels. Crushing injuries lead to direct devitalization. In cases of extensive flap lacerations, care must be taken to ensure that as much of the integument as possible is preserved, although all non-viable skin must be excised

subcutaneous fat is relatively vulnerable and the zone of fat necrosis is often more extensive than that of the overlying skin. Extension of the wounds along fasciotomy lines (see Figures 13.1 and 13.2) allows for access to and excision of the subcutaneous fat as necessary.

Devitalized muscle may be difficult to assess, especially in cases of multiplanardegloving. The four 'C's should be looked for: colour (pink not blue), contraction, consistency (devitalized muscle tears in the forceps during retraction) and capacity to bleed. It is important to inspect the muscle groups behind the tibia as the fractured bone ends are often driven posteriorly and devitalized muscle fragments may be lodged in the medullary canal.

Bone debridement is reliant upon the surgical exposure and delivery of the bone ends to enable removal of particulate foreign material and a complete assessment of bone and soft tissue viability. Lavage is indicated but is not a substitute for debridement and should only follow after an adequate surgical removal of contaminants and devitalized tissue is performed.

(f) At this stage the injury can be classified and definitive reconstruction planned jointly by the senior members of the orthopaedic and plastic surgical team. There will be occasions when the soft tissue damage is difficult to assess. A secondlook should be undertaken 24-48 h later. However, multiple serial debridement has been shown to be associated with worse outcomes11 and is unnecessary.

(g) If definitive skeletal and soft tissue reconstruction is not to be undertaken in a single stage, then a vacuum foam dressing (or antibiotic bead pouch if there is significant segmental bone loss) is applied until definitive surgery

At the end of wound excision the wound bed should approach elective surgical conditions whenever possible, allowing the insertion of internal fixation if appropriate, followed by flap closure.

BONE Debridement

1- Extension of the traumatic wound is along the nearest fasciotomy incisions
2- Whilst a bloodless field during soft tissue debridement may be helpful, deflatingthe tourniquet before bone debridement allows satisfactory confirmation of a'capacity of the bone endsto bleed'.Thisis probably the most useful determinantof bone viability
3- Careful surgical delivery of bone ends through the wound extension aids circumferential assessment.
4. Particulate foreign matter is removed with periodic irrigation to keep clear visibility of the surgical field.
5. Loose fragments of bone which fail the 'tug test' are removed.
6. Fracture ends and larger fragments which fail to demonstrate signs of viability are removed.
7. Major articular fragments are preserved as long as they can be reduced and

fixed with absolute stability.

8. Lavage follows once a clean wound is obtained by a meticulous zone-by-zone debridement.

9. High pressure pulsatile lavage is not recommendet

Temporary Wound Dressings

Following excision of all non-viable tissues, if the soft tissue reconstruction is notperformed immediately, the wound should be covered with a dressing which preventsbacterial ingress and avoids dessication. The application of gauze soaked in antisepticsolutions such as povidone iodine does not have the desired antibacterial effect asthepovidone iodine is rapidly inactivated by serum at the concentrations availablecommercially, and there is a small risk of systemic toxicity.Furthermore, repeateddressing changes should be avoided to reduce bacterial ingress.

- 1. Negative pressure dressings: Foam dressings with the application of negative pressure meet some of the criteriaof an ideal dressing in the form of the Vacuum Assisted Closure
- Negative pressure dressings may reduce bacterial ingress and tissue desiccation as well as avoid pooling of serous fluid.
- 2. Negative pressure dressings are not used as a substitute for meticuloussurgical
- wound excision.
- 3. Negative pressure dressings are not a substitute for coverage of exposed
- fractures with vascularized flaps.
- 4. Antibiotic impregnated bone cement beads under a semi-permeable membrane are associated with reduced infection rates.
- 5. These beads are most applicable in patients with segmental bone loss, gross
- contamination or established infection, perhaps in combination with negative
- pressure dressings

-

- **Timing of Soft Tissue Reconstruction**

1. Local flaps are safely performed at the same time as skeletal fixation. Internalfixation is only undertaken if soft tissue coverage can be performed at thesame time.
2. Free flap reconstruction is best performed on scheduled trauma lists by experienced, dedicated senior surgical teams following adequate preparation ofthe patient, including imaging such as angiography or computed tomography(CT) scanning of comminuted fractures. This should be undertaken in a specialist center.

3. There is little evidence for the 5-day rule. Microsurgery is best performedbefore the vessels become friable or fibrosed and this becomes increasinglylikely after the first week. We recommend that definitive soft tissue reconstruction be undertaken within the first 7 days after injury.

SOFT TISSUE RECONSTRUCTION

1- Non exposed bone, split thickness graft is suitable
2- If exposed bone or deep tissue loss or exposed vital structure,

A. *Upper third leg :* Gastrocnemius muscle flap, Sural flap or recurrent genicular perforator flap
B. *middlethird leg* : solius muscle flap, Fasciocuteneous flap or perforator flap
C. *Lower third leg* : ReversedSural flap or perforator flaps
D. If this is not suitable: free flap is the solution

When Things Go Wrong with SoftTissues

1. Necrosis of a local flap over the fracture site is managed by early return to theatre and revision surgery to achieve healthy soft tissue coverage.
2. Limited tip congestion may respond to leech therapy.
3. Some local fasciocutaneous flaps may be more prone to develop complications in patients with comorbidities.
4. Free flap complications are reduced by patient preparation, careful planning and performing the anastomoses outside the zone of injury: ideallyproximally.
5. There is a low threshold for immediate re-exploration of a free flap withsuspected circulatory compromise.
6. Deep infection requires a return to the operating theatre, fracture site exploration, debridement, dead space management and antibiotic therapy. Fracture fixation may need revision.

Guidelines for Primary Amputation

1. A primary amputation is performed as a damage control procedure if there is uncontrollablehaemorrhage from the open tibial injury (usually from multiple levels of arterial/venous damage in blast injuries) or for crush injuries exceeding a warm ischemic period of 6 h.

2. Primary amputation is also needed for incomplete traumatic amputations where the distal remnant is significantly injured.

3. A primary amputation is considered an option where injury characteristics include one or several of the following:

(a) Avascular limbs exceeding a 4-6 h hour threshold of warm ischaemia
(b) Segmental muscle loss affecting more than two compartments
(c) Segmental bone loss greater than one-third of the length of the tibia.

4. Absent or reduced plantar sensation at initial presentation is not an indication for amputation.

5. Amputation levels are preferably transtibial or transfemoral (if salvage of theknee is not possible). Through-knee amputations are not recommended foradults.

6. The decision to amputate primarily should be taken by two consultant surgeons with, if possible, patient and family involvement.

7. Discussion with the nearest specialist center is advised when there is uncertainty or disagreement between surgeon recommendations and patient/family wishes.

Degloving

Degloving of the limb occurs in the plane superficial to the deep fascia and the extent of injury is often underestimated.

1. Thrombosis of the subcutaneous veins usually indicates need to excise the overlying skin.

2.. Circumferential degloving often indicates that the involved skin is notviable.

3. In severe injuries, multiplanardegloving can occur, with variable involvement of individual muscles and these may be stripped from the bone.
4. A second look may be necessary 24 -48 hours later to ensure that all thenon-viable tissues have been excised
5. definitive reconstruction is done within7 days.

The General Organization For
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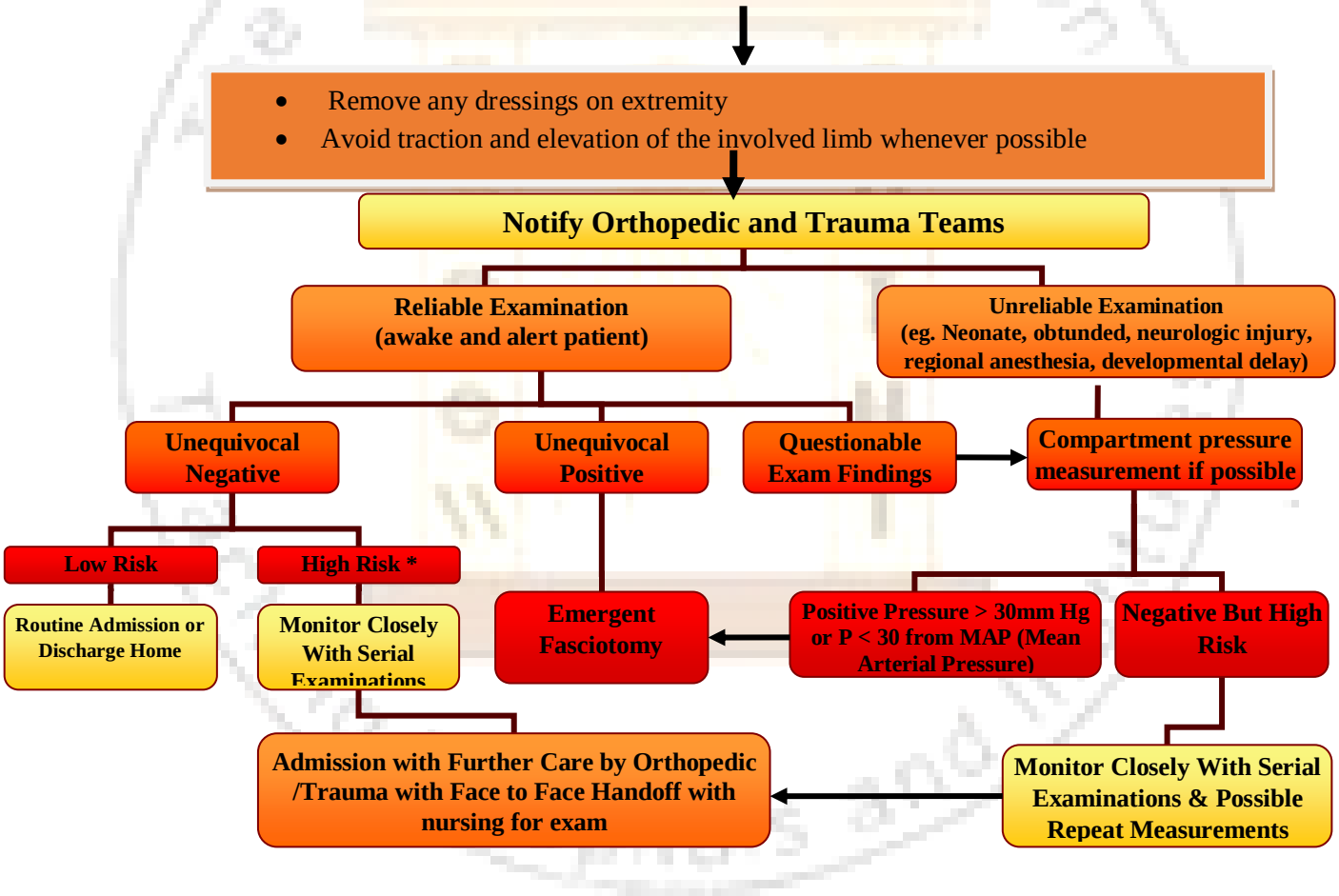
Technical Affairs

الهيئة العامة للمعاهد
والمستشفيات التعليمية
الأمانة الفنية

Acute Compartment Syndrome Clinical Pathway

Suspected Compartment Syndrome:
Clinical Suspicion based of history, mechanism of injury or provider experience:
Swollen/tense compartment: pain out of proportion to expectations despite adequate analgesia and increased with passive stretching to affected compartment
OR
the "Five Ps": pain,paresthesias, paralysis, pallor, and pulselessness
OR
The "Three As": Anxiety, agitation, increasing analgesic requirements



*high risk patients

Fractures/dislocations	Thromboembolism	Arterial injury	Constrictive dressings/splints/casts
Crush injuries	Drug abuse	Venomous bite	Burns
Reperfusion injury	Tourniquet use	Penetrating injury	Extravasation of fluids

Postpartum Hemorrhage

Definition:

Primary postpartum hemorrhage (PPH) is defined as the blood loss from the genital tract of at least 500 ml after a vaginal birth (or at least 1000 ml after a cesarean section) within 24 hours of delivery. However, this is an arbitrary value as patients with a low BMI may have a low blood volume and anemic women may have fewer physiological reserves. Hence, they may not tolerate a blood loss of 500 ml and may decompensate much earlier.

It is one of the leading causes of maternal mortality in the developing world.

Secondary PPH is defined as abnormal or excessive bleeding from the birth canal between 24 hours and 12 weeks postnatally.

Severity of PPH:

- Primary PPH can be minor (500-1000 ml) or major (more than 1000 ml).
- Major PPH can be further subdivided into moderate (1001-2000 ml) and severe (more than 2000 ml). It is important to be aware that minor PPH can easily progress to major PPH.
- Life-threatening hemorrhage (loss of more than 40% of total blood volume)
- Clinicians should be aware that the visual estimation of blood loss is inaccurate and that clinical signs and symptoms should be included in the assessment of PPH.
- Shock index=H.R./SBP(0.5 – 0.7 : no shock, > 0.9 : shock).
- Rule of 30: if SBP decrease by 30mmhg, pulse increase by 30 beats/min, HB and hematocrite decrease by 30%, this means that she lost 30% of her blood volume.

Prediction of PPH:

- Clinicians must be aware of risk factors for PPH. Women with risk factors for PPH should only be delivered in a hospital with a blood bank on site.
- Risk factors for PPH may present antenatally or intrapartum; care plans must be modified when risk factors arise.
- Most cases of PPH have no identifiable risk factors.
- PPH is caused by abnormalities of one or more of four basic processes; the four Ts: tone, trauma, tissue and thrombin. The most common cause of PPH is uterine atony.

A. Uterine atony:

- Prolonged labor/precipitate labor
- Uterine overdistension (polyhydramnios, multiple pregnancy, fetal macrosomia)
- Previous PPH
- Placenta previa
- Fibroids
- Uterine inversion
- General anesthesia
- Anemia

B. Trauma:

- Episiotomy
- Perineal lacerations
- Rupture uterus
- Instrumental delivery
- Cesarean section
-

C. Retained tissue:

- Retained placenta
- Placenta accreta

D. Coagulation failure:

- Placental abruption
- Pre-eclampsia
- Infection (chorioamnionitis, septicemia)

Prevention of PPH:

* Antenatal anemia should be investigated and treated appropriately as this may reduce the morbidity associated with PPH.
* Uterine massage is of no benefits in the prophylaxis of PPH.
* Management of the third stage of labor:
  Prophylactic ecbolicsshould be offered routinely in the third stage of labor as they reduce the risk of PPH by about 60%.

- Oxytocin and ergometrine-oxytocin (syntometrine)
  - Oxytocin (10IU IM) is the agent of choice. With CS, 5IU by slow IV injection should be given. A bolus dose may be inappropriate in some women such as cardiac patients (a low-dose infusion is a safer alternative).
  - The advantage of a reduction in the risk of minor PPH needs to be weighed against the adverse effects associated with syntometrine.Syntometrine may be used in the absence of hypertension in women at increased risk of PPH.

- Misoprostol
  - Oxytocin is superior to misoprostol in the prevention of PPH.

- Carbetocin(a longer-acting oxytocin derivative)
  - No significant differences between carbetocinand oxytocin in terms of risk of PPH.

For women at increased risk of PPH, it is possible that a combination of preventive measures might be superior to syntocinon alone to prevent PPH

- Tranexamic acid
  - Clinicians should consider the use of IV tranexamic acid (0.5-1 gm), in addition to oxytocin at CS to reduce blood loss in women at increased risk of PPH.

Management of PPH:

- Management of PPH involves four components, all of which must be undertaken SIMULTANEOUSLY:

I. Communication
II. Resuscitation
III. Monitoring & investigation
IV. Arresting the bleeding

I. COMMUNICATION:

- Relevant staff with an appropriate level of expertise should be alerted.
- The first-line obstetric and anesthetic staff should be alerted when women present with minor PPH (blood loss 500-1000 ml) without clinical shock.
- A multidisciplinary team involving senior members of staff (including the anesthetic team, blood bank and the laboratory specialists) should be called to attend to women with major PPH (blood loss >1000 ml) and ongoing bleeding or clinical shock.

II. RESUSCITATION

a. Measures for <u>minor PPH</u> without clinical shock:
   - Intravenous access (one 14-gauge cannula)
   - Immediate venepuncture(20 ml) for:
     -group and save
     -FBC

-coagulation screen, including fibrinogen
- Start warmed crystalloid infusion.

b. Full protocol for <u>major PPH</u> and ongoing bleeding or clinical shock:

- A and B –assess airway and breathing
  - High flow oxygen (10-15 l/min) via a facemask should be administered, regardless of maternal oxygen saturation. If the airway is compromised owing to impaired conscious level, anesthetic assistance should be sought urgently.
- C –evaluate circulation
- Place the patient in a flat position.
- Keep the woman warm
- Immediate venipuncture (20 ml) for:
  - cross-match (4 units minimum)
  - FBC
  - coagulation screen, including fibrinogen
  - renal and liver function for baseline
- Establish two 14-gauge IV lines
- Fluid replacement: until blood is available, infuse up to 3.5 L of warmed fluids, initially 2 L of isotonic crystalloids. Further fluid resuscitation can continue with additional crystalloids or colloids (succinylatedgelatin, gelofusine). Hydroxyethyl starch (voluven) should NOT be used. The nature of the infused fluid is of less importance than rapid administration and warming of the infusion.The best equipment available should be used to achieve rapid warmed infusion of fluids.
- Blood transfusion: compatible blood (supplied in the form of red cell concentrate) should be transfused as soon as possible. Patients with acute hemorrhage can have normal Hb. Therefore, the clinical picture should be the main determinant of the need for blood transfusion and time should not be unnecessarily spent awaiting laboratory results. The goal is to maintain Hbgreater than 8gm%. Special blood filters should

NOT be used as they slow infusions. If immediate transfusion is indicated, give group O, RhD-negative red cell units. Switch to group-specific red cells as soon as feasible.

- Fresh frozen plasma: transfuse 4 FFP units for every 4 units of red cell concentrate if:
  - hemorrhage is continuing and the results of hemostatic tests are not available
  - PT/APTT are prolonged
  - coagulopathy is suspected (placental abruption)
- Platelets: transfuse platelets at a trigger of 75000/microliter to maintain a level greater than 50000/microliter during ongoing PPH.
- Cryoprecipitate should be used to maintain a fibrinogen level of greater than 2 g/l, even if PT/APTT are normal.
- Tranexamic acid (TXA)
  - Early use of IV TXA (within 3 hours of birth) in addition to standard care is recommended for women with PPH. (WHO recommendation)
  - first dose of 1 g IV is given over 10 minutes (to avoid hypotension)
  - second dose of 1 g IV may be given if bleeding continues after 30 minutes or if bleeding restarts within 24 hours of the first dose
  - do not exceed 2 g in 24 hours
  - TXA administration beyond 3 hours does NOT confer any clinical benefit
  - TXA should be avoided if there is a clear contraindication to antifibrinolytic therapy (e.g. thromboembolic event duringpregnancy)
- The routine use of rFVIIais not recommended in the management of major PPH unless as part of a clinical trial. It may be considered as a treatment for life-threatening PPH, but should not delay or be considered a substitute for a life-saving procedure.
  - suggested dose is 90 micrograms/kg, which may be repeated in the absence of clinical response within 15-30 minutes
  - rFVIIawill not work if there is no fibrinogen. Therefore, fibrinogen should be above 1g/l
  - effectiveness may be suboptimal with severe thrombocytopenia (less than 20000/microliter)

- Thromboprophylaxis should be administered once the bleeding is arrested and any coagulopathy is corrected as there is a high risk of thrombosis. Alternatively, anti-embolism stockings, foot impulse devices or intermittent pneumatic compression devices can be used if chemical thromboprophylaxis is contraindicated (e.g. in case of thrombocytopenia).

  The main therapeutic goals of the management of massive blood loss are:
  - Hbgreater than 8 g%
  - Platelet count greater than 50000/microliter
  - PT less than 1.5 times normal
  - APTT less than 1.5 times normal
  - Fibrinogen greater than 2 g/l

  The*GOLDEN HOUR* refers to the time in which resuscitation must begin to ensure the best chance of survival. The probability of survival decreases sharply after the first hour, if the patient is not effectively resuscitated.

III. MONITORING & INVESTIGATION

For minor PPH without clinical shock:

- Blood group and save
- FBC
- Coagulation screen, including fibrinogen
- Pulse, respiratory rate and BP recording every 15 minutes.

For major PPH and ongoing bleeding or clinical shock:

- Cross-match (4 units minimum)
- FBC
- Coagulation screen, including fibrinogen
- Liver and renal function for baseline
- Monitor temperature every 15 minutes
- Continuous pulse, respiratory rate and BP recording (using oximeter, electrocardiogram and automated blood pressure recording)
- Foley catheter to monitor urine output
- A central venous line not only provides a means of accurate central venous pressure (CVP) monitoring but also a route for rapid fluid replacement

- Consider transfer to ICU once the bleeding is controlled or monitoring at high dependency unit on delivery suite, if appropriate
- Recording of parameters on a flow chart
- Documentation of fluid balance, blood, blood products and procedures

IV. ARRESTING THE BLEEDING

* The most common cause of primary PPH is uterine atony. Therefore, the initial management of PPH should involve a sequence of mechanical and pharmacological measures to stimulate myometrial contractions. These measures should be instituted in turn:
  - Rubbing up the uterine fundus
  - Ensure that the bladder is empty (Foley catheter, leave in place)
  - Oxytocin 5 IU by slow IV injection (dose may be repeated)
  - Ergometrine 0.5 mg by slow IV or IM injection (contraindicated in women with hypertension)
  - Oxytocin infusion (40 IU in 500 ml isotonic crystalloids at 125 ml/hour) unless fluid restriction is necessary
  - Carbetocin (a longer acting oxytocin derivatives)
  - Misoprostol 800 micrograms sublingually. Regardless of the route of administration (sublingual, rectal, vaginal), misoprostol takes 1 to 2.5 hours to increase uterine tone. Clinicians should be aware of this delay in the clinical effect of misoprostol.

    If significant hemorrhage continues after these measures, the team should consider transfer to the operating theatre for examination under anesthesia with an awareness of the impending need for surgical treatment

* If pharmacological measures fail to control the hemorrhage, surgical interventions should be performed sooner than later. Compression of the aorta may be a temporary but effective measure to allow time for resuscitation to catch up with the volume replacement and the appropriate surgical support to arrive.
  - Tamponade using various types of hydrostatic balloon catheters has superseded uterine packing as a first-line surgical management of atonic PPH (Foley catheter,

Bakri balloon, Sengstaken-Blakemore catheter, condom, Ruschballoon). The success rate is high (80-90%).

- Tamponade test: a positive test (control of PPH following inflation of the balloon) indicates that laparotomy is not required.
- In most cases, 4-6 hours of tamponade should be adequate to achieve hemostasis and ideally it should be removed during daytime hours in the presence of senior staff.

- Hemostatic brace sutures (B-Lynch suture, modified compression sutures) are effective in controlling severe PPH and in reducing the need for hysterectomy. The success rate is 70-80%. It is recommended that a laminated diagram of the brace technique be kept in theatre.
  - Compression sutures are associated with a low complication rate. A higher risk of uterine ischemia may occur if the procedure is combined with vessel ligation.
  - Combined use of hemostatic suturing and balloon tamponade has been reported.
- Stepwise uterine devascularization
- Internal iliac artery ligation (bilateral)
- Selective arterial embolization by interventional radiology (if available)
- Hysterectomy
  - SOONER RATHER THAN LATER (especially in case of placenta accretaor uterine rupture)
  - The decision should be made by an experienced consultant and preferably discussed with a second experienced clinician when feasible
  - Subtotal hysterectomy is the operation of choice unless there is trauma to the cervix or PPH following placenta previa.

SECONDARY PPH:

Causes of secondary PPH:

- Endometritis (secondary PPH is often associated with endometritis)
- Retained products of conception (RPOC)
- Uterine subinvolution (fibroid, uterine overdistension)
- Trophoblastic disease (rare)

Investigations:

- High vaginal and endocervical swabs
- Blood cultures if there is fever
- FBC
- Pelvic ultrasound may help to exclude the presence of RPOC although the diagnosis of retained products is unreliable. Therefore, the clinical findings (including the severity of bleeding and whether the cervical osis open) should be taken into account before the decision to perform surgical evacuation is made
- B-hCG

Management of secondary PPH:

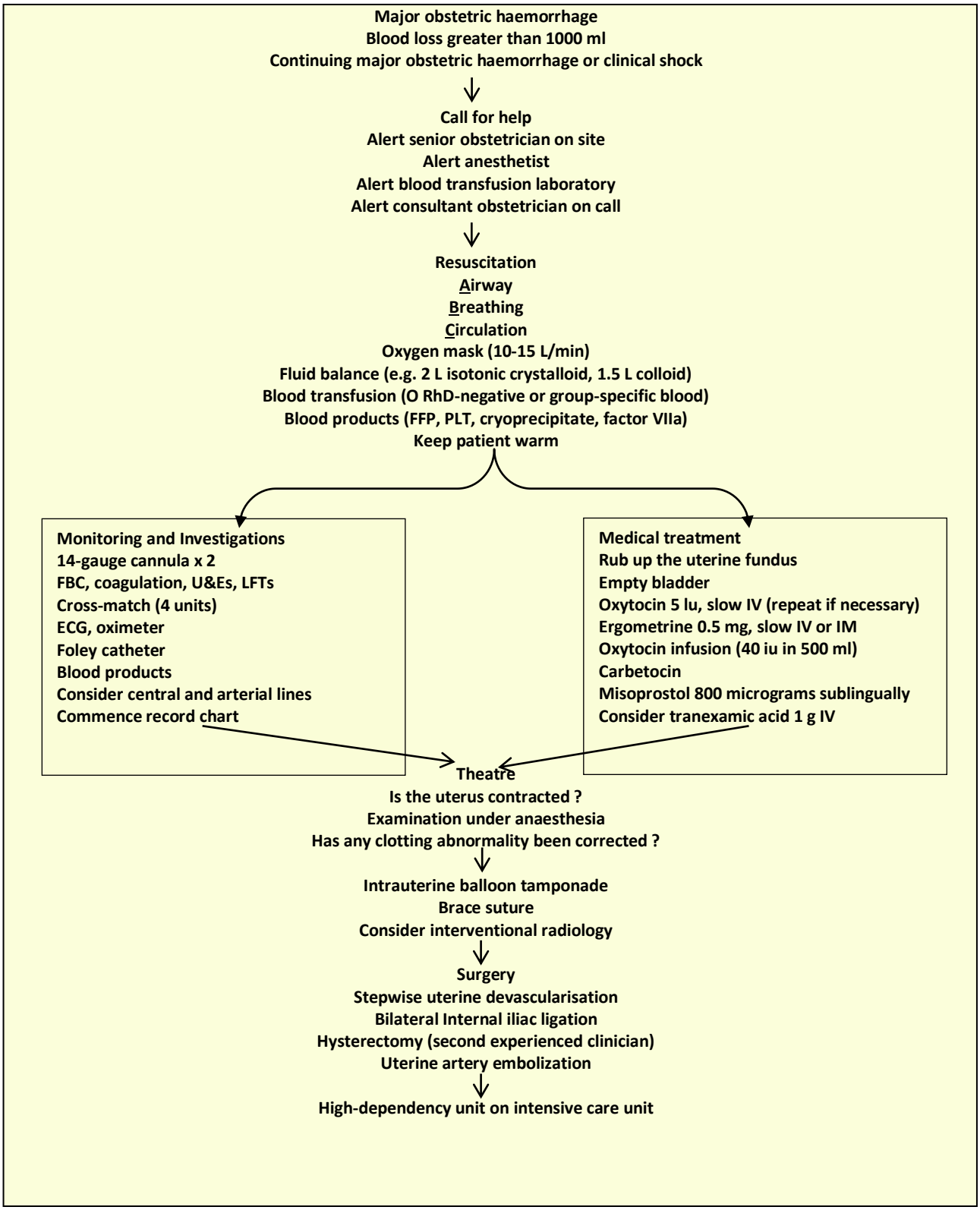
- Antimicrobial therapy: a combination of clindamycin and gentamycin is appropriate. Once endometritis has clinically improved with IV therapy, there is no additional benefit from further oral therapy.
- Surgical evacuation of RPOC: should be undertaken or supervised by an experienced clinician as the risk of perforation is high.
- Uterotonics such as misoprostol and ergometrine have been recommended in the management of secondary PPH, although evidence to support their use is limited.
- Uterine balloon tamponade use in cases of secondary PPH with ongoing bleeding has been reported.

Appendix 1: The causes of PPH

The four Ts	Risk factors/notes
Tone: abnormalities of uterine contraction	
Overdistension of uterus	Polyhydramnios, multiple gestation, macrosomia
Intra-amniotic infection	Fever, prolonged rupture of membranes
Functional/anatomic distortion of uterus	Rapid labour, prolonged labour, fibroids, placenta praevia, uterine anomalies
Uterine relaxants, e.g. magnesium and nifedipine	Terbutaline, halogenated anaesthetics, glyceryl trinitrate
Bladder distension	May prevent uterine contraction
Tissue: retained products of conception	
Retained cotyledon or succenturiate lobe	
Retained blood clots	
Trauma: genital tract injury	
Lacerations of the cervix, vagina or perineum	Precipitous delivery, operative delivery
Extensions, lacerations at caesarean section	Malposition, deep engagement
Uterine rupture	Previous uterine surgery
Uterine inversion	High parity with excessive cord traction
Thrombin: abnormalities of coagulation	
Pre-existing states	
Haemophilia A	History of hereditary coagulopathies or liver disease
Idiopathic thrombocytopenic purpura	Bruising
von Willebrand's disease	
History of previous PPH	
Acquired in pregnancy	
Gestational thrombocytopenic	Bruising
Pre-eclampsia with thrombocytopenia e.g. HELLP	Elevated blood pressure
Disseminated intravascular coagulation	
a) Gestational hypertensive disorder of pregnancy with adverse conditions	Coagulopathy
b) in utero fetal demise	Fetal demise
c) severe infection	Fever, neutrophilia/neutropenia
d) abruption	Antepartum haemorrhage
e) amniotic fluid embolus	Sudden collapse
Therapeutic anticoagulation	History of thromboembolic disease

Appendix II: A flow chart of the different steps for the management of major PPH

Resuscitation, monitoring, investigation and treatment should occur simultaneously



ANTEPARTUM HEMORRHAGE

Definition:

Antepartum hemorrhage (APH) is defined as bleeding from or into the genital tract, from 24 weeks of pregnancy and prior to birth. It complicates 3-5% of pregnancies and is a leading cause of maternal and perinatal mortality and morbidity.

Causes of APH:

- Abruptio placenta (30-35%).
- Placenta previa (20%). However, incidence is expected to be higher in Egypt due to the cesarean section epidemic.
- Uterine rupture (rare).
- Vasa previa (rare).
- Local causes (cervicitis, vulvovaginal varicosities, and genital tumors).
- Unexplained (usually attributed to marginal separation of the placenta).

Risk factors for APH:

Risk factors for placental abruption include:

- Previous abruption is the strongest risk factor for abruption.
- Pre-eclampsia
- Abdominal trauma
- Intrauterine infection.
- Pregnancy following assisted reproductive techniques.
- IUGR

- Smoking
- Drug abuse (cocaine, amphetamines).
- Multiparity.
- PROM.
- Non-vertex presentations.
- Polyhydramnios.
- First trimester bleeding.
- Advanced maternal age.
- Maternal thrombophilia.

Risk factors for placenta previa include:

- Previous placenta previa.
- Previous cesarean delivery.
- Multiple gestation.
- Multiparity.
- Advanced maternal age.
- Assisted conception.
- Previous abortion.
- Previous uterine surgical procedure.
- Smoking.
- Deficient endometrium (uterine scar, endometritis, manual removal of placenta, curettage, submucous fibroid).

Risk factors for uterine rupture include:

- Previous uterine surgery.
- High parity.
- Trauma.
- Injudicious use of ecbolics.

Risk factors for vasa previa include:

- Low lying placenta.
- Succenturiate lobe.

Prediction and prevention of APH:

- APH can not reliably be predicted, 70% of cases of placental abruption occur in low-risk pregnancies.
- Women should be advised, encouraged and helped to change modifiable risk factors (such as smoking and drug misuse).
- Folic acid during pregnancy reduces the risk of placental abruption (no conclusive evidence).
- There are insufficient data to support a role for antithrombotic therapy (low dose aspirin and/or low molecular weight heparin) in the prevention of abruption in women with thrombophilia.
- It is considered good practice to avoid vaginal and rectal examinations in women with placenta previa (PP), and to advise these women to avoid penetrative sexual intercourse.
- Asymptomatic women with placenta previa should be advised to avoid moderate and strenuous exercise, heavy lifting, or standing for prolonged periods of time.
- Women receiving antenatal anticoagulant therapy should be advised that if they have any vaginal bleeding, they should not take any more doses. They should attend hospital to be

assessed and further doses should only be administered after consultation with medical staff (expert hematological advice should be sought).

Initial Assessment:

- Women with APH presenting to a primary health unit or to a hospital with limited resources should be assessed, stabilized if necessary and transferred to a hospital maternity unit with facilities for resuscitation and performing emergency operative delivery (such as anesthetic support and blood transfusion resources).
- The role of initial assessment in women with APH is to establish whether urgent intervention is required to manage maternal or fetal compromise. The process of triage includes history taking to assess coexisting symptoms such as pain, an assessment of the extent of vaginal bleeding, an assessment of the cardiovascular condition of the mother, and an assessment of fetal wellbeing.
- Women presenting with a major or massive APH that is persisting or if the woman is unable to provide a history due to a compromised clinical state, a rapid assessment of maternal wellbeing should be performed and resuscitation started immediately. **The mother is the priority** in these situations and should be stabilized prior to establishing the fetal condition. She should be resuscitated and stabilized before any decision is made regarding delivery of the baby.

History :

If there is no maternal compromise, a full history should be taken:

- Amount of bleeding

  The amount of vaginal bleeding may not be a reliable indicator of the severity of hemorrhage since bleeding may be retained in the uterine cavity in case of concealed placental abruption.

- Pain
  - Mild to moderate abdominal pain is usually present with placental abruption. Back pain is prominent when the placenta is on the posterior uterine wall. Placental abruption should be considered when the pain is continuous.
  - Labor should be considered if the pain is intermittent.
  - Placenta previa is characterized by painless bleeding.
  - The sudden onset of constant sharp pain may be a presenting feature of uterine rupture.
- Associated or initiating factors
  - If the APH is associated with rupture of membranes, bleeding from a ruptured vasa previa should be considered
  - Symptomatic pregnant women with cervical cancer usually present with APH (mostly postcoital)
  - Abdominal trauma (placental abruption, uterine rupture). The possibility of abruption should always be considered in pregnant women who are being evaluated for trauma (e.g. motor vehicle crash, fall, domestic violence).
  - Rapid uterine decompression, such as after uncontrolled rupture of membranes in the setting of polyhydramnios or after delivery of a first twin (placental abruption).

- Fetal movements
- Previous episodes of vaginal bleeding during the present pregnancy
- Position of the placenta, if known from a previous scan
- Obstetric history (gestational age, previous abruption or PP)
- Previous uterine surgery. (The possibility of uterine rupture should always be considered in women with APH and a previous cesarean delivery or any other uterine surgery).
- Risk factors for placental abruption and placenta previa should be identified
- History of previous cervical smear to exclude neoplastic lesions of the cervix

Examination :

Examination of the woman should be performed to assess the amount and cause of APH

General examination

- Blood pressure
- Pulse
- Other signs of hemodynamic compromise (pallor, cyanosis)
- Respiratory rate
- Temperature

Abdominal examination

- The woman should be assessed for tenderness or signs of an acute abdomen. The tense or woody feel to the uterus on abdominal palpation indicates a significant abruption. Abdominal palpation may also reveal uterine contractions.
- A soft, non-tender uterus may suggest a lower genital tract cause or bleeding from placenta or vasa previa.
- High presenting part or transverse lie raise the suspicion of placenta previa. Also, a high or unreachable presenting part may be a presenting feature of uterine rupture.
- In cases of placental abruption, fetal compromise may be noted during cardiotocography (CTG). Abnormal or pathologic CTG may be a presenting feature of uterine rupture. Acute fetal compromise with no maternal shock suggests vasa previa.

Vaginal examination

- A speculum examination must be carried out on the first occasion a woman presents with even a small amount of vaginal bleeding or spotting in pregnancy, to ensure that the cervix is seen and cervical malignancy is excluded. Speculum examination on subsequent occasions can be useful to identify cervical dilatation or visualize a lower genital tract cause for the APH.
- Digital vaginal examination should not be performed until an ultrasound has excluded placenta previa (No PV until No PP). Digital vaginal examination can provide information on cervical dilatation if APH is associated with pain or uterine activity.

Ultrasound examination

- In all women presenting with APH, an ultrasound scan should be performed to confirm or exclude placenta previa. Ultrasound has limited sensitivity in the identification of retroplacental hemorrhage (placental abruption is a clinical diagnosis). However, when the ultrasound suggests an abruption, the likelihood that there is an abruption is high.

Maternal investigations:

Minor hemorrhage

- Full blood count.
- Group and save.
- A coagulation profile is not indicated unless the platelet count is low.

Major or massive hemorrhage

- Cross-matching of 4 units
- Full blood count
- Coagulation profile
- Urea and electrolytes
- Liver function tests

Fetal assessment:

- Ultrasound should be carried out to establish fetal heart pulsation if fetal viability can not be detected using external auscultation.
- CTG should be performed in women presenting with APH once the mother is stable or resuscitation has commenced, to aid decision making on the time and mode of delivery.

- In case of suspected vasa previa, various tests to differentiate between fetal and maternal blood are available, but are often not applicable. Fetal compromise would be detected on CTG and delivery would be indicated irrespective of the test result and without delay.

Following initial assessment, women will fall into one of two categories:

I) Bleeding heavy and continuing or clinical shock (major or massive APH)

Mother or fetus is or soon will be compromised.

II) Bleeding mild or settling (minor APH)

Neither mother nor fetus is compromised

I. Management of major/massive APH:

The major goals of management are to achieve and maintain maternal hemodynamic stability (resuscitation) and to treat the underlying cause in order to stop bleeding. A multidisciplinary team involving senior staff (including the anesthetic team and laboratory specialists) should be involved.

A) Resuscitation

The cornerstones of resuscitation are restoration of both blood volume and oxygen-carrying capacity.

- A and B - assess airway and breathing

  High flow oxygen (10-15 l/min) via a facemask should be administered, regardless of maternal oxygen saturation. If the airway is compromised owing to impaired conscious level, anesthetic assistance should be sought urgently.

- Left lateral tilt to relieve venocaval compression
- Keep the woman warm
- Immediate venepuncture (20 ml) for:
  - Cross-match (4 units minimum)
  - FBC
  - Coagulation screen, including fibrinogen
  - Renal and liver function for baseline
- Continuous recording of pulse, BP and respiratory rate (using oximeter, electrocardiogram and automated BP recording). Monitor temperature every 15 minutes.
- Foley catheter to monitor urine output.
- Establish two 14-gauge IV lines.
- Fluid replacement: until blood is available, infuse up to 3.5 L of warmed fluids, initially 2 L of isotonic crystalloids. Further fluid resuscitation can continue with additional isotonic crystalloids or colloids (succinylated gelatin, gelofusine). Hydroxyethyl starch (voluven) should not be used. The nature of the infused fluid is of less importance than rapid administration and warming of the infusion.
- Blood transfusion: compatible blood (supplied in the form of concentrate) should be transfused as soon as possible. Patients with acute hemorrhage can have normal Hb. Therefore, the clinical picture should be the main determinant of the need for blood transfusion and time should not be unnecessarily spent awaiting laboratory results. The goal is to maintain Hb greater than 8 gm%. Special filters should not be used as they slow infusions. If immediate transfusion is indicated, give group O, RhD-negative red cell units. Switch to group-specific red cells as soon as feasible.

- Fresh frozen plasma: transfuse 4 FFP units for every 4 units of red cell concentrate if:
  - hemorrhage is continuing and the results of hemostatic tests are not available
  - PT/APTT are prolonged
  - coagulopathy is suspected (placental abruption)
- Platelets: transfuse platelets at a trigger of 75000/microliter to maintain a level greater than 50000/microliter during ongoing APH.
- Cryoprecipitate should be used to maintain a fibrinogen level of greater than 2 g/l, even if PT/APTT are normal. Two cryoprecipitate pools (each pool is composed of 5 units) may be given in the face of relentless bleeding, while awaiting the results of the coagulation studies.
- Consider transfer to ICU once the bleeding is controlled or monitoring at high dependency unit on delivery suite, if appropriate.

B) Arrest of bleeding

- Delivery through emergency CS in most of the cases, unless vaginal delivery is imminent.
- In case of placental abruption with IUFD, vaginal delivery is recommended unless vaginal delivery is not imminent and rapid control of bleeding is required because of maternal hemodynamic instability or significant coagulopathy.

Beware of DIC and renal failure, especially with placental abruption.

II. Management of minor APH:

- Immediate venipuncture (20 ml) for:
  - group and save

- FBC
- coagulation screen (if platelet count is low)

- IV access (one 14-gauge cannula)
- Pulse, BP and respiratory rate recording every 15 minutes
- Start warmed crystalloid infusion
- Ultrasound to confirm placental location and to establish fetal well-being (growth, amniotic fluid volume and doppler measurements).
- Delivery

  Deliver the patient if she is 37 weeks or more or if there is fetal distress or congenital anomalies that are incompatible with life or if the baby is dead.

- Expectant management

  If the bleeding stops and the maternofetal condition is stable, expectant management should be the option of choice to achieve fetal maturity.

  - Prevention and treatment of anemia is recommended
  - Steroids should be given if gestational age is <36 weeks (unless the cause is lower genital tract bleeding and imminent delivery is unlikely)
  - Anti-D Ig is recommended for Rh(D)-negative women after any presentation with APH, independent of whether routine antenatal prophylactic anti-D has been administered. In the event of recurrent vaginal bleeding after 20 weeks of gestation, anti-D Ig should be given at minimum of 6-weekly intervals.
  - Do not use prophylactic tocolytics in women with APH.

- In cases of placenta previa, cervical cerclage to prevent or reduce bleeding and prolong pregnancy is not supported by sufficient evidence to recommend this practice outside a clinical trial.
- Women with pregnancies complicated by APH (including unexplained APH) are at increased risk of adverse perinatal outcomes including small for gestational age fetus and fetal growth restriction. Serial ultrasound for fetal growth should be performed.
- Home-based care

  All women with APH heavier than spotting and women with ongoing bleeding should remain in hospital at least until the bleeding has stopped.

  At present, there is no evidence to support recommendations regarding duration of inpatient management following APH. Each woman should be assessed on an individual basis and clinical judgment applied. Where the bleeding has been spotting and has settled, and tests of fetal and maternal wellbeing are reassuring, the woman can go home. She should be encouraged to contact the maternity unit if she has any further bleeding, pain or a reduction in fetal movements.

  Any home-based care requires:

  1. Close proximity to the hospital
  2. Ready access to the hospital
  3. The constant presence of a companion
  4. Full informed consent by the woman

  Women managed at home should attend immediately if there is bleeding, contractions or any pain. They are advised to avoid excess physical activity, including sexual intercourse.

- Timing of delivery:
  - Delivery timing should be tailored according to antenatal symptoms and, for women presenting with uncomplicated placenta previa, delivery should be considered between 36^{+0} and 37^{+0} weeks of gestation.
  - Late preterm (34^{+0} to 36^{+0} weeks of gestation) delivery should be considered for women presenting with placenta previa or a low-lying placenta and a history of vaginal bleeding or other associated risk factors for preterm delivery.
  - In the absence of risk factors for preterm delivery in women with placenta accreta spectrum, planned delivery at 35^{+0} to 36^{+6} weeks of gestation provides the best balance between fetal maturity and the risk of unscheduled delivery.
  - With placental abruption, delivery is recommended at 37-38 weeks of gestation.

Postnatal Management:

- Postpartum hemorrhage should be anticipated in women who have experienced APH. Therefore, active management of the third stage of labor should be strongly advised. Consideration should be given to the use of oxytocin and ergometrine combined to manage the third stage in women with APH resulting from placental abruption or placenta previa in the absence of hypertension. A prophylactic oxytocin 40 IU in 500 ml infusion over 4 hours (125 ml/hour) should also be considered for the postnatal period.
- Anti-D Ig should be given to non-sensitized RhD-negative women.
- In women with pregnancies complicated by major or massive APH, thromboprophylaxis should be administered once the bleeding is controlled and any coagulopathy is corrected, as there is a high risk of thrombosis. If chemical thromboprophylaxis is contraindicated (e.g. in case of thrombocytopenia), anti-embolism stockings, foot impulse devices or intermittent pneumatic compression devices can be used.

Anesthesia:

- In a case of APH where maternal and/or fetal condition is compromised and cesarean section is required, general anesthesia should be considered to facilitate control of maternal resuscitation and to expedite delivery.

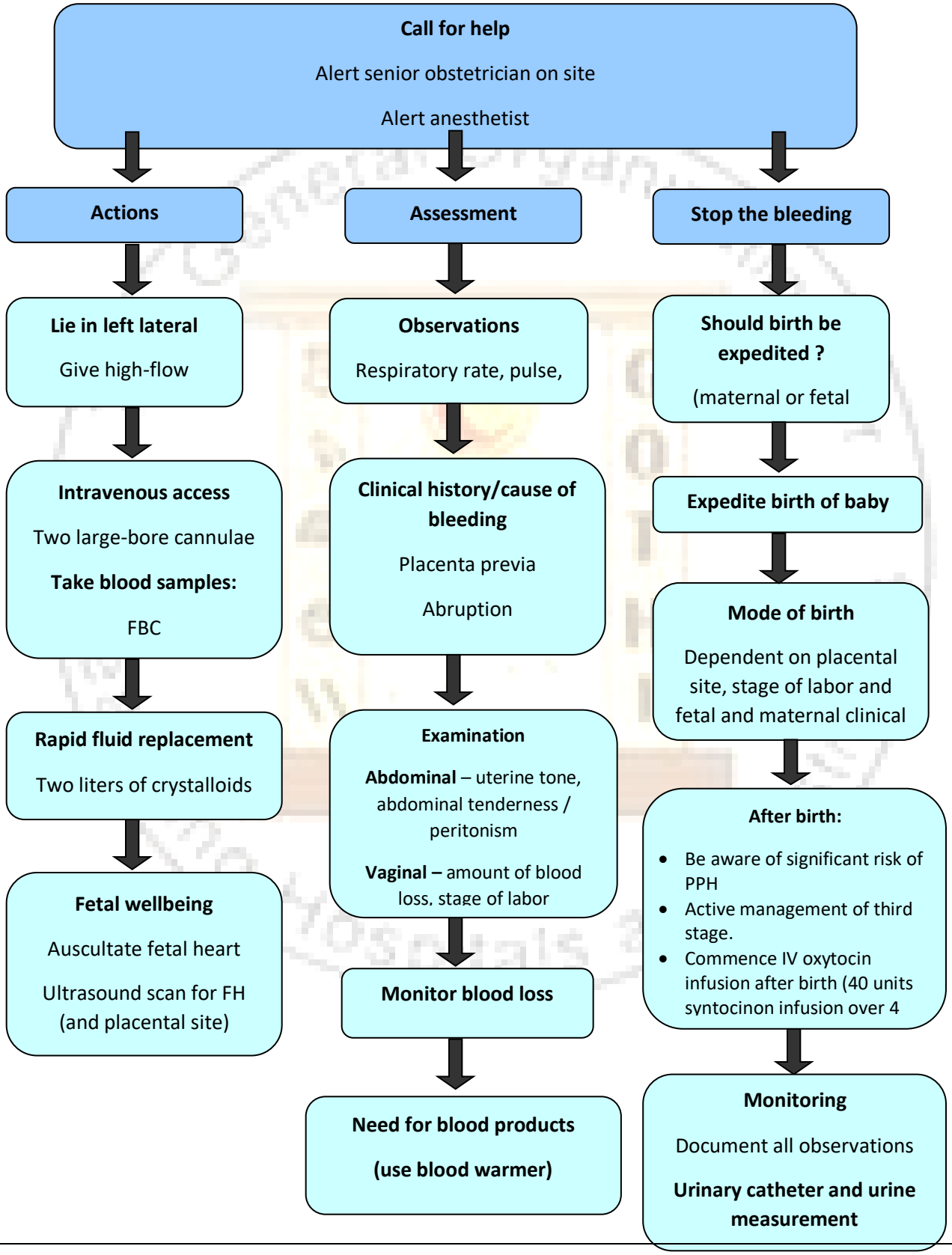
Neonatal care:

- Major or massive APH may result in fetal anemia and fetal compromise. The neonate should be assessed by a senior neonatologist.
- In minor APH, clinical judgment should be used. With continuing hemorrhage, it would be appropriate to request neonatal support at the time of delivery.
- Anterior placenta previa that necessitates incising the placenta at the time of cesarean section is an indication for attendance by an experienced neonatologist.

Appendix I: Maternal and Fetal Complications of APH:

Maternal complications	Fetal complications
1. Maternal shock 2. Postpartum hemorrhage. 3. Complications of blood transfusion. 4. Consumptive coagulopathy (especially with placental abruption). 5. Renal tubular necrosis (especially with placental abruption). 6. Infection. 7. Prolonged hospital stay. 8. Anemia. 9. Psychological sequelae	1. Fetal hypoxia. 2. Small for gestational age and fetal growth restriction. 3. Prematurity (iatrogenic and spontaneous). 4. Fetal death.

Appendix II: Algorithm for the Management of antepartum haemorrhage



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ENT Critical Condition Curriculum for residence

Advisory Board for Ear, Nose and Throat Surgery (GOTHI)

DEFINITION: *Any condition where a misdiagnosis can be associated with devastating consequences for life or permanent disability.*

Critical Conditions -

Otolaryngology manages a large number of individual conditions as described in the syllabus. Assessment of a trainee's ability to manage these is through the supervision level decisions made when assessing the shared CiPs. Otolaryngology also has a list of critical conditions, which are of significant importance for patient safety and to demonstrate a safe breadth of practice. These critical conditions will be assessed individually by means of the Case Based Assessment (CBD) and Clinical Evaluation Exercise (CEX), which will both provide formative feedback to the trainee and feed into the summative assessments of the AES and ARCP. To ensure that trainees have the necessary skills in the critical conditions,

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Critical Conditions - Otolaryngology

Otolaryngology has a list of critical conditions, which are of significant importance for patient safety and to demonstrate a safe breadth of practice. ***Such as***

1) Adult airway obstruction (malignancy, inhalation injury etc.)

2) Paediatric airway obstruction

3) Upper aero-digestive tract foreign body and chemical injury (including batteries)

4) Complicated infections of the upper aero-digestive tract including complicated sinusitis, complicated ear infections.

5) Deep neck space abscess and necrotizing fasciitis

6) Blunt and penetrating trauma to the neck

7) Epistaxis

8)Nasal trauma and deformity

9) Acute Facial Palsy

10)Tracheostomy Care Module

11) Management of tonsillar hemorrhage.

Description

The trainee should be able to diagnose the critical ENT cases, to perform the primary safe management with full assessment, referring for specialist advice when necessaryand detect when it needs higher level of management with proper reporting to more senior consultant.

• Demonstrates effective communication with colleagues, patients and relatives

• Makes appropriate peri- and post-operative management plans in conjunction with anaesthetic colleagues

• Delivers ongoing post-operative surgical care in ward and critical care settings, recognising and appropriately managing medical and surgical complications, and referring for specialist care when necessary

• Makes appropriate discharge and follow up arrangements

• Carries out all operative procedures as described in the syllabus

• Manages potentially difficult or challenging interpersonal situations

• Gives and receives appropriate handover

Supervision levels:

Level I: Able to observe only

Level II: Able and trusted to act with direct supervision:

a) Supervisor present throughout

b) Supervisor present for part

Level III: Able and trusted to act with indirect supervision

Level IV: Able and trusted to act at the level expected of a day-one consultant

Level V: Able and trusted to act at a level beyond that expected of a day-one consultant

1. Adult airway obstruction (malignancy, inhalation injury etc.)

Topic	*Airway obstruction in adults*
Category	Head and Neck
Sub-category:	None
Objective	*To understand the aetiology, presenting signs, symptoms and management of patients presenting with upper airway disorders in the emergency in adults.*
Knowledge	Demonstrate a detailed knowledge of the anatomy & physiology of the larynx, trachea, pharynx and oral cavity Understand the microbiology and pathology of disorders of the upper aerodigestive tract. Understand the classification of diseases that may present with airway obstruction. Understand the principles of patient management of patients presenting with airway obstruction. Know the different methods of securing an airway safely (surgical &non surgical) in an emergency setting Understand the indications & techniques for emergency tracheostomy Understand the principles of the use of cricothyroidotomy and tracheostomy during a Can't Intubate, Can't Oxygenate Event.
Technical Skills and Procedures	Be able to elicit an appropriate clinical history and correctly interpret physical signs. Be aware of the role of appropriate investigation in the management of airway obstruction Demonstrate the ability to work effectively with anaesthetists and those involved in critical care who manage the 'shared airway'. Demonstrate expertise in the safe assessment of patients with critical airways.
Technical Skills and Procedures	Be competent at performing the following diagnostic procedures; fibreopticnasopharyngoscopy, direct laryngoscopy, Be competent at performing endotracheal intubation Be proficient at performing a surgical tracheostomy in the elective & emergency setting both under general and local anaesthesia Percutaneous tracheostomy Be competent at foreign body removal from the airway in adults Tracheostomy change Emergency Front of Neck Airway procedures including cricothyroidotomy and tracheostomy

2. Pediatric airway obstruction

Topic	Airway pathology in childhood
Category	Pediatric Otolaryngology
Sub-category:	Airway Disorders
Objective	Safe recognition of the main patterns of presentations and likely etiologies of children with airway obstruction at birth, in infancy and in later childhood. Includes primary management to enable definitive treatment of main conditions.
Knowledge	Anatomy of the pediatric airway, and differences between the adult and child. Physiology of airway obstruction Clinical features of airway obstruction Clinical measures to determine severity of obstruction Know the causes, presenting symptoms of airway pathology in children, Know the treatment options and natural history of main conditions causing airway pathology in children at different ages e.g. laryngomalacia, vocal cord palsy, subglottic cysts, haemangioma, RRP, Laryngeal cleft, tracheobronchomalacia, acute epiglottitis and laryngotracheobronchitis (croup). Understand the genetic disorders associated with airway pathology in children
Technical Skills and Procedures	Paediatric flexible pharyngolaryngoscopy in the outpatients Paediatric tracheostomy emergency and elective Paediatric tracheostomy care including tube change

3.Upper aero-digestive tract foreign body and chemical injury (including batteries)

Topic	**Foreign bodies in the ear canal and Upper aero-digestive tract**
Category	Pediatric Otolaryngology
Sub-category:	Foreign bodies in the ear nose and throat
Objective	Safe definitive management of children with suspected and actual foreign bodies in the ear nose and pharynx; primary management of inhaled foreign bodies to facilitate safe transfer for tracheobronchoscopy if required.
Knowledge	Anatomy and physiology of the pediatric airway Recognition of anatomical differences between the adult and pediatric airway. Recognition of the clinical features of foreign bodies in the ear, nose, and throat Knowledge of the natural history and the complications associated with foreign bodies. Concept of the shared airway and differing anaesthetic techniques
Clinical Skills	HISTORY AND EXAMINATION Ability to take a thorough history from the child/carer Otoscopy&Anterior rhinoscopy Flexible pharyngolaryngoscopy DATA INTERPRETATION
Technical Skills and Procedures	Otomicroscopy and removal of foreign body Removal of nasal foreign body and examination with paediatric and rigid scopes Pharyngo-oesophagoscopy and foreign body removal

4. Complicated infections of the upper aero-digestive tract including complicated sinusitis, complicated ear infections.

Topic	**Rhinosinusitis; orbital and intracranial complications of rhinosinusitis**
Objective	Optimum recognition and management of patient with rhinosinusitis; particularly complicated sinus disease e.g. subperiosteal abscess, intracranial sepsis.
Clinical Skills	HISTORY AND EXAMINATION Ability to take a thorough history Anterior Rhinoscopy Flexible Nasendoscopy.Otoscopy DATA INTERPRETATION Awareness of imaging techniques Assessment of abnormalities on CT scanning of the paranasal sinuses and MR brain. PATIENT MANAGEMENT Medical and surgical management of rhinosinusitis and its complications.
Technical Skills and Procedures	Initial medical treatment and proper selection of the diagnostic tool proper reporting to more senior consultant.

5.Deep neck space abscess and necrotising fasciitis

Topic	**Etiology and management of cervical sepsis**
Objective	*To understand the aetiology, presenting signs, symptoms and management of a patient with cervical sepsis.*
Knowledge	Know the anatomy of the fascial compartments of the neck. Understand the pathogenesis(including congenital abnormalities) and clinical presentation of deep neck space infections. Know the microbiology of deep neck space infections. Understand the principles of medical and surgical management of deep neck space infection, including image guided drainage procedures. Understand the complications of deep neck space infections and their management.
Clinical Skills	Be able to elicit an appropriate history from a patient with deep cervical sepsis. Be able to demonstrate the relevant clinical signs from a patient with deep cervical sepsis. Be able to order and interpret the results of appropriate investigations, including imaging and microbiological cultures, in a patient with deep cervical sepsis. Be able to undertake treatment of a patient with deep cervical sepsis or complications thereof.
Technical Skills and Procedures	rigid endoscopic examination of the upper aerodigestive tract proper management of the compromised upper airway in deep cervical sepsis, including tracheostomy. Manage the patient in conjunction with anaesthetists/intensivists knowing the needof incision and drainage of a deep cervical abscess, as well as demonstrating awareness of the complications of such procedures.

6. Blunt and penetrating trauma to the neck

Topic	**Trauma to the ear, upper aero digestive tract and neck**
Knowledge	Anatomy of the head and neck Mechanisms of trauma to the facial skeleton and soft tissues Know the causes and presentation of nasal septal haematoma Know the causes and presentation of ear trauma (external, middle and inner) Know the causes and presentation of trauma to the neck, pharynx and larynx Knowledge of common aetiologies and awareness of the possible presentations of non-accidental injury to the ENT department.
Clinical Skills	HISTORY AND EXAMINATION Ability to take a thorough history Assessment of the external nose and nasal airway Clinical examination of the ear Assessment of the neck including the airway Recognition of the signs of respiratory distress Resuscitation of a patient in hypovolaemic shock secondary to bleeding
Technical Skills and Procedures	Nasal fracture manipulation Laryngoscopy, Pharyngoscopy Drainage of septal haematoma Drainage of haematoma of pinna Pediatric Tracheostomy

7. *Epistaxis*

A.Epistaxis in a child

Topic	**Epistaxis in a child**
Objective	Optimum recognition and management of children with epistaxis;
Knowledge	Nasal anatomy & physiology Pathophysiology, epidemiology, & natural history of paediatric epistaxis Understand the aetiologies of paediatric epistaxis (local including nasopharyngeal angiofibroma, and systemic including coagulopathies) Know the relevant investigation and treatments of paediatric epistaxis
Clinical Skills	HISTORY AND EXAMINATION Ability to take a thorough history from the child/carer Anterior Rhinoscopy Flexible Nasendoscopy DATA INTERPRETATION Interpretation of full blood count & other haematological investigations; awareness of significance of coagulation tests PATIENT MANAGEMENT
Technical Skills and Procedures	Nasal cautery Appropriate nasal packing in a child

B. Epistaxis in adult

Topic	Epistaxis
Objective	*To understand the aetiology, presenting symptoms and signs and management of epistaxis.*
Knowledge	Know the anatomy of the nose Understanding of local and systemic aetiologies of epistaxes Detailed knowledge of the anatomy and physiology of nasal vasculature Detailed understanding of the presenting symptoms and signs of epistaxes Detailed knowledge of management including first aid measures, nasal cautery, packing and operative techniques in the management of epistaxes Know the complications of epistaxes and the management of them. Understanding of the role of radiology and embolization in managing epistaxis
Clinical Skills	Demonstrate expertise in taking an appropriate clinical history. Ability to elicit physical signs both local and systemic if appropriate Awareness of relevant haematological and imaging investigations. Awareness of management principles in patient with epistaxis Ability to resuscitate critically ill patient
Technical Skills and Procedures	Diagnostic nasendoscopy Packing of nose Removal of nasal packing Cautery of nasal septum Ethmoid Artery ligation Sphenopalatine artery ligation Maxillary artery ligation External Carotid artery ligation Approach to ICA epistaxis

8. **Nasal trauma and deformity**

Topic	**Nasal trauma and deformity**
Category	Rhinology
Sub-category:	None
Objective	*To understand the presenting features, diagnosis, complications and management of nasal trauma and deformity. This module gives some idea of the breadth and depth of required knowledge and surgical skills. This list should not be considered to be fully inclusive or exhaustive.*
Knowledge	Know the anatomy of the nose, paranasal sinuses and facial skeleton. Understanding of the mechanisms of trauma responsible for nasal and facial injuries. Understanding of objective assessment of airway e.g. rhinomanometry Knowledge of the appropriate imaging techniques Knowledge of the specific complications of nasal trauma Knowledge of the management of nasal trauma Knowledge of the management of nasal deformity Glasgow Coma Scale
Clinical Skills	Ability to take a relevant history and perform an appropriate clinical examination Knowledge of the relevant special investigations and correct interpretation egrhinomanometry Ability to adequately resuscitate the critically ill patient
Technical Skills and Procedures	Fracture nose reduction Insertion septal button Packing of nose Management of traumatically induced epistaxis (see epistaxis section) Septoplasty Septorhinoplasty Surgical repair septal perforation-open and endonasal

9. Acute Facial Palsy

Topic	**Facial palsy**
Category	Otology
Sub-category:	Facial Paralysis
Objective	*To understand the aetiology, presenting signs, symptoms and management of facial nerve palsy. This module gives some indication of the breadth and depth of required knowledge, clinical and surgical skills. This list should not be considered to be fully inclusive or exhaustive*
Knowledge	The anatomy and physiology of facial nerve and related structures The aetiology, classification and neuro-physiology of facial paralysis Indications for investigations including radiology, electrophysiology and laboratory tests. Facial nerve grading Management of acute and chronic facial nerve palsy Management and prevention of ocular complications Principles of peri-operative facial nerve monitoring Principles of rehabilitation for facial paralysis
Clinical Skills	HISTORY AND EXAMINATION Obtain appropriate history Clinical examination including assessment of facial nerve function Otoscopy DATA INTERPRETATION Neuro-physiological tests of inner ear function and facial nerve Interpretation of radiological tests Interpretation of laboratory investigations PATIENT MANAGEMENT Demonstrate communication skills and empathy Appreciate the psychological effects of facial disfigurement Be able to advise the patient of the treatment options, and liaise with other health care professionals.
Technical Skills and Procedures	Setup and use of intra-operative facial nerve monitor Cortical mastoidectomy Modified radical mastoidectomy Full decompression of facial nerve Facial nerve anastomosis Resection of facial neuroma

10- Tracheostomy Care Module

Topic	**Tracheostomy Care Module**
Objective	*To be able to manage patients with short and long term tracheostomies in an emergency, elective & community setting and provide an expert resource to other health professionals in the management of tracheostomies*
Knowledge	Anatomy of larynx, trachea and neck Physiology of respiration Indications for tracheostomy In depth knowledge of different types of tracheostomy tubes and relative indications for use Role of health professionals in the multidisciplinary management of patients with tracheostomy Indications for surgical & percutaneous tracheostomy Principles of weaning
Clinical Skills	Tracheostomy care; suction, inner tube care, humidification Appropriate selection of correct tube to suit patient Supervision of weaning and extubation Troubleshooting in a variety of situations Management of persistent trachea cutaneous fistula Management of patients with failed extubation Multi-disciplinary management of patients with long term tracheostomy tubes
Technical Skills and Procedures	Flexible nasendoscopy Management of blocked & displaced tube Tracheostomy change

11)Post-Tonsillectomy Bleeding

Description: Post-tonsillectomy bleeding is thought to occur in approximately 5% of cases following tonsil surgery. A bleed in the first 24 hours is considered a PRIMARY bleed and those occurring after 24 hours are a SECONDARY bleed (most frequently in days 5-9, up to 28 days). The majority of post-tonsillectomy bleeds will be minor and self-limiting. However, small bleeds (so-called "herald bleeds") can precede a more severe hemorrhage in the following 24 hours and consequently all reports of bleeding should be taken very seriously. In its most serious form, post-tonsillectomy bleeding can cause hemorrhagic shock and aspiration, requiring an urgent return to the operating theatre to control.

Red Flags:

• Young patients (less than 18 years old) compensate haemodynamically, and may deteriorate rapidly

• Primary bleeds often require a return to theatre

• Consider coagulopathy if there is excessive and/or recurrent bleeding. Von Willebrand Factor disease is the most common congenital coagulopathy.

• Airway compromise (especially pre-existing in patients with Obstructive Sleep Apnea Syndrome)

How to Assess: Phone Call Advice (ENT Admitting Officer):

If you are called by a patient/relative or a GP about bleeding, respond as follows:

1. If actively bleeding, advise going urgently to the nearest ED, preferably with ENT cover.

2. If bleeding has stopped, it may be safe to advise close monitoring at home for another 24 hours. Sucking ice can help if the bleeding has stopped or is minimal. If bleeding increases or recurs, advise going urgently to the nearest ED, preferably with ENT cover.

History:

Bleeding

• Active, heavy or light, intermittent?

• Ask patient about the amount of blood and when it started. A patient may present with active bleeding or with a history of recent bleeding, e.g. coughing blood or seeing blood on their pillow.

-Try to estimate the blood loss (e.g. teaspoon, egg cup). This may be difficult, as blood may have been swallowed.

• In children, a higher degree of care is needed, as excessive or difficulty swallowing may be the only clue to bleeding

• Some patients may vomit a small amount of dark, altered blood during the first couple of days, which may in fact represent blood swallowed during surgery and not a new bleed

• Find out who performed the operation, when and where it took place. Notify the surgeon about bleed if possible.

Examination:

• Wear gloves/gown and protective eye wear as per standard precautions • Check ABC: is patient hemodynamically stable or in shock?

• Remain calm and reassure the patient

• Look for a clot on the tonsillar bed, check meticulously for any slow bleeding

• A sloughy, white appearance is normal after tonsillectomy

Acute Management:

If bleeding actively:

• Notify ENT registrar

• Immediate and continuous haemodynamic monitoring

• Consider calling **a CODE BLUE** if patient is unstable – summon senior anaesthetic/ENT assistance – this is a difficult airway situation

• May also need to call theatre to organise urgent theatre (including anaesthetic/ENT consultant)

• High flow oxygen if tolerated

• Sit the patient in an upright position to facilitate the removal of blood

• Insert large bore intravenous access and take blood for full blood count, urea and electrolytes and group and hold. If unstable, urgent cross-match. Patient may need coagulation profile.

• Intravenous fluids (in children 20mls/kg as initial bolus), analgesia and antiemetics

- Analgesia: oral or IV paracetamol regularly. Avoid NSAIDs.

• Establish when the patient last ate and drank. Ensure the patient remains nil by mouth.

• **Tonsil procedure:**

- If you can visualise the bleeding spot (i.e. right vs. left tonsillar fossa/superior vs. inferior), firmly apply a large cotton swab stick soaked in 1:10,000 adrenaline

- Alternatively, silver nitrite cautery may be attempted on the bleeding spot following application of topical Cophenylcaine® spray. This should be performed with ENT assistance.

NOTE:

Both methods may stimulate the gag reflex and should only be attempted by an experienced operator. Both these measures should NOT delay theatre if required.

- If blood clot is visible on tonsillar bed:

▪ Leave it if patient has only had one small bleed and is not actively bleeding

▪ Remove/suction it out if there has been recurrent bleeding or if actively bleeding in order to visualise the bleeding spot. However, this can cause profuse bleeding, so only perform with ENT assistance and prepare in advance necessary equipment (silver nitrate cautery, bipolar cautery, suction, resuscitation equipment) and address potential need for urgent theatre.

Self-terminated bleeding:

• Examine the patient thoroughly

• Tonsil procedure

- If blood clot is visible on tonsillar bed, leave it if the patient has had a single small bleed and is not actively bleeding

- Remove/suction the clot if there has been recurrent bleeding. This should be performed with ENT assistance and with IV access in situ as per 'If bleeding actively' section.

Further management:

• Patient usually should be admitted for 24hrs to observe for further bleeding

• Consider the use of 3% H2O2 dilute in 1-2x volume of water as four hourly mouth washes if bleeding slowly or has stopped (the benefit of this has not been established)

• The use of antibiotics is not necessarily indicated for the treatment of all post tonsillectomy secondary hemorrhage patients. Routine use of antibiotics in patients who do not have clear features of infection (e.g. pyrexia, raised white cell count or C reactive protein) remains uncertain.

• Tranexamic acid – there is no direct evidence for its use in post-tonsillectomy bleeding but there is strong evidence that it reduces the need for transfusion in surgical bleeding in general. Dosage: 1 gramtranexamic acid via IV infusion.

Instructions for the use of logbook

Aim of the logbook

The purpose of the logbook is to provide one source of evidence for the ENT scientific council that you have attained the desired level of competency required for licensure. It is the place where you are going to document experiences and operations you performed during your training.

The logbook is divided into several sections. These instructions will help you completing those sections correctly.

Personnel information

Please fill in all your personnel information required . This will help the trainee Administrators to process your logbook during scientific council evaluation yearly and finally before sitting for the final exam. Your personnel photo should be attached to the logbook and you should sign the personnel information page

First year logbook

You will spend the first year of training in rotations related to general surgery, emergency and ICU. You will find appropriate tables to report the patients you have seen and the procedures you assisted in during this period. Your level of participation in each case or procedure must be approved and signed by your trainer.

Second to fifth year logbook

From the second to the fifth year of training you will be dedicated to otolaryn-gology services. Your logbook is divided into:

- Pediatric otolaryngology
- Head and neck surgery
- Rhinology
- Otology

In each section you must report the cases you managed and the operative proce-dures you performed. Your level of participation must be defined and **your trainer must sign clearly each activity**

Academic activities

1. Academic activities that must be documented in the logbook are lectures, journal clubs, morbidity and mortality conferences, and workshops or other conferences attended.

2. Workshops and conferences tables are the place where you will record your CME activities whether inside or outside the training center. Any attended activity must be signed by the workshop or conference organizer/coordinator

Annual summary table

From the second year of training you are requested to provide documented sum-mary of all operative activities you participated in as assistant or first surgeon. The tables are present in the last chapter of the logbook and should be signed by your trainer and educational supervisor

Assessment of logbook activities

1. Your trainer will assess your logbook weekly for completion and provide feedback

2. Your educational supervisor will assess your logbook monthly or every two months, provide verbal or written feedback and counter sign impor-tant activities

3. The examination committee of the council will revise your logbook:

A) Annually before your progress from one year of training to another

B) At the end of training before the final exam

To be noted that unsatisfactory completion of the logbook would lead to delay of training progression. Unsatisfactory logbook at the end of training will prevent you from entering the final exam

Important Notice: It is your responsibility to maintain accurate and completed logbook and to regularly update your records. Shall you meet any difficulty; you must con-tact your trainer or your specialty administrator at the Egyptian Fellowship Board.

Important notice for various categories of procedures

The scientific council has divided operative procedures into categories; mi-nor, moderate and major. This categories are presented as tables in each rele-vant section in the logbook.

- **Over all , a minimum of: 100 minor, 70 moderate and 30 major surgeries must be performed by the candi-date as assistant or first surgeon**
- **In minor and moderate categories: the same operation should not be repeated to the extent of exceeding 40% of the total in the same category.**

Status Epilepticus

•Seizure definition:

It's transient occurrence of signs and/or symptoms resulting from abnormal excessive neuronal activity in the brain.

•Epilepsy:

- ≥2 unprovoked seizures occurring >24 hours apart
- 1 unprovoked seizure and probability of further seizure in ≥60%

•Seizures are divided into:

- Focal aware seizure
- Focal seizures with impaired awareness
- generalized (motor or non-motor)

•Status epilepticus (SE):

It's a medical emergency that is defined as continuous or recurrent seizure activity without regaining consciousness lasting for > 5 minutes.

Treatment should be initiated within the first 5 minutes and neurological sequelae are anticipated after 30 minutes.

•Refractory status epilepticus:

It's failure to respond to therapy (30 min), usually with at least two medications.

• Super refractory status epilepticus:

It's SE that has failed to resolve, or recurs, within 24 hours or more despite therapy that includes a continuous infusion such as midazolam and/or pentobarbital.

Devastating epileptic encephalopathy in school-age children (DESC), also called fever-induced refractory epileptic encephalopathy in school age children (FIRES), is a syndrome of refractory SE that is associated with acute febrile infections, appears to be Para-infectious in nature, and appears to be highly drug resistant but is often responsive to the ketogenic diet.

• Systemic abnormalities anticipated with status epilepticus

Hyperpyrexia, hypotension, hypoglycemia, acidosis, increases intracranial tension, hypoxemia may occur during SE and exacerbate the cerebral damage.

Generalized muscle contractions lead to increase body temperature, rhabdomyolysis can cause hyperkalemia and myoglobinuria which can cause acute kidney injury.

• RECOGNITION AND ASSESSMENT

- Detailed and accurate history from an eyewitness (beginning, middle and end of the episode)
- When history unclear, recording the episode with camera /mobile phone can be very useful
- Common causes are febrile status epilepticus (most common in children), drug related, and CNS infection or post infectious encephalitis.
- Any underlying problem: learning difficulties, cerebral palsy, hypoxic ischemic encephalopathy, head injury or other CNS insult, Neurocutaneous syndromes.
- Family history may be positive in certain idiopathic generalized epilepsies, some symptomatic epilepsies (tuberous sclerosis), and autosomal dominant frontal lobe epilepsies.

- **Primary Investigations:**
  - CBC, blood glucose level, electrolytes (Na, K, Ca, Mg)
  - Brain CT scan should be done to all patients.
  - CSF analysis and culture
  - Blood culture, toxicology screening and tests for IEM are often needed
  - Anti-seizure drugs (ASD)levels for all patients known to be taking them
  - EEG is needed in all patients for:
    - ✓ Ruling out pseudo-status epilepticus or other movement disorders
    - ✓ Helps in identifying type not SE
    - ✓ Distinguish between postictal depression and later stages of SE in which clinical manifestations are subtle or absent.
    - ✓ Helps in monitoring therapy, particularly in patients who are paralyzed or intubated.
- **Management**

❖SE is a medical emergency so all patients should be admitted to ICU and support with oxygen.

❖Adjust patient's position, roll the patient to his side, and bend the knees keeping the legs at right angle to his body.

❖AS placement of IV catheter may be difficult or delayed in these patients, many ASD may be given by alternative routes e.g. buccal and intranasal midazolam and IM phosphenytoin and benzodiazepines, all of which are safe and well tolerated and quickly absorbed.

❖Follow each step until seizures resolve, but do not treat post-ictal posturing as seizure.

❖Prepare next step in algorithm immediately after previous one administered.

❖Don't give more than 2 doses of benzodiazepine, including prehospital doses.

❖A trial of Pyridoxine IV or PO 100mg is warranted especially in infants.

❖With all options of medical treatment respiratory depression is a potential side effect for which the patient should be monitored and managed as needed.

Status Epilepticus Algorithm

Pre-Arrival
Prepare equipment:
- Non-rebreather, BVM
- Suction, oral/nasal airway, pulse ox
- IV, mucosal atomizer, IO, iStat and Dstix
- Medications

Ongoing seizure > 5 minutes
Recurrent seizure without return to baseline mental status

Seizure	Status Epilepticus	Early Refractory SE	Refractory SE	Established SE
	5 min	10 min	20 min	30 min
Anti-epileptics Lorazepam (Ativan) OR 0.1 mg/kg IV/IM (max 4mg) Midazolam (Versed) OR 0.1 mg/kg IV (max 4mg) 0.2 mg/kg IN 13-40kg:5mg IM, >40kg:10mg Diazepam (Valium) 2-5yo 0.5 mg/kg PR 6-11yo 0.3 mg/kg PR ≥12yo 0.2 mg/kg PR (max 20) **Diagnostics / Therapeutics** Airway, oxygen, pulse ox Glucose, iStat (Na, Ca) Tx hypertension, fever IV access (or IO)	**Anti-epileptics** Repeat benzodiazepine q 5min up to 2 more doses Fosphenytoin OR 20-30 mg/kg PE (max 1gm) IV (150mg/min) Phenytoin 20-30mg/kg (max 1 gm) IV (50mg/min) **Diagnostics / Therapeutics** Consider ingestions (INH, TCA, ETOH) Chem panel incl Mg, PO4 Anti-epileptic drug levels CBC, LFTs, coags, Utox, ICON Consider neurology consult	**Anti-epileptics** Levetiracetam (Keppra) 20-40 mg/kg (max 3gm) IV at 5mg/kg/min Valproate 20-40 mg/kg IV at 5mg/kg/min (avoid in patients with liver disease, thrombocytopenia, metabolic disorder) **Diagnostics / Therapeutics** Consider head CT Consider lumbar puncture EKG Neurology consult	**Anti-epileptics** Phenobarbital 20-40 mg/kg IV at 2mg/kg/min Consider empiric pyridoxine If < 2yo, 100mg IV Consider empiric thiamine & glucose If suspect ETOH abuse, thiamine 100mg IV **Diagnostics / Therapeutics** Prepare to intubate Admit PICU Consider central line Arrange continuous EEG	**Coma induction** Midazolam 0.2 mg/kg (max 10mg) IV then infusion 0.1 mg/kg/hr Pentobarbital 5 mg/kg IV then infusion 0.5mg/kg/hr **Add-on options** Ketamine 1.5 mg/kg IV, then infusion at 1 mg/kg/hr Propofol 2 mg/kg IV, then infusion *in adults only* 1 mg/kg/hr, titrate up to effect* General anesthesia

Abend NS et al. Semin Pediatr Neurol 2014;21(4):263-274

*Risk of propofol infusion syndrome in children

Paediatric Status Epilepticus

Starship Child Health

AUCKLAND DISTRICT HEALTH BOARD Te Toka Tumai

Call for help: 777

Consider treatable causes
- Hypoglycaemia
- Electrolyte disturbance
- Meningitis/ Encephalitis
- Epilepsy
- Hypertensive emergency
- Toxins
- Intracranial bleed
- Blocked shunt

Assess ABC/Airway positioning/High flow O2

Check blood glucose
If BSL < 3.0 give 2ml/kg of 10% glucose

Seizure greater than 5 minutes

Intravenous (IV) or Intraosseous (IO) access?

YES

IV Midazolam 0.15mg/kg *(max 10mg)*
(including pre-hospital treatment)

5 minutes

IV Midazolam 0.15mg/kg *(max 10mg)*
(including pre-hospital treatment)

5 minutes

IV Levetiracetam 40mg/kg *(max 3g)*
Dilute 1:1 with 0.9% sodium chloride to a minimum volume of 10ml and infused over 5 mins
OR if already on Levetiracetam, then
IV Phenytoin 20mg/kg over 20 minutes *(max 1g)*
Dilute 1:1 with 0.9% sodium chloride to a minimum volume of 20ml and infused over 20 mins

5 minutes post infusion

IV Phenytoin 20mg/kg over 20 minutes if not given above *(max 1g)*
OR IV Levetiracetam 40mg/kg over 5 minutes if not given above *(max 3g)*
Prepare for Rapid Sequence Intubation (RSI):
Ensure CODE BLUE has been called, get RSI checklist and locate equipment, medications and personnel

Consider Rapid Sequence Intubation followed by Benzodiazepam infusion
OR 4th line anticonvulsant (sodium valproate/phenobarbitone) if intubation not appropriate

NO

IM Midazolam 0.2mg/kg *(max 10mg)*
OR Buccal or Intranasal Midazolam 0.5mg/kg *(max 10mg)*
(including pre-hospital treatment)

Achieve IV access or if seizure continues after further 5 minutes and no IV access insert IO

10 minutes

IV or IO access

IM Midazolam 0.2mg/kg *(max 10mg)*
OR Buccal or Intranasal Midazolam 0.5mg/kg *(max 10mg)*
(including pre-hospital treatment)

IV or IO access

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ACUTE STROKE PRACTICE GUIDELINES
For The Emergency Department

OUTCOMES / GOAL

1. Rapid identification of stroke events.
2. Manage acute stroke appropriately and efficiently according to recent guidelines.
3. Evaluate in a cost-effective manner.

TRIAGE STAFF (ER DEPARTMENT)

1. Triage physician to see patient rapidly upon arrival. If presenting with stroke signs/symptoms, notify emergency neurology physician.
2. Stoke symptoms include:
   - Sudden onset of numbness or weakness of the face, arm or leg, especially on one side of the body;
   - Confusion, trouble speaking or understanding speech;
   - Trouble seeing in one or both eyes;
   - Trouble walking, dizziness, or loss of balance or coordination;
   - Severe headache with no known cause or "worst headache of my life.")

EMERGENCY DEPARTMENT EVALUATION

1. Anticipate initial patient assessment for:
   - History: age, time of symptom onset (when last normal), duration, type of symptoms, medications (antiplatelet and anti-coagulants), past medical history (CAD, HTN, DM, previous TIA/stroke, PVD, seizures/epilepsy, tobacco, illicit drug use.
   - Exam: visual fields, extraocular muscles, speech impairment, weakness or sensory deficits, incoordination, ataxia.
2. Anticipate orders for:
   - Emergency CT brain without contrast
   - Labs for CBC, RBS, INR, PTT, troponin and Chemistry
   - 12 lead ECG
   - CXR (if clinically indicated)

HYPERACUTE STROKE CARE

A. Ischemic Stroke:

1. Consider thrombolytics for all ischemic stroke patients who present with symptom onset of 3 hours or less. Select patients may be considered for thrombolytics between 3-4.5 hours of onset. Follow international practice standard for intravenous administration of rT-PA in acute ischemic stroke as appropriate with goal of door to thrombolytics less than 60 minutes.
2. Consider interventional thrombectomy maneuvers for onset of symptoms of 24 hours or Less if clinically / radiologically in candidate patients.
3. Arrange for ICU / Stroke Unit admission

B. Hemorrhagic stroke:

1. If CT subsequently shows intracranial hemorrhage (subarachnoid or intracerebral), refer international Practice Guidelines for the Inpatient Management of Patients with Intracerebral and Subarachnoid Hemorrhage.
2. Arrange for ICU / Stroke Unit admission

ADMISSION CRITERIA

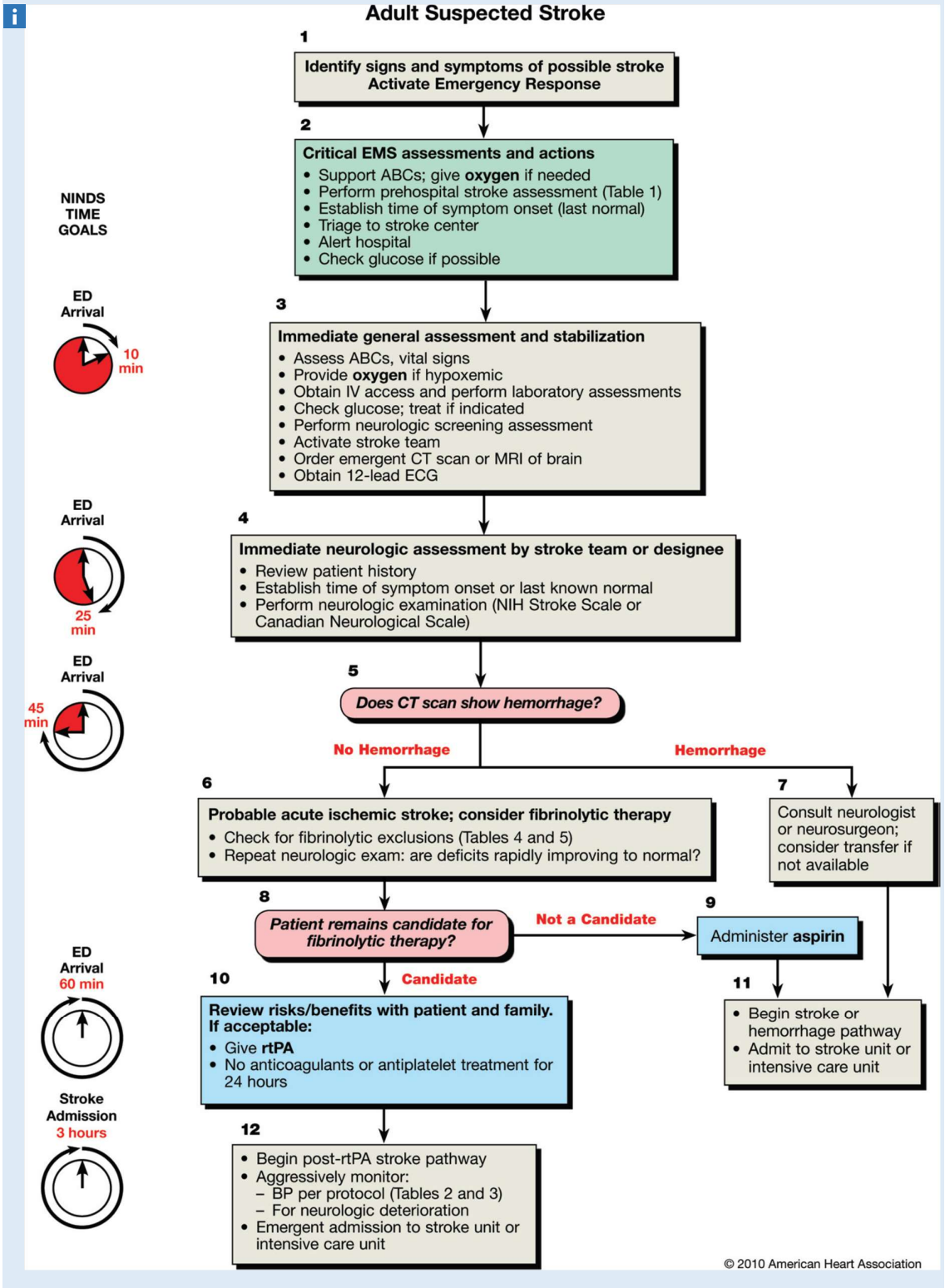
A. Criteria for admission to Neurosciences ICU / Stroke Unit:

- Acute ischemic stroke candidate for thrombolysis / thrombectomy.
- Patients with hemispheric stroke in whom impending mental status decline and loss of protective airway reflexes is of concern.
- Patients with basilar thrombosis or tip of the basilar syndrome.
- Patients with crescendo TIAs.
- Patient requiring blood pressure augmentation for a d ocumented area of hypoperfusion.
- Patients requiring IV blood pressure or heart rate control.
- Patients requiring every 1-2 hour neurological evaluation depending on symptom fluctuation or if ongoing ischemia is suspected.
- Patients with worsening neurological status.

B. Criteria for Admission to Neurology Ward

- Acute stroke symptom onset > 24 hours and not meeting above criteria.
- Non-crescendo TIAs for ischemic stroke workup.

PATIENT FLOWCHART



Protocol of Management of Peripheral Nerve Injury

Introduction:

When neurosurgeons encounter patients with traumatic peripheral nerve injuries, the initial step is to accurately localize and characterize the lesion because this information helps dictate patient management, including the timing of any surgical intervention. When inaccurately assessed, improper management may result, thereby diminishing the chance of a good functional outcome. Even when ideally managed, for a number of reasons, the functional outcome of peripheral nerve injuries may be suboptimal, including lesion severity, long distance between the lesion and the denervated target organs (muscle fibers, sensory receptors), and significant connective tissue proliferation at the lesion site. This protocol reviews the more common types of traumatic nerve injuries and the major neurosurgical interventions available. In all situations, a neurosurgeon with experience in peripheral nerve trauma should be involved early.

Demographics:

The majority of peripheral nerve injuries are traumatic, and the incidence of traumatic nerve injuries is approximately 350,000/year. Traumatic nerve injuries involve a single upper extremity nerve in a young male. In a large series of 456 patients, 74% were male, the mean age was 32.4 years, the upper extremity was involved in 73.5%, and 83% were mononeuropathies. Among traumatic upper extremity mononeuropathies, the radial nerve is the most frequently involved, followed by the ulnar nerve, and then, the median nerve; the sciatic nerve is the most frequently involved lower extremity nerve, followed by the peroneal nerve. During peacetime, most peripheral nerve injuries follow motor vehicle accidents.

Nerve injury types:

Peripheral nerve injuries follow any force capable of disrupting the neuron cell body, its protoplasmic projections (axons), or their coverings (myelin). Although neurons may be injured in a large number of ways (e.g., traction, compression, transection, electrical, radiation, thermal, and injection), the pathologic outcomes

are limited to demyelination and Wallerian degeneration. With focal demyelination, there are no distant pathological effects, whereas with axon disruption, the distal segment undergoes Wallerian degeneration (axon loss). Most nerve injuries are mechanical in nature (compression, traction, transection). Of these, traction injuries predominate in the civilian sector. Less common mechanisms include friction (dynamic compression of the nerve against another structure), pressure (injuries related to pressure changes within an enclosed space through which the nerve

passes), and ischemic injuries, as well as traumatic injuries related to thermal, electrical, or radiation insults. The anatomic location of an extremity nerve within a limb correlates with the type of injury to which it is most susceptible. For example, due to its course around the humerus, the axillary nerve is most susceptible to traction injuries from shoulder dislocation. The fascicular composition of a nerve also dictates its vulnerability to traction or compression injury. Because different segments of the same nerve have differing fascicular structure, segmental susceptibilities also occur. Hence, one segment may be more susceptible to traction, another to compression, and another to both forces. Nerve roots are susceptible to both traction and compression because of their lack of epineurium and perineurium, a lesser number of collagen fibers within the endoneurium, and the organization of their nerve fibers into parallel bundles.

Traction injuries:

Nerves are somewhat resistant to stretch injury, a reflection of their tensile strength and elasticity. The tensile strength of a nerve reflects its ability to withstand loads that elongate it. The structural features than generate resistance to stretch injury include the undulating course of the nerve through the limb and the undulating course of its nerve fibers through the fascicle (the undulatory features provide slack), as well as the tensile properties of the perineurium. As tension is applied to a nerve, its undulatory features are lost first, at which point, further resistance to stretch is provided by the perineurium. As the tension increases further, the perineurium ruptures. The point at which perineurial rupture occurs reflects the magnitude, duration, and rate of application of the traction force.

Compression injuries:

The compressive strength of a nerve reflects its ability to withstand loads that reduce its diameter. Whereas the perineurium provided resistance against traction injury, the epineurium provides resistance against compression injury by dissipating external pressure applied to it. Therefore, nerves with more epineurial tissue are able to resist larger compressive forces. Fascicular structure also contributes to compressive force resistance. A larger number of smaller fascicles dissipates compressive force better than a smaller number of larger fascicles. For example, the peroneal division of the sciatic nerve has a smaller number of larger fascicles and less epineurial tissue than does the tibial division, accounting for its greater vulnerability to compression. Other reasons for its greater susceptibility include less epineurial adipose tissue, a poorer blood supply, and nerve anchoring at two sites (the sciatic notch and the fibular head). Compressive forces produce nerve injury in two ways—damage to the nerve fiber (axon or myelin) and endoneurial edema formation. Endoneurial edema impedes nerve function by increasing the endoneurial fluid pressure (e.g., compartment syndrome), which reduces axon transport, may impede the intraneural microcirculation, and may generate fibrosis. Concomitant stretch injuries (from nerve fiber angulation) and ischemic injuries (from vascular compromise) may be present. When compressive

forces are short in duration (e.g., sitting on a hard toilet seat, leg crossing), the resultant neurological features are typically transient and positive (tingling) and reflect ischemia (when the external pressure on the blood vessels exceeds the systolic blood pressure). When compressive forces are of longer duration, negative features (numbness, weakness) may occur from myelin or axon disruption. With compressive injuries, because the basement membrane surrounding the Schwann cell is unaffected and because the connective tissue elements are not disrupted,reinnervation is favored and, hence, the prognosis tends to be good. Body sites more susceptible to nerve compression include those areas where the nerve passes through a narrow opening, passes across the edge of another structure, or is superficial. Nerves are more susceptible to external compressive forces where they pass over a bone (radial nerve/spiral groove, common peroneal nerve/fibular neck, ulnar nerve/ulnar groove). Compressive force susceptibility may also be genetic (tomaculous neuropathy).

Transection injuries:

With transection injuries, because the nerve fibers (and connective tissue elements) are severed, these disorders are axon loss in type. They are categorized as complete or incomplete and, depending on the mechanism of transection, as sharp or blunt. With complete injuries, the severed ends pull away from each other, creating a gap that prevents reinnervation by proximodistal axon regrowth. Thus, transection injuries have a worse prognosis.

Lesion assessment:

The pathophysiology of the lesion dictates its clinical and electrodiagnostic (EDX) manifestations. With demyelinating conduction slowing (DMCS), positive symptoms (e.g., tingling) occur because the action potentials are able to traverse the lesion site, albeit at a slower rate. When the fibers are demyelinated to differing degrees, there is a resultant loss of synchrony among the propagating action potentials. As a result, vibratory perception and muscle stretch reflexes are diminished or absent because they require the synchronous arrival of action potential volleys. With demyelinating conduction block (DMCB) and axon loss, the clinical manifestations are negative (numbness, weakness) because the action potentials cannot traverse the lesion. With DMCB, because the affected motor axons remain in contact with the muscle fibers, there is no associated muscle atrophy. Also, because DMCB primarily involves the larger diameter, more heavily myelinated nerve fibers, large fiber sensory modalities (vibration, proprioception,discriminative touch) are affected out of proportion to the small fiber modalities (pain, temperature, crude touch). With axon loss, muscle atrophy is evident because denervated muscle fibers decrease in their transverse diameter. With axon loss, the associated sensory loss involves the large and small fiber sensory modalities more evenly. With axon loss lesions, the EDX abnormalities observed depend on lesion severity and the timing of the study. With mild axon disruption,isolated fibrillation potentials may

be the only abnormality. Because of the high innervation ratio (muscle fibers innervated per motor neuron) of the skeletal muscle, a large number of fibrillation potentials (typically hundreds) are generated per motor axon disrupted, rendering the needle electromyogram (EMG) examination the most sensitive EDX study for motor axon loss. Unfortunately, fibrillation potentials typically do not appear until day 21 (and as late as day 35). With more severe lesions, low amplitude sensory responses are observed. The sensory response amplitude decrement from Wallerian degeneration begins around day 6 (as some of the sensory axons fail to conduct action potentials) and is complete by day 10 (when there is uniform conduction failure among the affected axons). With lesions of even greater severity, low-amplitude motor responses appear. The motor response amplitude decrement begins around days 2–3 and is complete by day 6. This reflects the fact that neuromuscular junction (NMJ) degeneration precedes axon degeneration and the motor responses are dependent on NMJ transmission. Because the motor response amplitude value (and the negative area under the curve value) reflects the number of functioning muscle fibers, it is useful in severity assessment. Because the latency and conduction velocity values only reflect the fastest conducting nerve fiber, these measurementsare insensitive to axon loss. Even when all of the fastest fibers are involved and the latency and conduction velocity values become abnormal, their degree of abnormality is small in comparison to the degree of amplitude decrement. In general, once the motor response falls to about 50% of its normal size, the sensory response from the nerve becomes absent (or nearly so) and a neurogenic MUAP recruitment pattern may be observable. The latter becomes more obvious as the severity increases further. Focal demyelination and neurogenic recruitment do not require time to mature and, thus, are apparent at lesion onset, assuming the lesion is severe enough to manifest them. Most abrupt-onset lesions are traumatic in origin and axon loss in nature. With traumatic median neuropathy, DMCB or axon loss (or a combination) is observed. Conversely, with slowly progressive entrapment of the median nerve (i.e., carpal tunnel syndrome), the earliest pathophysiology is focal DMCS. As the disorder becomes more severe, axon loss appears. Consequently, traumatic median neuropathies have a completely different pathophysiology (and natural history) than median neuropathies from carpal tunnel syndrome. For this reason, median nerve trauma occurring at the carpal tunnel should never be referred to as acute carpal tunnel syndrome.

Severity assessment by strength assessment:

The Medical Research Council (MRC) scale is a nonlinear scale for grading muscle strength that was first published in 1941, revised in 1943, and republished in a document entitled “Aids to the Investigation of Peripheral Nerve Injuries (War Memorandum No. 45)”. Grade 0 indicates no visible muscle movement; grade 1 indicates muscle movement without joint movement; grade 2 indicates contractile force unable to overcome gravity; grade 3 indicates that the generated contractile force is able to overcome gravity without added resistance; grade 4 indicates that the

generated contractile force overcomes gravity plus added resistance (providedby the examiner); and grade 5 indicates normal contractile force against full resistance. Based on these definitions, grade 4 represents about 70% of the scale, extending from grade 3 to grade 5. Because of the wide range of muscle strength represented by grade 4, a plus or minus sign is typically used to subdivide it into mild, moderate, and severe weakness (4+, 4, and 4−, respectively).

The MRC scale for assessing muscle strength:

Grade	Observation
0	No visible muscle contraction
1	Visible muscle contraction without active movement
2	Active movement with gravity eliminated
3	Active movement against gravity
4-	Active movement against weak resistance
4	Active movement against moderate resistance
4+	Active movement against strong resistance5
Normal power	

Severity assessment by EDX assessment:

In general, when patients with traumatic peripheral neuropathies are initially followed (e.g., blunt trauma), EDX testing is typically performed after post-trauma day 21. It is important that EDX providers appropriately apply the various EDX studies so that the lesion is ideally localized and characterized. The basic concepts of lesion localization and characterization are reviewed in most standard EDX textbooks. The following discussion is a summary of the most important concepts and an EDX example demonstrating the determination of the associated pathophysiologies.

Motor NCS:

Unlike the MRC scale, which estimates muscle power using a nonlinear 6-point scale (0–5), the motor NCS are much more specific, providing a linear 100-point scale. This is accomplished by comparing the motor response recorded from the weak muscle to the one recorded from the homologous muscle on the contralateral (asymptomatic) side, expressed as a percentage. The innervation ratio of a muscle (i.e., the number of muscle fibers innervated per anterior horn cell) is constant. Thus, the percentage of denervated muscle fibers within the muscle correlates with

percentage of disrupted motor axons within the nerve. Following muscle fiber denervation, motor axons adjacent to the denervated fibers sprout collaterals andreinnervate the denervated muscle fibers. As a result of this mechanism of reinnervation, termed collateral sprouting, the innervation ratio of the adopting motor neuron increases. Thus, following reinnervation by collateral sprouting, the percentage calculation underestimates lesion severity. Prior to collateral sprouting, the distal motor response amplitude values of the symptomatic and asymptomatic sides can be compared to provide an estimate of severity. In the setting of DMCB lesions, the percentage of blocked motor nerve fibers can be calculated by comparing the motor response amplitude values of the motor responses recorded with stimulation below and above the lesion. Mixed lesions are calculated using both techniques in sequence, starting with the axon loss determination. An example of this is provided here. Example. A 48-year-old man presents on day 28 for EDX assessment of a traumatic right peroneal neuropathy. The right superficial peroneal sensory and peroneal motor (recording EDB) responses are absent, the right peroneal distal motor response (recording TA) is low in amplitude and shows a DMCB across the fibular head, and the needle EMG show 3+ fibrillation potentials with neurogenic motor unit action potential (MUAP) recruitment in a right common peroneal nerve distribution. There are no chronic changes (e.g., increased MUAP duration). Thus, this is a mixed lesion (DMCB and axon loss) localized to the fibular head. The amplitude values of the right and left common peroneal motor responses can be used to semi-quantify the underlyingpathophysiologies. For example, when the amplitude of the peroneal motor response recording TA is 3.0 mV with ipsilateral below-fibular head stimulation, 1.5 mV with ipsilateral above-fibular head stimulation, and 6.0 mV with contralateral below-fibular head stimulation, the responsible pathophysiologies are easily determined. The percentage of axon loss is calculated by comparing the distal motor response amplitude values of the two sides using the formula, 1 − symptomatic side / asymptomatic side × 100%, as follows:

= 1–3.0/6.0 x 100%

= 1−0.5 x 100%

= 0.5 x100%

= 50%

The percentage of DMCB is calculated using the formula, 1 – proximal response / distal response × 100%, as follows:

= 1−1.5/3.0 x 100%

= 1–0.5 x 100%

= 0.5 x 100%

= 50%

Thus, of the motor axons not affected by axon loss, 50% are affected by DMCB. Hence, 50% are axon loss, 25% are DMCB (half of 50%), and 25% are normal. When only the ipsilateral values are used, the erroneous impression that there is a DMCB affecting 50% of the fibers results. When the motor responses show response dispersion, the amplitude values are unreliable. In this setting, the negative areaunder-the-curve values should be utilized. In summary, because reinnervation via collateral sprouting increases the innervation ratio, the calculated percentage of axon loss represents an underestimate. Consequently, beyond three months, it is more accurate to state that the axon loss involves at least the calculated percentage.

Sensory NCS:

Because of their low amplitude, short negative phase duration, greater number of phases, and wider range of conduction velocities, sensory responses are quite susceptible to physiological temporal dispersion and, thus, overestimate the severity of axon loss. In general, after Wallerian degeneration has occurred and before reinnervation through collateral sprouting has occurred, when the nerve is about 50% disrupted, the motor responses are about 50% decreased and the sensory responses are about 90% decreased (or absent). Although sensory responses are not helpful for lesion severity estimation, their susceptibility makes them better for the initial localization of axon loss lesions.

Needle EMG examination:

The needle EMG study cannot approximate the percentage of motor axon involvement and, thus, is of limited value in grading lesion severity. The quantity of fibrillation potentials observed primarily reflects the timing of the study, not the severity of the lesion. Also, the grading scale (1+ to 4+) is nonlinear (i.e., a grade of 2+ does not represent twice as many fibrillation potentials as a grade of 1+). The presence of a neurogenic MUAP recruitment pattern indicates that at least 50% of the motor nerve fibers are unable to transmit action potentials. However, it does not differentiate between axon loss and DMCB. In general, the observation of an MUAP firing at a rate of 20 Hz or more is abnormal because this rate is 3 standard deviations above the mean firing rate for a 30% maximum isometric contraction.

Nerve injury classification:

With the Seddon and Sunderland nerve injury classification systems, the degree of connective tissue disruption dictates lesion severity. Because this is a histological determination, it cannot be employed in the acute setting. Nonetheless, it is important to be familiar with these two systems.

The Seddon classification system:

With the Seddon system, there are three grades of severity: neurapraxia (myelindisruption), axonotmesis (axon disruption), and neurotmesis (nerve disruption).

Neurapraxia:

Neurapraxia, the mildest grade, consists of focal myelin disruption that blocks action potential propagation (DMCB). EDX testing identifies neurapraxia and, after day 6, differentiates it from axon loss. Neurapraxia has an excellent prognosis, with full motor recovery following remyelination. The production of voltage-gated sodium channels along the demyelinated segment may restore action potential propagation through the lesion prior to remyelination.

Axonotmesis:

With axonotmesis (tmesis, a cutting), there is axon disruption and resultant Wallerian degeneration. Because the connective tissue elements (endoneurium, perineurium, epineurium) are unaffected, the neural tubes (endoneurium) are spared. Thus, the prognosis is excellent because the regenerating axons are able to advance unimpeded within their original endoneurial sheath. Because collateral sprouting occurs within the muscle, in the setting of incomplete axon loss, reinnervation via collateral sprouting is the primary mechanism of reinnervation.

Neurotmesis:

With neurotmesis, the nerve is divided and recovery cannot occur without surgical intervention. In general, unless there is exclusionary evidence, it is best to assume that focal nerve trauma represents a neurotmetic injury so that the opportunity for surgical intervention is not lost.

<u>The Sunderland classification system:</u>

The Sunderland system employs 5 grades of nerve injury. With this system, the degree of connective tissue involvement is better defined and, thus, provides more accurate prognostication. Sunderland grade 1 is equivalent to neurapraxia and Sunderland grade 2 is equivalent to axonotmesis.

Sunderland grades 3–5:

With lesions more severe than axonotmesis, recovery through proximodistal axonal advancement primarily reflects two factors: (1) the ability of the regenerating axons to advance across the lesion site and (2) their ability to enter the proper endoneurial tubes within the distal stump. These factors depend on certain characteristics of the lesion, including its severity, degree of connective tissue disruption and proliferation, presence of a gap or debris between the proximal and distal stumps, underlying etiology, and patient's age.

Sunderland grade 3:

With grade 3 lesions, which often follow severe traction or compression, the endoneurium is disrupted. This permits axon misdirection—the advancing axon enter the wrong endoneurial tube (aberrant reinnervation). The outcome depends on the tube entered and may be asymptomatic (a motor axon enters an endoneurial tube leading to the same muscle) or symptomatic (when it leads to an alternative end organ (sensory receptor, autonomic gland, different muscle)). Examples of nerve fiber–receptor mismatch with seventh nerve lesions include crocodile tears (lacrimation during eating when a motor axon advances to the lacrimal gland) and synkinesis (when a motor axon advances to a different facial muscle). Because axons are more intermingled proximally, the likelihood of aberrant reinnervation is greater with proximal nerve lesions. The outcome of Sunderland third-degree lesions varies widely (from good to negligible), depending on the number of endoneurial tubes disrupted, the degree of associated fibrosis (bleeding and intrafascicular edema lead to intrafascicular fibrosis and possible neuroma formation), the level of the lesion along the nerve, the distance between the lesion and the denervated end organs, and the completeness of the lesion. Without surgical intervention, incomplete recovery frequently occurs.

Sunderland grade 4:

With grade 4 injuries, there is perineurial disruption and, hence, fascicular structural damage. Thus, there is greater axonal misdirection and greater fibrosis.As a result of the perineurial disruption, advancing axons may exit the fascicle or they may form a neuroma within the nerve (neuroma-in-continuity). Surgical intervention is required.

Sunderland grade 5:

With grade 5 injuries, there is epineurial disruption and the nerve trunk may be severed (neurotmesis in the Seddon system). Like fourth-degree injuries, surgical intervention is mandatory.

Sunderland grade 6:

Nerve injuries of mixed grade, which are the most challenging to treat. With these lesions, fascicular treatment is individually based. It is important to distinguish axon loss lesions that might recover spontaneously (second- and some thirddegree) from those that require surgical intervention (most third-degree and all fourth-degree and fifth-degree). Acutely, their clinical features are identical and, thus, they cannot be differentiated noninvasively. In addition to lesion severity, functional outcome also reflects time to operative repair and the age of the patient. Regarding the former, although satisfactory outcomes may follow surgical intervention after 12 months, the best motor outcomes are associated with surgical intervention before 6 months, with less ideal outcomes between 6 and 12 months and the poorest outcomes after 12 months. Because denervated sensory receptors do not undergo degeneration, sensory recovery may occur up to 48 months later. Regarding age, operative outcomes are better for younger individuals (under 20) than for older persons (over 50).

Neuromuscular imaging:

Introduction:

Clinical and EDX testing are useful initially for lesion localization, after which neuromuscular imaging using high-resolution US or magnetic resonance (MR) imaging are employed for lesion visualization and characterization. As stated previously, the basic unit of a peripheral nerve is the axon (either myelinated or unmyelinated), the fluid between the Schwann cell membrane and the axolemma is the endoneurial fluid, and the interfascicularepineurial tissue of larger nerves contains various amounts of adipose.

Neuromuscular ultrasound:

Neuromuscular ultrasonography (US) began in the 1980s and, through technical advancements, led to the development of high-resolution US (12–18 MHz transducer) and the enhanced ability to quickly identify and noninvasively and painlessly assess long lengths of specific nerves. Of the measured parameters (e.g., echotexture, vascularity), nerve cross-sectional area (CSA) is most commonly reported. To avoid overestimating the CSA, the transducer is oriented perpendicular to the longitudinal axis of the nerve. The hyperechoic (bright) signal of the epineurial tissue is manually outlined or estimated by placement of an ellipse of best fit. By providing anatomical details, neuromuscular US compliments EDX testing. One major disadvantage of the US is its dependence on the skills and experience of the operator.

Magnetic resonance neurography:

Specialized RF receiver coils (phased-array coils), which are flexible and specific to each body region, integrate the data from individual coils into a single image, thereby mimicking a single, much larger coil [28]. This technique maintains the high signal-to-noise ratio (SNR) of smaller coils and overcomes their small field of view (FOV). Nonetheless, the FOV is still small and requires lesion localization (clinically or electrodiagnostically) prior to imaging. The degree of spatial resolution is proportional to the size of the matrix (e.g., 512 × 512 provides a larger FOV than 256 × 256), and the SNR is increased by imaging with higher field strengths. Standard spin-echo T1-weighting imaging (T1WI) best displays regional anatomy, whereas fast spin-echo T2-weighted imaging (T2WI) best demonstrates intraneural pathology. Fat suppression techniques (e.g., STIR) are required so that adjacent extraneural fat does not obscure the desired intraneural signal. As with the US, at least two planes of imaging are required. With nerves, parallel (in-plane)

images and perpendicular (cross sectional) images are collected. In-plane imaging identifies nerve displacement and caliber changes, whereas perpendicular imaging permits fascicular pattern assessment and better nerve caliber assessment [28]. Because nerve caliber varies along its length, contralateral comparison studies are helpful. Contrast is helpful when neoplastic, inflammatory, post-XRT, or infectious processes are suspected, but otherwise is typically unnecessary. Abnormal findingsinclude loss of perineural fat, diffuse or focal enlargement, diffuse or focal T2 hyperintensity, abnormal fascicular patterns, and T1 enhancement. With nerve trauma, MRI helps characterize the lesion (e.g., intraneural hematoma, neuroma, loss of continuity, diffuse or focal perineural fibrosis). The muscles innervated by the affected nerve may show features of denervation or fibrofatty changes.

Magnetic resonance neurography (MRN):

Recent advancements in MR imaging, especially diffusion-weighted imaging (DWI) and diffusion tensor imaging (DTI), have significantly increased the utility of MR neurography for assessing peripheral nerve injuries, including the connective tissue elements.

DWI neurography (DWIN):

Peripheral nerves are highly anisotropic (i.e., water diffusivity is markedly limited along its perpendicular axis and facilitated along its longitudinal axis). DWN provides predominantly qualitative information about axonal integrity; quantitative information is derived through the apparent diffusion coefficient (ADC). The ADC reflects water displacement into the extracellular space, with high values usually representing edema. The advantages of DWN include high background signal contrast ratio, large FOV, short acquisition times, and quantification through ADC; disadvantages include low spatial resolution, contamination by other DWI hyperintense structures, T2-shinethrough effect, and the low specificity of ADC.

DTI neurography (DTIN):

Normally, diffusion of free water along the longitudinal axis is facilitated and movement transversely is restricted. Measured parameters include fractional anisotropy (FA, the most important measurement), mean diffusivity (MD), axial diffusivity (AD), and radial diffusivity (RD). DTIN data are also reconstructed to create 3D images. The advantages of DTIN include high anatomical detail and quantification (FA and diffusivity); disadvantages include artifact susceptibility, small FOV, and long acquisition times. The FA value reflects fascicular integrity and water movement direction. A low FA value suggests isotropic diffusion and, hence, neural disruption, whereas a higher value suggests greater anisotropy and, thus, neural integrity. AD reflects longitudinal diffusion, RD reflects transverse diffusion along the two axes perpendicular to axial flow, and MD reflects the average of these three movements.

Nerve trauma:

With nerve trauma, there is increased water in the extracellular space and various degrees of connective tissue disruption. At the injury site, DWIN may show DWI hyperintensity, nerve thickening, increased ADC, and increased MD. Potential DTIN findings include decreased FA (loss of fiber anisotropy), increased RD (due to disruption of myelin sheaths and connective tissue elements), and edema. Thus, the FA and RD values change along a continuum as the Sunderland grade increases from grade 2 (minimal FA decrease and minimal RD increase) to grade 5 (large FA decrease and large RD increase); conversely, following regeneration, the FA value progressively increases and the RD value progressively decreases. As the edema lessens, the ADC and MD values normalize. With compressive forces, such as with carpal tunnel syndrome, there is enlargement of the extracellular space (increased CSA on US and MRN), increased ADC (edema), and loss of nerve anisotropy (FA value reduction and diffusivity changes). Following surgical release, the FA value increases, typically within 6 months, and the RD value decreases. Standardization studies are required to determine the exact role of these newer MRN techniquesin the assessment and management of peripheral nerve trauma.

Surgical management:

Introduction:

Despite incredible advances in peripheral nerve repair, the ideal timing of surgical intervention is often unclear and reflects a number of factors, including the following: the circumstances surrounding the injury, the timing of the symptoms, the type and severity of the injury, the required

regeneration distance, the degree of fascicular disruption, the morbidity and mortality risks of the procedure, the degree of concomitant tissue trauma and contamination, and the age and comorbidities of the patient. Among these, nerve injury type and severity primarily dictate management.

In general, immediate surgical exploration is usually employed when neurological deficits develop in the distribution of a nerve following sharp penetrating injury, when nerve transection is suspected, and with severe or open trauma, injection injury, or following surgery, whereas with less severe or closed trauma or suspected neurapraxia, conservative treatment (e.g., splinting, physical therapy, and neuropathic pain medications) with serial clinical and EDX assessments for evidence of improvement, worsening, or new symptoms is typically employed.

When evidence of worsening, features suggestive of a neuroma (neuromas may be identified by ultrasound or magnetic resonance neurography), or failure to recover as expected occurs, surgical intervention is considered. Unfortunately, the ideal observation period is not always clear. Although a number of factors contribute to this determination, in general, motor function recovery is best when surgical intervention is undertaken within the first three months by an experienced peripheral nerve surgeon. Consequently, many authors recommend exploration during the first 3–6 months when recovery is absent or minimal. Shorter observation periods (e.g., 3 weeks) are frequently employed with high-energy injuries or those producing total or near-total paralysis. Although an advancing Tinel sign indicates distal nerve regeneration, it may reflect advancing sensory axons, in which case the time delay may eliminate the opportunity to achieve successful motor recovery through surgical intervention.

Approach to axon loss (grades 2–5):

Distinguishing grades 2–5 is important because their management differs: seconddegree injuries have an excellent prognosis without surgical intervention, fourth- and fifth-degree lesions require surgical intervention, and third-degree injuries often benefit from surgical intervention (spontaneous recovery is less frequent).

Two major issues are relevant—the completeness of the lesion and the distance between the lesion site and the denervated muscle fibers (i.e., the regenerative distance). With incomplete lesions, reinnervation via collateral sprouting occurs through the intramuscular motor branches of the unaffected motor axons. With complete lesions, reinnervation must occur through proximodistal axon advancement. When the regenerative distance exceeds 20 in, even when the axons successfully reach their target, they are too late because denervated muscle fibers undergo fibrofatty degeneration at 20–24 months. This is referred to as the Rule of 20. Thus, with an incomplete lesion and a 4-in regenerative distance, both mechanisms of reinnervation are available,

spontaneous improvement is expected, and an observation period is employed. Should improvement not occur at the expected time, enough time still remains for successful surgical intervention. Conversely, with a complete lesion located more than 20 in from the denervated muscle fibers, neither mechanism is available. For situations between these extremes, management must be individualized. Importantly, unnecessary surgical delay is the most common nonsurgical error and, hence, early surgical consultation is mandatory so that all options remain available to the patient.

Surgical interventions:

Although a number of surgical interventions are available, only a brief discussionof the major ones is provided here. The underlying injury mechanism contributes to initial management. In addition, whether the injury is open or closed is important.

With open injuries, surgical exploration is required, whereas with closed injuries, clinical and EDX monitoring is employed.

In general, with closed injuries, EDX testing is performed at 3 weeks and repeated every three months for evidence of recovery or worsening.

With compression injuries, timing plays a role. Most acute-onset compressive lesions (e.g., Saturday night palsy) are associated with DMCB and axon loss, whereas with chronic compression (e.g., carpal tunnel syndrome), demyelinating conduction slowing (DMCS) predominates. With these lesions, the nerve fibers closest to the compressive force and those with the thickest myelin tend to be more extensively involved. EDX testing can help tease out the various percentages and dictate management. The sensory and motor NCS and the needle EMG studies should be performed bilaterally.

Most closed traction injuries are associated with lesions in continuity and are treated conservatively because, at least initially, there is no way to determine the likelihood of recovery, the need for surgery, or the type of surgical intervention required. At day 7, motor NCS can differentiate grade 1 lesions (DMCB) from axon loss lesions (grades 2–5), but the latter grades cannot be differentiated from each other. As a result, with axon loss, a period of watchful waiting may be employed and, when signs of recovery fail to appear, surgical exploration is considered. The duration of the observation period must be individualized and varies with the circumstances of the injury (e.g., it is much shorter in the setting of high-energy injuries or those associated with total or near-total paralysis).

With sharp transection injuries, the ends of the transected nerve can often be reattached, whereas with blunt transection injuries, the damaged portions of each end can be removed and a cable graft placed. The term, cable graft, refers toa nerve graft composed of several sections of nerve similar to a cable, used for repair of nerve injuries involving multiple fascicles. Sharp transections are usually repaired within 72 h, when they are easier to assess (prior to scarring), easier to repair (prior to retraction), and the motor fascicles are still identifiable, whereas blunt transection repairs are usually delayed for 3–4 weeks, at which point the nonconducting fibrotic segments of both stumps are appreciable (termed the zone of injury).

When repaired prior to this time, failure rates of 100% have been reported. When transection injuries result in nerve gaps, a number of approaches may be used, depending on the distance between the nerve stumps, the diameter of the nerve, the location of the lesion along the nerve, and the availability of a proximal stump.

Nerve mobilization helps to approximate the two stumps. When the gap is less than 1 cm and stump approximation does not generate excessive tensiononthenerve, end-to-end repair (also known as end-to-end suturing, end-toendneurorrhaphy, and direct repair) is preferred because it allows the severed fascicles to be matched and reconnected.

Because the axons composing a fascicle change as the axons advance, lengthy lesions do not permit fascicular matching. With end-to-end suturing of monofascicular lesions (or polyfascicular lesions in which the severed fascicles cannot be matched), the epineurium is usually sutured to avoid trauma to the perineurium (termed an epineurial or epineural repair), whereas when individual fascicles are repaired, the perineurium is sutured (termed fascicular repair). When some fascicles can be matched and others cannot, both techniques are employed.

Unfortunately, even with proper alignment, only about 50% of individuals demonstrate functional recovery. Following removal of the fibrotic segments, when the ends cannot be approximated in a tension-free manner or when there is a gap between the two ends exceeding 1 cm, an end-toend repair is not performed.

Thus, either a nerve conduit or a nerve graft must be interposed. A nerve conduit is a synthetic nerve tube that may be resorbable or nonresorbable.With nerve tube repairs, the nerve stumps are inserted into the ends of the tube and sutured in place.

Nerve conduits collect the axoplasm and other neural stump fluids, from which a fibrin-based scaffold forms, thereby permitting cell migration. Schwann cells grow into the tube at both ends and axonal advancement occurs from the proximal stump.

Because the concentration of neurotrophic factors (released from the nerve ending after injury) within the conduit is critical in assisting nerve regeneration, based on the formula for the volume of a cylinder ($V = \pi r2L$), conduits are limited to thinnerdiameter nerve repairs and lengths not exceeding 3 cm. Also, a small portion of proximal nerve can beminced and placed in the center of the conduit for additional neurotrophic support.

Nerve grafts may be autografts or allografts and are useful for gaps in the 1–5 cm range. For a number of reasons, nerve autografts are considered superior. They provide endoneurial tubes, Schwann cells, and neurotrophic factors; have inherent flexibility and strength; and are immune-compatible, inexpensive, and their ready availability. Sensory nerves are more frequently used for autografting (e.g., sural, superficial peroneal, intercostal, lateral, and medial antebrachial cutaneous, dorsal ulnar cutaneous).

The sural nerve is often chosen because of its ease of harvest, limited morbidity, moderate dispensability, and length of nerve available (30–50 cm) [50]. Multiple sural nerve segments may be required for large-diameter nerve repairs.

It is interposed using an end-to-end suturing technique. To avoid postoperative recoil and tension, donor nerve length should exceed gap length by about 20%. The primary disadvantage of autografting is the loss of donor nerve function and possible neuroma formation. These two disadvantages are eliminated through the use of an allograft.

Cellular allografts (non-processed cadaver nerve grafts) are used for extensive injuries that require long lengths of nerve graft material. With cellular allografts, immunosuppression is required until native cells incorporate the graft (e.g., 18–24 months).

Acellular allografts (processed cadaver nerve grafts) are treated to eliminate the immunogenicity of the donor tissue and to provide an extracellular matrix to help direct axonal advancement. Although superior to conduits, acellularized autografts depend on proliferating host Schwann cells to support axonal regeneration and, consequently, the proliferative demand is greater with autografts of longer lengths, thereby also limiting this approach for large defects.

Thus, they are inferior to nerve autografts. For gaps exceeding 5 cm, vascularized nerve grafts and nerve transfers are utilized. Vascularized nerve grafts are used for long gaps among large nerve trunks when the recipient region is ischemic or scarred. For proximal injuries where the proximal stump is not available (avulsions) or not identifiable, devastating brachial plexus injuries, injuries with lengthy regenerative distances, or injuries in which the presentation has been delayed, a healthy adjacent nerve (the donor) can be attached to the distal stump of the severed nerve (the recipient). With this technique, termed nerve transfer or neurotization, the donor nerve may be used in its entirety or the transfer may be limited to one or more of its fascicles. Because the donor nerve is coapted to the recipient nerve near the motor endplate region of the affected nerve, reinnervation occurs earlier.

Finally, it is also possible to perform an end-to-side neurorrhaphy for long gap injuries. With this technique, the donor function is not lost. In a recent study of this technique in a rodent model, it was shown that axon regrowth from the donor nerve to the recipient nerve is more efficient when the donor nerve undergoes a 50% cross-section at the coaptation site (greater axonal sprouting) and the recipient nerve is degenerated (better attracts the axonal sprouts).

A large number of procedures are currently available for the surgical treatment of traumatic nerve injuries, each of which has its limitations. In the future, techniques to enhance axon regeneration, such as the application of devices for the sustained delivery of nerve growth factors, the implantation of Schwann cells at the injury site, and the delivery of stem cells able to differentiate into Schwann cells, will undoubtedly improve the outcome of those patients undergoing nerve grafting procedures.

Angle Closure Glaucoma

Presentation:

Symptoms;

Ocular>> Decreased visual -acuity, blurred vision, “haloes” around lights

Ocular pain, frontal headache of variable degree on the side of the affected eye

Systemic >> vagal symptoms (nausea and vomiting, abdominal cramps, bradycardia or arrhythmia

Signs:

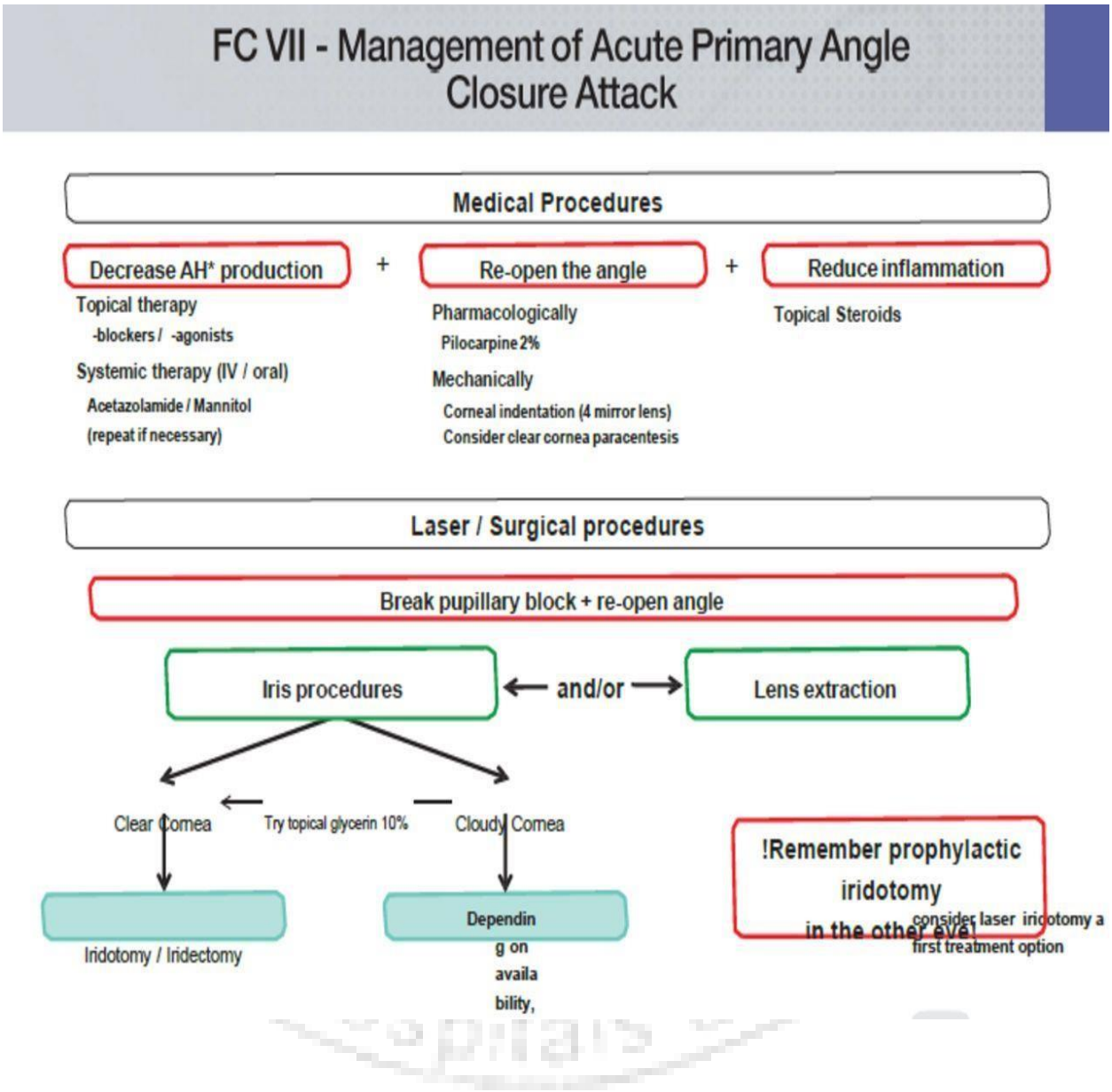
1- Slitlamp

- Venous congestion and ciliary injection
- Corneal epithelial edema
- Shallow or flat peripheral anterior chamber
- Pupil mid-dilated and irreactive vertically oval
- Fundus: the disc may be normal or show glaucomatous excavation; disc oedema, with venous congestion and retinal haemorrhages possible

2-Gonioscopy shows extensive iridotrabecular contact

360° 3-High IOP, often above 40 mmHg

Management (European glaucoma society guidelines)



A: Medical Treatment Medical

Serves to lower IOP, to relieve the symptoms and help clear the cornea so that LPI is possible. All the steps of medical therapy below should be implemented concurrently.

1 Reduction of aqueous production

1- Systemic carbonic anhydrase inhibitors (CAIs). 250mg (if IOP above 50mmhg) – two tablets

(Possible contraindication in people with poor renal function or sulfa allergy)

2- Topical beta-blockers and alpha-agonists. **(Check contraindications first)**

2 Dehydration of vitreous body

Hyperosmotics are the effective agents but carry significant systemic risk in some patients:

1- Patients must be evaluated for heart or kidney disease because hyperosmotics increase blood volume which increases the load in the heart.

2- Glycerol may alter glucose blood levels and should not be given to diabetics

- glycerol 1.0 – 1.5 g/Kg orally
- mannitol 1.0 – 2.0 g/Kg IV over 30 minutes (e.g. for a 70 kg patient 350 mL to 700 mL of 20% mannitol IV)

3 Pupillary constriction

pilocarpine 1% or 2%.

Miotics are likely to constrict the pupil only after IOP has been lowered.

Miotics in large doses can cause systemic side effects due to trans-nasal absorption leading to abdominal spasms

4 Reduction of inflammation

Intensive topical steroid, e.g., every 5 minutes for three times, then 4-6 times daily, depending on duration of raised IOP and severity of inflammation.

B: Laser and surgical treatment

Nd:YAG LPI should be attempted if the cornea is sufficiently clear.

Thermal laser pre-treatment (e.g,. argon) of dark irides reduces total Nd:YAG energy required –

Surgical iridectomy may be required when Nd:YAG LPI is not possible.

Eye Chemical Burns Protocol

Chemical burns to the eye can be divided into three categories:

- **Alkali burns** : are the most dangerous. Alkalis-chemicals that have a high pH- penetrate the surface of the eye and can cause severe injury to both the external structures like the cornea and the internal structures like the lens. In general, more damage occurs with higher pH
- **Acid burns** : usually less severe than alkali burns, because they do not penetrate into the eye as readily as alkaline substances. The exception is a hydrofluoric acid burn, which is as dangerous as an alkali burn. Acids usually damage only the ant segment of the eye ; however, they can cause serious damage to the cornea and also may result in blindness.
- **Irritants** : are substances that have a neutral pH and tend to cause more discomfort to the eye than actual damage.

Ocular Chemical Burn Symptoms :

Early signs and symptoms of a chemical eye burn are:

- Redness , Pain ,Irritation ,Tearing ,Inability to keep the eye open
- Sensation of something in the eye ,Swelling of the eyelids
- Blurred vision

- A true loss of vision signifies a very serious burn. Glaucoma, or an increase of the pressure inside the eye, can occur, but may be delayed by hours to days.

Management Protocol For ocular chemical Burns:

A,B,C,D

A: For all chemical injuries, **the first thing** you should do is **open the eyelids & immediately irrigate** the eye thoroughly. Ideally, specific eye irrigating solutions should be used for this, but if none are available regular tap water will do just fine.

- Begin washing the eye before taking any other action and continue for at least 15-30 minutes with sterile isotonic saline solution. If sterile saline is not available, use cold tap water. The longer a chemical is in your eye, the more damage will occur. Diluting the substance and washing awayany particles that may have been in the chemical are extremely important.

B: Short History to know **type of chemical** exposed , time of exposure ,Symptoms .

C: **Examine The Eye** starting from structures surrounding the eye ,eye lids ,lashes , cornea , lens towards the posterior segment .

- Eyelids, in particular, require careful assessment. Evert the eyelids to look for foreign material.
- stain the cornea with a dye called fluorescein to help determine the extent of the damage.

Acute chemical burns grading on the basis of corneal clarity , Severity of limbal ischemia

Grade 1	Clear cornea	No limbal ischemia	Excellent prognosis
Grade 2	Haze cornea but iris details are visible	Less than 1/3 limbal ischemia	Good prognosis
Grade 3	Total loss of corneal epithelium with stromal haze obscuring iris details	1/3 – ½ limbal ischemia	Guarded prognosis
Grade 4	Opaque cornea	More than ½ limbal ischemia	Poor prognosis

If the burns are minor (G1,2) , you will send the patient home with topical antibiotic ,lubricants eye drops and oral analgesics. Occasionally, you may need to prescribe dilating eye drops to help with comfort and relieve pain. you may cover the eye with an eye patch,or therapeutic CL to enhance the epithelization .

Any significant burn, especially an **alkali** or hydrofluoric **acid** burn, may require admission to the hospital or you will need prolonged therapy with potentially many medications to heal your eye.

- Until the surface of the eye heals, it is at a higher risk for an infection; therefore, topical antibiotics may be used in the form of eye drops or ointments.
- Topical steroids may be used to reduce inflammation and to facilitate healing early in the recovery period after a serious chemical injury. These medications should be used judiciously under the guidance of cornea specialist , because they can cause long-term complications, such as infections and glaucoma.
- Other medications used to support corneal repair include topical citrate and ascorbate drops, oral antibiotics (for example, tetracycline and doxycycline), and oral vitamin C.
- If your eye pressure is too high, glaucoma medications may be used temporarily to control the pressure.
- Pain medications by mouth may be necessary, and dilating eye drops are often also used to control pain and to aid recovery.

D: Close Follow up within 24 h - 48h at cornea unit.

Surgical measures may be necessary after severe chemical injuries when the initial injury has healed according to the damage.

Extraocular foreign body removal

1-

corneal foreign body

2-

conjuctival foreign body

1-
corneal foreign body

Indications:

Corneal foreign body removal is indicated when a foreign body is on the cornea.

Anesthesia:

Anesthesia is necessary prior to foreign body removal and usually facilitates the initial eye examination. Instill a topical anesthetic ophthalmic solution (eg, proparacaine 0.5% [Alcaine, Ophthetic]).

Equipment:

Equipment used for corneal foreign body removal includes the following: Topical anesthetic ophthalmic solution (eg, proparacaine 0.5% [Alcaine, Ophthetic]) Fluorescein strips Cotton-tipped applicator Irrigation fluid with plastic syringe Device to remove the foreign body Eye spud (specialized equipment designed for the removal of corneal foreign bodies).

The tip is less sharp than a needle, so iatrogenic injury is less likely to occur during the procedure.

A sterile 25-gauge needle, placed onto a syringe (1-3 mm), can be used.

Some clinicians like to bend the needle at a slight angle. Loupes or a slit lamp

Topical antibiotic ophthalmic ointment (eg, erythromycin) or ophthalmic drops .)

Eye patch

Positioning:

Have the patient press their face against the forehead strap and chin rest as demonstrated below so that the patient cannot move his head (and, hence, eye) forward toward the eye spud or needle during removal of the foreign body.

The clinician’s hand should be similarly anchored, either against the patient's face or on part of the slit lamp itself. Again, this prevents the clinician from inadvertently penetrating the patient's cornea with the spud or needle during the procedure.

When removing an object from the left eye, place hand on the left maxillary bone.

When removing an object from the right eye, place hand against the bridge of the nose or the infranasal aspect of the face.

Technique

Technique is as follows:

Explain the procedure, benefits, risks, and complications to the patient or the patient's representative and obtain informed consent.

Place 2 drops of anesthetic ophthalmic solution inside the lower eyelid

Apply a wet fluorescein strip inside the lower eyelid to instill fluorescein onto the cornea. Under ultraviolet light, examine the cornea to locate the foreign body. Document a negative Seidel sign. (A positive Seidel sign indicates corneal penetration with oozing aqueous humor; it appears under ultraviolet light as a "dark waterfall," clearing away excess fluorescein on the cornea.)

Instilling fluorescein onto the cornea. Inspect the lower eyelid while the patient looks up.

Inspect the upper eyelid by everting with an applicator while the patient looks down. Sweep the recesses of the upper conjunctival fornix.

If the foreign body is superficial, irrigate the eye to moisten the cornea and attempt to remove the foreign body by using a gentle rolling motion with a wetted cotton-tipped applicator.

Take care not to apply pressure, which may push the foreign body deeper into the cornea, or scrape, which may create a large corneal abrasion.

An embedded foreign body cannot be removed with irrigation or with a cotton-tipped applicator.

An embedded foreign body can be removed by using a gentle flicking motion with an eye spud, if available, or with a 25- or 27-gauge needle. Place the hub of the needle on the tip of a cotton swab or a 3-mL syringe. Approach the cornea from the side, with the needle in a plane tangent to the cornea and the bevel away from the corneal surface. This minimizes the chance of corneal perforation. Once dislodged from its embedded position on the cornea, remaining corneal debris can be removed with a wetted cotton-tipped applicator

Document a negative Seidel sign after the foreign body is removed

Postoperative treatment :

Topical ophthalmic antibiotics:

Ophthalmic antibiotic ointments (eg, bacitracin, ciprofloxacin) have an advantage by functioning as a lubricant.

Ophthalmic solutions (eg, sulfacetamide, ofloxacin) are easier to apply and, therefore, enhance patient compliance.

Corticosteroid ophthalmic solutions or ointments should be avoided because they increase the likelihood of superinfection and slow healing.

Pain control

Opioid analgesic agents (eg, hydrocodone/acetaminophen [Vicodin], oxycodone/acetaminophen [Percocet]) can be used to relieve moderate to severe pain and have been found to allow patients to sleep more comfortably at night.

Nonsteroidal anti-inflammatory drug (NSAID) ophthalmic solutions (eg, ketorolac) can provide significant pain relief and have not been found to slow healing.

Patching

The use of patching has been controversial. Most recently, studies have shown that corneal abrasions due to a foreign body are best treated without eye patching.

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2.conjuctival

foreign body removal :

planning
Procedural

Before pressing on the eyelid or eye, make certain that no penetrating injury of the eye has occurred.

Such an injury should always be considered when there is a history of power tool use or of hammering metal with metal .

If something on the bulbar conjunctiva is not easily removable with a cotton swab, it could be a penetrating foreign body or a conjunctival pigmented

Periprocedural Care

Equipment:

The following equipment is required for removal of a conjunctival foreign body

: Topical anesthetic drops

A cotton swab moistened with saline A cotton applicator stick or paper clip for eversion of the upper lid

A saline irrigation bottle

The patient should be comfortably seated, with the back of the head against a firm surface. Alternatively, the patient can be recumbent, with the head on a pillow. If a slit lamp is available, the procedure may be performed at the instrument lamp, with the patient's head in the chin rest and the forehead pressed against the plastic strap**.**

Technique:

If a patient complains that something has gotten into the eye, it is usually in the upper or lower conjunctival fornix, more
commonly the upper one. Do not be content with finding only 1 foreign body: search for more than 1, and expect to find several more. First, inspect the eye for any foreign bodies on the bulbar conjunctiva, which overlies the sclera (the white of the eye).
Have the patient look up, down, left, and right. The eye may be somewhat reddened if a conjunctival foreign body is present.
Next, inspect the inferior conjunctival cul-de-sac by having the patient look up while the examiner pulls the lower lid down.
Elevation of the upper lid while the patient looks down usually does not permit effective visualization of foreign bodies in the upper conjunctival cul-de-sac. Upper lid eversion is required.
Have the patient look down, then place a cotton stick applicator or paper clip in the upper lid recess. While the patient continues to look down, grasp the upper lid margin lashes with the fingertips of one hand, and pull the lid downward and toward you . The conjunctival surface of the inside of the upper lid is now visible.

Remove the cotton applicator stick or paper clip, and use a thumb to press the lid margin of the everted lid against the patient's brow.

The stiff tarsal plate usually keeps the upper lid everted after the swab is removed, as long as the patient continues looking down. Looking up returns the lid to its usual position.

A foreign body on the conjunctival surface of the upper lid will usually be easily seen when the lid is everted

If the patient's discomfort before attempted foreign body removal precludes the use of these techniques, 2 drops of local anesthetic may be placed into the eye to allow examination and treatment. Once a drop of anesthetic is placed in the eye of a patient with foreign body sensation, the physician is
committed to either finding a conjunctival foreign body or finding another definite reason for the foreign body sensation. At this point, relief of symptoms is no longer a measure of successful diagnosis and treatment. Be sure that the patient's symptoms are resolved by the physician's actions, not by the anesthetic agent. If, after a topical anesthetic agent has been used, doubt remains as to whether the cause of the foreign body sensation has been found, wait at least 30 minutes for the anesthetic to wear off, then reassess the patient to ensure that the problem has been properly addressed. In a patient with successful removal of a conjunctival foreign body, no symptoms of foreign body sensation should return after the topical anesthetic has worn off. After conjunctival foreign bodies are removed, always examine the patient’s cornea carefully and stain it with fluorescein; this should be done even if a foreign body was not found on the conjunctiva.

Foreign bodies of the upper lid often cause vertical scratches in the cornea as a result of the foreign body having rubbed against the cornea with each blink. Transparent foreign bodies can be invisible in the tear film and become more visible using fluorescein stain. If no topical anesthetic was used, removal of the foreign body causes immediate and almost total resolution of the symptoms—and evokes marked gratitude on the part of the patient—in most cases. If significant symptoms persist, consider the possibility of a second foreign body or a significant corneal abrasion (either causally related to the foreign body or occurring as an independent injury). Following removal of the conjunctival foreign body, two drops of a topical broad-spectrum antibiotic drop should be placed in the affected eye. If there is no corneal abrasion or significant inflammation of the eye, no further treatment or follow-up is necessary. The patient should be instructed to return if the foreign body sensation returns or if any symptoms of pain, redness, or visual changes occur.

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Globe rupture

Once globe rupture is suspected:

1- urgent ophthalmologic consultation is required.

2- The patient should first be treated for any other potentially life-threatening injuries and remain NPO (nothing by mouth).

3- The affected eye should be protected using a Fox shield, cup, or another protective device.

4- Removal of any foreign bodies should not occur until the patient is in surgery.

5- Maneuvers that will increase intraocular pressure should be avoided (i.e., tonometry, lid retraction, or ocular ultrasound).

6- In patients who are awake and alert, it is recommended to reduce stressors that may increase intraocular pressure. Antiemetic's, pain control, and bed rest with elevation of the head of the bed to 30 degrees will aid in this process.

7- Although no specific prophylactic antibiotic regimen exists, preservative-free, topical antibiotic drops before surgery can be given prophylactically to theoretically decrease the risk of endophthalmitis.

8- In unstable patients or where intubation is required, it is essential to choose systemic medications that do not raise intraocular pressure.

9- Open globe injuries are tetanus-prone wounds, and patients should receive a booster if their immunization history is uncertain or incomplete. 10- CT orbit if intra ocular FB is suspected .

Surgical management :-

1. Surgical management by an ophthalmologist should commence as soon as the patient can safely undergo surgery, a delay in surgical intervention may lead to worse final visual outcomes and increase the risk of postoperative endophthalmitis.
2. Initial surgical management of a globe rupture includes microsurgical corneal and/or scleral wound repair.

   Corneal lacerations can be closed with 10-0 nylon interrupted sutures (corneal suture knots should be buried to prevent postoperative complications).

   Scleral wounds may be closed with 7-0, 8-0, or 9-0 no absorbable sutures / 6-0,7-0, 8-0 vicryl and an attempt should be made to burry or cover these suture knots as well.

   Before closure, incarcerated uvea must be reposited or excised, and prolapsed vitreous should be excised from the wound.

If there is hyphemia AC wash should be done .

If there is rupture of lens capsule removal of lens mater should be done .

The anterior chamber should also be reformed and repressurized to an appropriate IOP.

Following wound repair, intravitreal or intracameral antibiotics are necessary, as they have been shown to decrease the risk of endophthalmitis and also during the operation we will use vancomycin and fortum antibiotic on the saline used during surgery .

3. If there is a perforating injury that affects that eye posteriorly, further surgical intervention may be necessary.

   While anterior wounds require suturing, the surgeon may choose to leave the posterior wound unrepaired so that extrusion of vitreous or retinal disruption during attempted closure is avoidable.

Urinary Tract Infection (UTI) management protocol

Symptoms & Signs:

- Cystitis or lower UTI:

Dysuria, frequency, urgency, nocturia, suprapubic pain or tenderness, offensive urine or frank haematuria

- Pyelonephritis or upper UTI:

Fever, chills, night sweats, rigors, nausea, vomiting, loin pain, renal angle tenderness with progression to SIRS (Systemic Inflammatory Response Syndrome) & possibility of Shock

Investigations:

- Dipstick urine: positive leucocyte esterase +/- nitrite reductase
- Urine microscopy: MSU (Mid-Stream Urine exam) for pyuria
- Urine culture
- Imaging in complicated cases: Ultrasound, or CT- abdomen

Definitions:

Uncomplicated AND Complicated UTI

- **Uncomplicated** UTI – symptomatic infection in a structurally and functionally normal urinary tract in non-pregnant women. Most commonly caused by E. coli.

- **Complicated** UTI – symptomatic infection associated with factors that increase the risk of serious complications or treatment failure. This includes all men, pregnant women, patients with structural or functional abnormalities of the urinary tract (e.g. neurogenic bladder, nephrolithiasis) or other concomitant immunocompromising diseases (e.g. diabetes).Associated with a wider range of pathogens including E. coli, Klebsiella, Proteus, Pseudomonas species.

Treatment:

Empirical use of Antimicrobials according to Infection

Urinary tract infection / Cystitis

Initial Treatment Strategies		
Standard Regimen	**Choose one option from row A (monotherapy)**	**Duration**
Acute Un-complicated Cystitis	Nitrofurantion 100 mg PO /12 hr TMP-SMX DS 160-800mg PO /12hr Fosfomycin 3gm PO once Amox-acillin-clavulenic 625mg/8hr ciprofloxacin 250 mg PO /12hr Levofloxacin 250mg PO/24 hr	(5 days) (3days) (single dose) (5-7days) (3-5days) (3-5days)
Acute complicated cystitis	**Choose one option from row A (monotherapy)**	**Duration**
	TMP-SMX 160-800 mg/12hr Ciprofloxacin 500 mg PO/12 hrLevofloxacin 750mg PO /24hr	(7-10 days) forall choices
	In case of inpatient or oral intolerance Ciprofloxacin 500 mg PO/12 hr Levofloxacin 750/24h Ceftriaxone 1g/24hr Piperacillin -tazobactam 4.5g/6hr	 (3-5 days) (3-5 days) (7days) (5-14 days)

Empirical use of Antimicrobials according to Infection

Urinary tract infection / Pyelonephritis

Initial Treatment Strategies for Pyelonephritis			
StandardReg-imen		Choose one option from row A (monotherapy)	Duration
Acute uncomplicated pyelonephritis	A	**In case of outpatient oral ttt** TMP-SMX 160-800mg PO /12hr Ciprofloxacin 500mg PO/12 hr Levofloxacin 750mg PO/24hr Cefpodoxime200mg/12hr Cefexime 400mg /24hr	(10-14 days) (5-7 days) (5-7 days) (10 days) (10 days)
		In case of inpatients and oralintolerance Ceftriaxone1g/24 hr Ciprofloxacin 400mgiv /12hr Levofloxacin 750mgiv /24hr Pip-eracillin -tazobactam 4.5g/6hr	(5-14 days) (5-7 days) (5-7 days) (5-14 days)
Acute compli-cated pyelone-phritis		Choose one option from row A	Duration
	A	**No MDR risk** Ceftriaxone1g/24 hr Cip-rofloxacin 400 iv/12hr or 500 po /12 hrLevofloxacin 750/24 hr	(7-10 days)
	A	Choose one option from row A **MDR risk** Cefepime 2g/12 hr Ertapenem 1g/24hr Imipenem 500mg/6hr Meropenem 1g/8hr Piperacillin -tazobactam 4.5 g /6hr Amikacin 15-20mg/kg/24hr	(7-14 days)

Empirical use of Antimicrobials according to Infection

Urinary Tract Infection / Prostaitis

Initial Treatment Strategies for Prostaitis			
Standard Regimen		Choose one option from row A(mono-therapy) or combination between one of A one with B	Duration
Acute Febrile Prostaitis	A	Ciprofloxacin oral 500/12hr or 400 /12hr iv Levofloxacin 750/24hr Cefepime mg1g/12 hr Piperacillin -tazobactam 4.5mg /8hr Moxifloxacin 400mg /24 hr	(5-7 days) (5-7days) (3-5days) (5-7days) (15 days)
	B	Gentamycin 5mg/kg QD Amikacin 15/kg QD	
Chronic prostatitis	Ciprofloxacin 500 po/12 hr or 400 iv /12 hr Levofloxacin 750/24 hr		14 – 21 days
Chronic prostatitis with certain pathogens detected		Choose one option from row A according to pathogen	Duration
	A	Azithromycin 5oomg /24 Hr Vibramycin100mg /12 hr Metronidazole 500mg/8hr	Only for c .trachomatis (3w) c.tracomatis or mycoplasma (10d) t.vaginal-is(14d)

Empirical use of Antimicrobials according to Infection

ASymptomatic Bacteruria

Initial Treatment Strategies for Asymptomatic bacteruria			
Standard Regimen		Choose one option from row A(monotherapy)	Duration
	A	Pregnant female Nitrofurantion 100/12hr AMOX-CLAV 625 /8hr Fosfomycin 3g once Cephalexin 500mg/6h Cefpodoxime 100mg/12 hr	(5-7days) (3-7days) (single dose) (3-7 days) (3 -7days)
Patient undergo urological procedure Cefazolin 2g iv or im /24h Ciprofloxacin 400mgIV OD			(0.5 – 1 hr before procedure)
Post renal transplantation Nitrofurantion 100mg PO/12 hr Ciprofloxacin 250mgPO/12 hr Fosfomycin 3g Cephalexin 500/12 hr			(5d) (3d) Single Dose (5 d)

Initial Treatment Strategies for catheter associated UTI			
Standard Regimen		Choose one option from row A(monotherapy)	Duration
complicated cystitis	A	Ertapenem 1g/24hr Imipenem 500mg /6hr Meropenem 1g /8hr Amikin 15-20mg/kg/24hr	(7-14 days) forall
Un- complicated cystitis	A	NitrofurantionPO 100mg/12hr TMP-SMX DS 160-800mg/12hr Po Fosfomycin 3g Ciprofloxacin 250mg/12hr Or 500mg/24hr PO Levofloxacin 250mg/24hr PO	(5days) (3days) single dose (3days) (3days)

Plasmapheresis protocol

(Therapeutic plasma exchange, TPE)

Prepared by: *Dr Walid Hemida, Consultant nephrologist, Damanhur medical national institute.*

Introduction:

Apheresis is derived from the Greek word, which means withdrawal. Plasmapheresis is a process in which there is removal of the patient's plasma, and its substitution by other fluids. In therapeutic procedures, the blood of a patient is modulated in the extracorporeal circuit with the aim of removing many large molecular weight (MW) substances from plasma, including pathogenic antibodies, cryoglobulins, immune complexes, lipoproteins, cells or cell fragments, toxic substances or other substances that are either proven or presumed to have a pathogenic effect.

Technique:

Plasmapheresis can be carried out either by centrifugal cell separators or with hollow-fiber plasma filters with wide pores (0.2 to 0.5 μm) using standard hemodialysis (HD) equipment. Centrifugal devices allow withdrawal of plasma from a bowl with either continuous or intermittent return of blood cells to the patient.

Both methods of plasma exchange require large volumes of colloid replacement. A single plasma volume exchange will lower plasma macromolecule levels by approximately 60%, and five exchanges over 5 to 10 days will clear 90% of the total body immunoglobulin.

Vascular access:

Vascular access is usually achieved using standard central venous catheters, but also, existing arteriovenous fistulas (AVFs) can be used if available. The femoral access is the most preferable one, as the blood and replacement fluids returned to the venous line take a longer journey from the femoral vein to be diluted before reaching the heart.

Why femoral vein vascular access is preferable? To avoid arrhythmia that may occur due to return of hypocalcaemic blood to A-V node, low risk of bleeding, more easily.

Anticoagulation:

Citrate is used as anticoagulant with centrifugal plasma exchange and heparin for membrane plasma filtration; however, citrate is superior for patients at higher bleeding risk in view of its lack of systemic anticoagulation.

In case of heparin use, higher doses may be needed than in HD as a result of increased losses during the procedure (heparin is protein bound). Bolus doses of unfractionated heparin of 2000 to 5000 U are given initially and then 500 to 2000 U/h. Anticoagulant is administered pre-filter. Low molecular weight heparin (LMWH) is also used with a single bolus dose at initiation of exchange.

Replacement fluid:

Replacement solely with crystalloid is contraindicated because of the need to maintain colloid oncotic pressure. So, the ideal replacement fluids will be either fresh frozen plasma (FFP) or human albumin saline 5%.

- **FFP**: is preferable as it contains normal proteins, coagulation factors, Igs and complements but has hazards of infection, allergy, contains citrate (causes hypocalcemia) and needs compatibility.
    - Each plasma unit contains from 200 to 250 ml , so to prepare one plasma volume in 70 kg patient we need from 10 to 12 units of plasma
- **Albumin saline** 5%: no hazards of infection, less allergy, no compatibility but more expensive, has defect in electrolytes and coagulation factors.
    - To prepare 500 ml Albumin saline 5%: 125 ml Human albumin 20% (2.5 bottles of 50 ml) to be added to 375 ml normal saline 0.9%.

Procedure

Pre-procedure:

- Consent: Need signed consent form in the sheet.
- Weight (wt) of the patient in Kg
- Calculate the estimated plasma volume (EPV) through one of the following equations:
  1. EPV = 0.065 X Body wt in kg X [1 – Hct]
  2. EPV = **35 - 40 ml X Body wt in kg**
     { 35 ml for patient with normal Hct & 40 ml for low Hct}
     Example: A patient of 70 kg and anemic (Hb%: 8g/dl)
     The EPV will be = **70 X 40 = 2800 ml**

Complications:

Hypocalcaemia

- Cause: FFP (citrate), Albumin (poor calcium)
- Presentation: starting from shivering, headache, headedness, twitches, tremors, muscle contractions up to tetany, arrhythmia
- Prophylaxis: one amp Ca glocunate for each litre of albumin saline & in case of FFP one amp for each 2 units
- Treatment: Ca infusion slowly

Vascular access

- Hematoma, pneumothorax, or retroperitoneal bleeding.

Hypotension:

- Cause either due to blood in circuits or decrease the intravascular oncotic pressure.

Bleeding risk:

- Cause: due to v. access, or defect of coagulation factors in albumin.

- Treatment: 2 units of FFP at the end of each session replaced with Albumin saline (if no contraindication).
- in case of contraindication to FFP: do PT, PTT, INR before the next session and if raised > 1.5 times of the normal range postpone for the next day or give FFP.

Hypersensitivity reaction: Prophylaxis (premedication):

- Prednisolone 50 mg 13, 7, 1 hour before start.

- Diphenhydramine 50 mg 1 hour before start.

- Ephedrine.

Hypokalemia (in case of albumin saline only not FFP)

- K^+ decreases by about 25% each session in case of albumin.
- Prophylaxis: Adding 20 meq Kcl to each liter replaced fluid.

Arrhythmia

- Mainly due to hypocalcemia.

ACE Inhibitors: (may cause anaphylactic reaction)

- Due to bradykinin release (filter membrane).
- Prophylaxis: D/C ACEI 48 hours before starting the procedure.

Indication for Plasmapheresis (TPE):

According to The Writing Committee of the Journal of Clinical Apheresis (JCA) Special Issue 2019, The American Academy of Neurology (AAN), National Institutes of Health (NIH) and The American Society for Apheresis (ASFA) have been listed the indications for TPE into categories:

Category I and Category II Indications

Plasmapheresis is considered an acceptable primary or adjunctive treatment either standalone or in conjunction with other modes of treatment like in:

- Acute inflammatory demyelinating polyneuropathy (Guillain-Barré Syndrome) when ONE of the following is established:
- Anti-glomerular basement membrane disease (Goodpasture's syndrome)
- Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP)
- Cryoglobulinemia
- Hyperviscosity syndrome (e.g., Waldenström's macroglobulinemia, multiple myeloma)
- Myasthenia gravis in preparation for surgery OR with respiratory crisis
- Pediatric post infectious autoimmune neuropsychiatric disorders (PANDAS) associated with streptococcal infections and Syndenham's chorea (severe exacerbation)
- Paraproteinemic polyneuropathy associated with monoclonal gammopathies of undetermined significance (e.g., MGUS)
- Thrombotic thrombocytopenic purpura (TTP)
- Rapidly progressive glomerulonephritis (RPGN) (e.g., Wegener's)
- Vasculitis, ANCA-associated (AAV)
- Lambert-Eaton myasthenic syndrome (LEMS)
- Catastrophic Antiphospholipid syndrome (CAPS)
- Focal segmental glomerulosclerosis-Recurrent in kidney transplant
- Mushroom poisoning
- Systemic lupus erythematosus (Severe complications)
- Transplantation, renal, ABO compatible (AM rejection)
- Transplantation, renal, ABO incompatible (Desensitization, living donor)

Category III and Category IV Indications

Optimum role of Plasmapheresis therapy is not established. Decision making should be individualized. Disorders in which published evidence demonstrates or suggests apheresis to be ineffective or harmful.

- ABO incompatible solid organ transplantation – liver
- Acute disseminated encephalomyelitis

- Acute liver failure
- Amyloidosis, systemic
- Amyotropic lateral sclerosis
- Autoimmune hemolytic anemia (warm autoimmune hemolytic anemia; cold agglutinin)
- Permatomyositis or polymyositis
- Heart transplant rejection
- Hematopoietic stem call disorders (aplastic anemia; pure red cell aplasia)
- Hypertriglyceridemic pancreatitis
- Idiopathic thrombocytopenic purpura
- Multiple sclerosis
- Pemphigus vulgaris
- Rheumatoid arthritis
- Schizophrenia
- Scleroderma (progressive systemic sclerosis)
- Sepsis
- Stiff-person syndrome
- Thyrotoxicosis

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Protocol for management of hyperkalemia

Prepared by: Dr Ghada Mashaal, Lecturer of nephrology, Damanhur teaching hospital.

Introduction

The intracellular prevalence of potassium is the result of a sodium-potassium (Na^+ -K^+) exchange pump on cell membrane that moves Na^+ out of cells and moves K^+ into cells.

The total body potassium in healthy adults is about 50 mEqlkg, and only 2% is in the extracellular fluid. Since the plasma accounts for about 20% of the extracellular fluid, the potassium content of plasma is only 0.4% of the total body potassium.

Hyperkalemia is defined as a serum potassium concentration greater than approximately 5.0-5.5 mEq/L in adults; the range in infants and children is age dependent. Levels higher than 7 mEq/L can lead to significant hemodynamic and neurologic consequences; levels exceeding 8.5 mEq/L can cause respiratory paralysis or cardiac arrest and can quickly be fatal.

Degrees of hyperkalemia are generally defined as follows:

- 5.5-6.0 mEq/L – Mild
- 6.1-7.0 mEq/L – Moderate
- ≥7.0 mEq/L – Severe

Levels higher than 7 mEq/L can lead to significant hemodynamic and neurologic consequences. Levels exceeding 8.5 mEq/L can cause respiratory paralysis or cardiac arrest and can quickly be fatal.

Etiology

Hyperkalemia can result from increased potassium intake, decreased potassium excretion, or a shift of potassium from the intracellular to the extracellular space. The most common causes involve decreased excretion. Alone, excessive intake or an extracellular shift is distinctly uncommon. Often, several disorders are present simultaneously.

1- **Increased potassium intake:** Increased intake may result from the following:

- High-potassium, low-sodium diets.
- Ingestion of potassium supplements – Ingested amounts would have to be massive to be the sole cause of hyperkalemia, but even relatively small amounts can produce hyperkalemia in a patient with impaired renal excretion.
- High concentrations of potassium in IV fluid preparations (eg, total parenteral nutrition formulas).
- Dietary salt substitutes – Several "no-salt" or "low-salt" substitutes contain about 10-12 mEq of potassium per gram and can be dangerous, especially with diminished kidney function.
- Penicillin G potassium therapy.
- Packed red blood cell transfusion (risk peaks at 2-3 weeks of cell storage).
- Cardioplegia solutions – These contain 20-30 mmol/L of potassium chloride.

2- **Decreased potassium excretion:**

Almost all patients who present with persistent hyperkalemia have impaired renal excretion of potassium. Mild degrees of renal failure generally do not result in resting hyperkalemia, because of compensation by adaptive mechanisms in the kidneys and GI tract. However, once the GFR falls below 15-20 mL/min, significant hyperkalemia can occur, even in the absence of an abnormally large potassium load. The simple lack of nephron mass prevents normal potassium homeostasis.

3- Drug effects or renal tubular acidosis, can decrease renal potassium excretion and cause hyperkalemia even in individuals with normal or only mildly decreased kidney function. Two other causes of decreased excretion of potassium are reduced distal sodium delivery and reduced tubular fluid flow rate.

Medications that can decrease potassium excretion include the following:

Potassium-sparing diuretics (eg, spironolactone, triamterene, amiloride)	Trimethoprim-sulfamethoxazole
NSAIDs	Mineralocorticoid receptor antagonists (MRAs)
ACE inhibitors	Heparin
Angiotensin-receptor blockers (ARBs)	Ketoconazole
Cyclosporine or tacrolimus	Metyrapone
Pentamidine	Herbs

4- Disorders that can cause type IV renal tubular acidosis, resulting in hyperkalemia, include the following:

Diabetes mellitus	Primary Addison syndrome due to autoimmune disease, tuberculosis, or infarct
Sickle cell disease or trait	Enzyme deficiencies
Lower urinary tract obstruction	Genetic disorders
Adrenal insufficiency	

5- Shift of potassium into extracellular space:

Like increased intake, this is rarely the sole cause of hyperkalemia, because the mechanisms for renal excretion are very efficient. However, the inability to transport potassium intracellularly exacerbates hyperkalemia in individuals who have impaired renal excretion.

Factors that can shift potassium into the extracellular space include the following:

Metabolic acidosis	Fluoride toxicity
Beta-adrenergic blockade	Cyclosporine
Acute tubular necrosis	Methotrexate
Electrical burns	Propofol infusion syndrome
Thermal burns	Rhabdomyolysis
Cell depolarization	Tumor lysis syndrome
Head trauma	Succinylcholine
Digitalis toxicity	

6- Genetic disorders: Genetic disorders that can result in hyperkalemia include the following:

Glomerulopathy with fibronectin deposits (GFND)	Disorders of chloride homeostasis
Disorders of steroid metabolism and mineralocorticoid receptors	Nephronophthisis
Congenital hypoaldosteronism	Hyperkalemic periodic paralysis (HYPP)
Pseudohypoaldosteronism	

Pseudohyperkalemia:

Means hemolysis and excessive leakage of potassium from cells during or after blood collection, has been reported in 20% of blood samples that show hyperkalemia.

Causes of pseudohyperkalemia:

Traumatic hemolysis during collection of the blood sample(most common cause)
Fist clenching (k^+ release from muscles) or tourniquet stasis
K^+ release from WBCS in severe leukocytosis(>50000/mm^3) or from platelets in severe thrombocytosis(> million/mm^3)
K^+ release during clot formation (serum $K^=$ > plasma $K^=$)

Signs and symptoms of hyperkalemia:

Many individuals with hyperkalemia are asymptomatic. When present, symptoms are nonspecific and predominantly related to muscular or cardiac function. Weakness and fatigue are the most common complaints. Occasionally, patients may report the following:

Frank muscle paralysis	Chest pain
Dyspnea	Nausea or vomiting
Palpitations	Paresthesia.

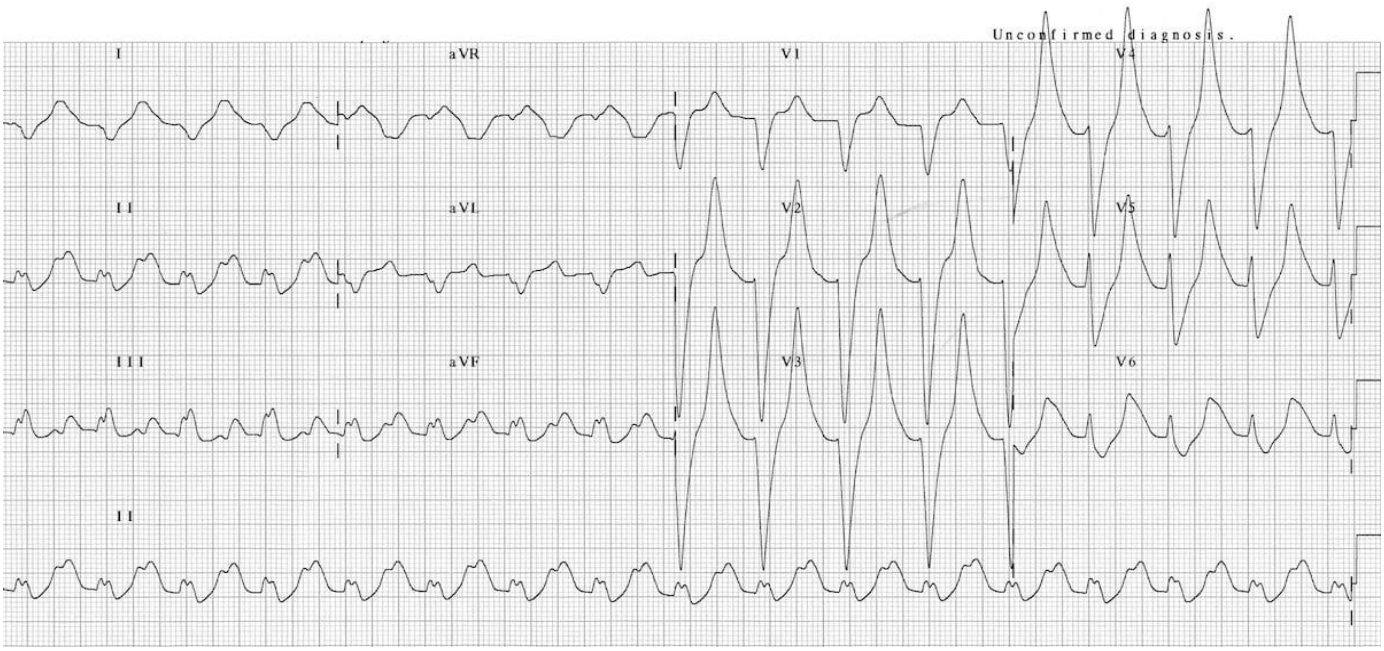
ECG changes in hyperkalemia:

ECG changes have a sequential progression, which roughly correlate with the potassium level. Early changes of hyperkalemia include tall, peaked T waves with a narrow base, best seen in precordial leads; shortened QT interval; and ST-segment depression. These changes are typically seen at a serum potassium level of 5.5-6.5 mEq/L.

At a serum potassium level of 6.5-8.0 mEq/L, in addition to peaked T waves, the ECG shows the following:

- Prolonged PR interval
- Decreased or disappearing P wave
- Widening of the QRS
- Amplified R wave

Potassium level (mmol/L)	Mechanism	ECG changes
5.5 – 6.5	Repolarisation abnormalities	Peaked T waves
6.5 – 7.0	Progressive atrial paralysis	P wave widening/flattening PR prolongation P waves eventually disappear
7.0 – 9.0	Conduction abnormalities	**Bradyarrhythmias:** Sinus bradycardia; high-grade AV block with slow junctional and ventricular escape rhythms; slow AF Conduction blocks (bundle branch block, fascicular blocks) Prolonged QRS interval with bizarre QRS morphology
> 9.0	All of above	Development of sine wave appearance (pre-terminal rhythm) Asystole Ventricular fibrillation PEA with bizarre, wide complex rhythm



Management of hyperkalemia:

1- General principles of management:

- Urgent management of hyperkalemia is considered in three steps:
  - STEP 1: Antagonism of the cardiac effects of hyperkaliemia
  - STEP 2: Rapid reduction in serum potassium by redistribution into cells
  - STEP 3: Removal of potassium from the body
- It is mandatory that all patients with hyperkalemia (K level ≥6.0 mmol/L) should have an urgent 12-lead ECG performed and, if there are any hyperkalemic changes or if K level ≥ 6.5 mmol/L, should be on a cardiac monitor (minimum 3-lead continuous ECG monitoring).
- It is recommended that serum potassium is assessed at least 1, 2, 4, 6 and 24 hours after identification and treatment of moderate or severe hyperkaliemia..
- The following therapies are listed to explain the physiology and posology. A recommendation for a hierarchy of use is as per the attached algorithm.

- Some therapies are essentially holding maneuvers (Calcium, glucose/insulin, and salbutamol). In the absence of another treatable pathology (e.g. overt dehydration), definitive therapy – dialysis – will be necessary.

2- **Antagonism of cardiac effects**:

Calcium reduces myocardial excitability in the face of hyperkalemia. It is available as 10% calcium gluconate. **The dose is 30 mL of 10% of calcium gluconate, administered intravenously over 10 minutes via large IV access with continuous cardiac monitoring**. The effect is seen within **3 minutes and lasts 30-60 minutes. The dose may be repeated – after 5 to 10 minutes** – if no effect is seen or if ECG changes recur after initial improvement. It MUST NOT be administered via a line containing bicarbonate as it will precipitate as calcium carbonate. As an alternative to 10% calcium gluconate, 10% calcium chloride may be administered. The dose of 10% calcium chloride is 10 mL as it contains 3 times more calcium than calcium gluconate. Calcium chloride has been recommended in the setting of hemodynamic instability, including cardiac arrest. Calcium should be used cautiously in patients taking digoxin as hypercalcemia potentiates the action of digoxin and may precipitate myocardial toxicity. In this case, it is necessary to infuse it more slowly (over 30 min in 100 mL of 5% glucose) to allow for an even distribution of calcium in the extracellular compartment.

3- Redistribution of potassium into cells

A- Insulin and Glucose (off label use):

- Insulin lowers serum potassium by stimulation of the Na+ /K -ATPase. This effect is reliable, reproducible, dose-dependent and effective.
- Check pre-treatment blood glucose level prior to insulin and glucose administration.
- Administer 10 units of actrapid insulin (using an insulin syringe) with 50 mL of 50% glucose (25 g of glucose). This should be administered intravenously via a central venous access device/large vein via a syringe pump over 15-30 minutes. Monitor for phlebitis if 50% glucose is administered via a large peripheral vein.
- If pre-treatment blood glucose < 126 mg/dl administer 10% glucose at 50 mL/hour for 5 hours (25 g) o target blood glucose 72 – 126 mg/dl to titrate rate of infusion if required.

- Monitor blood glucose.
- Anticipate and treat hypoglycemia promptly.
- The effect on serum potassium begins in 10-20 minutes, peaks at 30-60 min, and last for 4-6 hours. In most patients, the serum potassium drops by 0.5-1.2 mmol/L with this treatment. The dose may be repeated if necessary.

B- ß2-Adrenergic Agonists (Salbutamol) (off label use)

- Salbutamol exerts its effects via activation of Na+/K+ -ATPase.
- The recommended dose is **10- 20 mg of nebulised salbutamol**. Its effects are seen at about 30 minutes and peak at 90 minutes, lasting for 2-6 hours. It reduces serum potassium levels by 0.5-1.0 mmol/L.
- The effects of salbutamol are attenuated in patients on ß-blockers and digoxin. It is unclear whether treatment with salbutamol has a significant additive effect to insulin on its own.
- Treatment with salbutamol may cause a significant tachycardia and should be used in caution in those with ischemic heart disease.

C- Sodium bicarbonate

Previously used routinely in the treatment of hyperkalemia, sodium bicarbonate is now reserved for hyperkalemia associated with renal failure and acidosis.

The recommended dose is **1.26% sodium bicarbonate infused intravenously at a rate determined by the patient's fluid status and degree of acidosis**. Hyperkalemia and metabolic acidosis with cardiac arrest should be treated with **50 mL of 8.4% sodium bicarbonate** (which is available on the arrest trolley). Sodium bicarbonate should be used with extreme caution in the following situations:

- In anuric or hypovolemic patients – the significant sodium load may result in symptomatic fluid overload.
- Hypocalcaemia – bicarbonate causes precipitation of calcium; the resultant fall in ionized calcium may result in tetany or fits.

- Patients with type 2 respiratory failure – potential for paradoxical acidosis within the central nervous system.

4- Removal of potassium from the body

- **Intravenous fluids:**

  Most cases of acute (or acute on chronic) kidney injury in hospital are a result of intravascular volume depletion (i.e. pre-renal). In these situations, correction of volume status with intravenous fluids may be sufficient to restore renal function and promote a kaliuresis.

- **Diuretics:**

  In certain cases, increasing renal potassium elimination with diuretics may be adequate to lower total body potassium. However, in the setting of renal insufficiency (chronic or acute) the effectiveness of diuretic therapy may be limited. The use of diuretics is only indicated for those patients who are fluid replete.

  In the acute setting, the diuretic most often used is intravenous furosemide. The dose will vary depending on renal function, but in those with significant renal impairment, up to 250 mg may be used to try and promote a kaliuresis: the effect is mild.

- **Sodium zirconium cyclosilicate**

  Sodium Zirconium Cyclosilicate (SZC) is a non-absorbed potassium binder that preferentially **exchanges H+ and Na+ for K+ and ammonium ions throughout the entire gastrointestinal tract**. The K+-binding capacity of SZC is up to **9 times greater than that of sodium polystyrenesulfonate**. NICE has approved SZC as an option in the treatment of acute life-threatening hyperkalemia alongside standard care in hospitalized patients Correction phase: **the recommended dose is 10 g orally three times a day until normokalaemia (serum K 4.0-5.0 mmol/L)** is achieved. Usual treatment duration is 24-48 hours, maximum duration is 72 hours. Sodium Zirconium Cyclosilicate should be discontinued after 72 hours if normokalemia not achieved. Maintenance phase: a dose of

5 g daily can be prescribed once normokalemia is achieved, this can be titratred up to 10 g per day or down to 5 g alternate days depending on serum K levels. The maintenance dose should be discontinued once hypokalaemia develops (serum K level < 4.0 mmol/L) the contents of the sachet should be emptied into a glass containing approximately 45 mL of water and stirred well. The powder will not dissolve. Advise the patient to drink the tasteless liquid while still cloudy, if the suspension settles it should be stirred again.

- **Cation-exchange resins**

Ion-exchange resins are cross-linked polymers containing acidic or basic structural units that can exchange either anions or cations on contact with a solution. The most commonly used cation-exchange resin used is Calcium Resonium®.

The onset of action is slow (> 4 hours) and efficacy is unpredictable therefore it should only be used in conjunction with other measures in the management of acute hyperkalemia. It is also poorly tolerated due to taste and constipation. Cation exchange resins have been associated with colonic necrosis (most commonly seen with sodium polystyrene sulfonate used in conjunction with sorbitol). Therefore, they should not be used in those with bowel obstruction or an ileus.

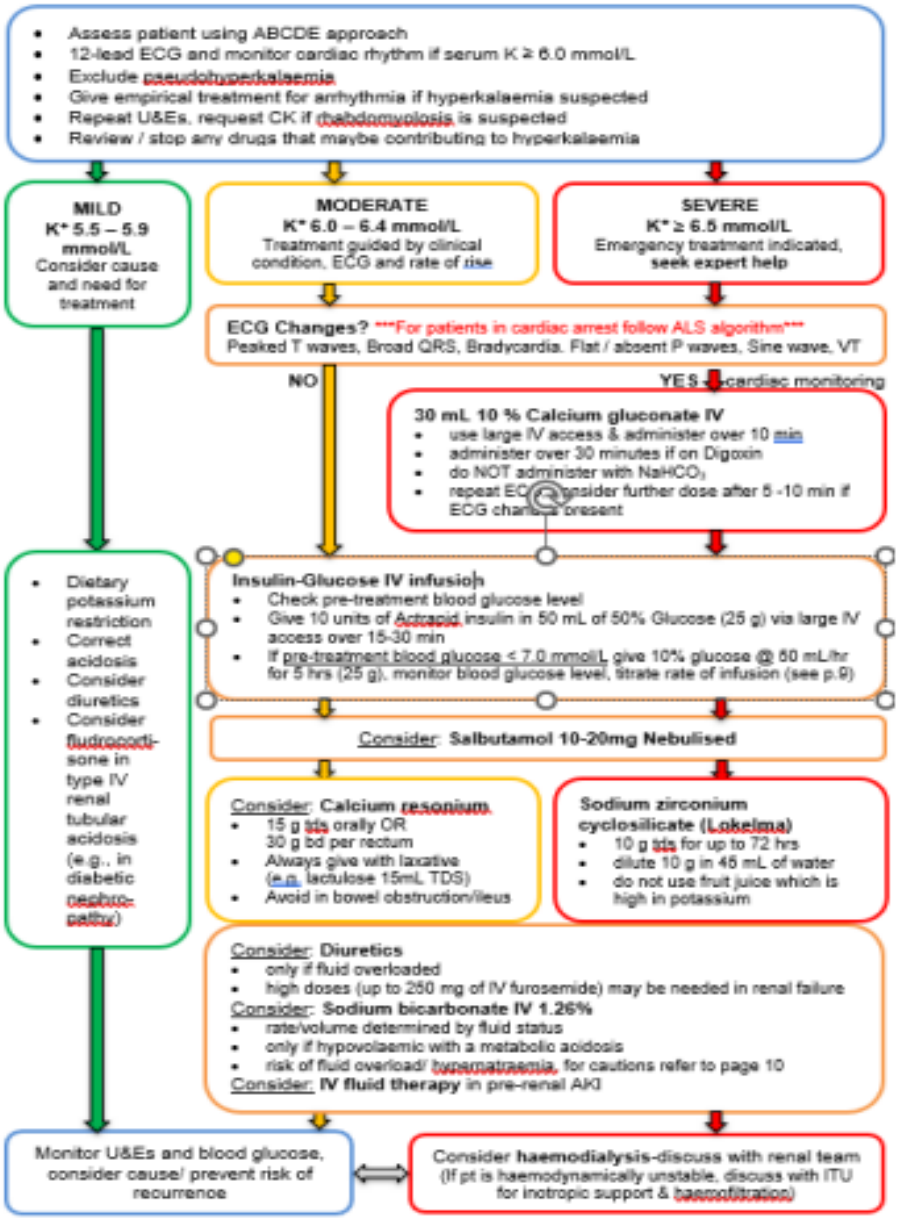
The recommended dose is 15 g orally three times a day; each dose should be given with 15 mL of lactulose to prevent constipation and to facilitate passage of the resin through the gut.

Calcium Resonium® may also be administered rectally in those unable to take or tolerate it orally. The recommended dose is 30 g as an enema retained for 9 hours and followed by irrigation.

- **Extracorporeal potassium wasting – dialysis**

All modes of renal replacement therapy are effective in removing potassium, with hemodialysis being the most rapid. Hemodialysis is indicated when hyperkalemia is refractory to medical management.

If hemodialysis is likely to be necessary, it is important to call the help of the renal team at an early stage.



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Intravascular Catheter Infection

Empirical use of Antimicrobials according to Infection

Intravascular Catheter Infection

Initial Treatment Strategies For Intravascular Catheter Infection

Standard Regimen		Choose one option from row A	Duration
Bacteremia (blood stream Infection)	A	Cefepime: IV: 2 gm/8 hr , Amoxacillin /clavulinic **+(-)** gentamycin: Oral : 625 mg / 8 hr or IV 1.2 gm /12 hr Gentamycin: 5-7 mg /kg one daily High dose extended interval dosing (one dailydose) Meropenem 1 gm/8 hr Imipenem/Cilastatin 500mg /6 hr **Choose one option from row A and one from row B(A+B)**	7-14 days For uncompliated s.aureus for >14 days from first negativeblood culture (treat uncompliated bacteremia 5-7days from day of first negative blood culture, plus antibiotic lock therapy for catheter slavage (not recommended for s.aureus)
Tunneled catheter Infection (HD catheter)	A	Vancomycin: IV: 15-20mg/kg/dose every8-12 hr initially	
	B	ceftazidime (1gm immediatly after HD) cefepime (1.5g- 2 g immediatly after HD) Meropenem (1 gm/24 hr)	

Notes: *linezolid not be used for empirical therapy

-For dialysis catheter we can use aminoglycoside for gram negative coveragemonitor drug level and watch for ototoxicity and nephrotoxicity

- For patient with persistent fungemia or bacteremia after catheter removal,give 4-6 weeks' antibiotic therapy.