Volume

1

VIETNAM PHYSICIAN EDUCATION PROGRAM

Participant Handbook

# General Surgery

VIETNAM PHYSICIAN EDUCATION PROGRAM

# General Surgery

Chapter

1

# The Basics of General Surgery

*This chapter will focus on clinical skills, basic pathology and an overview of surgical practice. These topics are of a broad nature and are intended to assist insetting the stage for this session. Included in this portion is a brief glossary as well.*

OBJECTIVES

Be able to perform a basic clinical examination

Understand the basics of surgical pathology.

Describe issues that arise in general surgical practice.

## Clinical Skills

All diagnoses are reached by taking a history, doing a clinical evaluation, and ordering special tests that might assist the doctor in making a diagnosis. In many situations, additional tests may not be needed (or available). This chapter will begin with taking an appropriate history.

When taking a patient HISTORY it helps to have in mind a particular classification of diseases:

*Congenital*

These diseases or abnormalities are present from birth. They are not necessarily genetic. Some of them may be inherited (genetic).

*Acquired*

- Trauma
- Infection or inflammation
- Neoplasia (benign or malignant)
- Collagen diseases
- Autoimmune
- Nutritional

- Blood and blood vessels
- Endocrine
- Degenerative
- Drugs

*Idiopathic*

Diseases often cannot be classified and are called *idiopathic*. Some psychiatric diseases fir under this category as well.

### Clinical Examination

Order of the examination is important, and should be carefully followed.

1. Observation
2. Auscultation
3. Palpation
4. Percussion

When reporting findings from examination, all descriptions should be as thorough and specific as possible.

| CATEGORY | SAMPLE DESCRIPTION |
|---|---|
| Site | "In the epidermis of the posterior aspect of the left thigh…" |
| Size | "…measures 3 X 2.5 cm…" |
| Surface | "…smooth…" |
| Shape | "…irregular borders…" |
| Special signs | "There is a fluctuance which is palpable…" |
| Nodes | "A firm 2 cm node is palpable in the left axilla…" |

Never forget to examine regional lymph nodes since they may be the only clinical indication of a disease process.

## Pathology

Once a provisional diagnosis (or differential diagnosis) has been made from the examination, it is necessary to have some knowledge of basic pathology in order to properly evaluate the condition. This section highlights some of the more common pathologies encountered in the practice of general surgery.

### Acute Inflammation: Local Effects

There are five local effects caused by increased blood flow, migration of white blood cells and exudates formation. The four principal effects of acute inflammation were described nearly 2,000 years ago by Celcus:

#### Redness (rubor)

An acutely inflamed tissue appears red, for example skin affected by sunburn, cellulitis due to bacterial infection or acute conjunctivitis. This is due to dilatation of small blood vessels within the damaged area.

#### Heat (calor)

Increase in temperature is seen only in peripheral parts of the body, such as the skin. It is due to increased blood flow (hyperemia) through the region, resulting in vascular dilatation and the delivery of warm blood to the area. Systemic fever, which results from some of the chemical mediators of inflammation, also contributes to the local temperature.

#### Swelling (tumor)

Swelling results from edema, the accumulation of fluid in the extra vascular space as part of the fluid exudate, and to a much lesser extent, from the physical mass of the inflammatory cells migrating into the area.

#### Pain (dolor)

For the patient, pain is one of the best known features of acute inflammation. It results partly from the stretching and distortion of tissues due to inflammatory edema and, in particular, from pus under pressure in an abscess cavity. Some of the chemical mediators of acute inflammation, including bradykinin, the prostaglandins and serotonin, are known to induce pain.

#### Loss of function

Loss of function, a well-known consequence of inflammation, was added by Virchow (1821-1902) to the list of features drawn up by Celsus. Movement of an inflamed area is consciously and reflexly inhibited by pain, while severe swelling may physically immobilize the tissues.

### Acute Inflammation: Systemic Effects

Clinical indications of an acute inflammatory process include:

General malaise

Fever

Pain, often localized to the inflamed area, e.g. the right iliac fossa in acute appendicitis

Rapid pulse rate

Laboratory investigations usually reveal:

A raised neutrophil count in the peripheral blood.

An increased erythrocyte sedimentation rate (ESR)

An increase in the concentration of acute-phase proteins in the blood. These are normally present in small concentrations, but this increases dramatically in response to acute inflammation. Produced by the liver, they are induced by circulating IL-1. Specific examples, the most common being C-reactive protein, may be measured in blood to monitor inflammatory processes.

Apart from the local features of acute and chronic inflammation described above, an inflammatory focus produces systemic effects.

### Pyrexia

Polymorphs and macrophages produce compounds known as endogenous pyrogens which act on the hypothalamus to set the thermoregulatory mechanisms at a higher temperature. Release of endogenous pyrogen is stimulated by phagocytosis, endotoxins and immune complexes.

### Constitutional symptoms

Constitutional symptoms include malaise, anorexia and nausea. Weight loss is common when there is extensive chronic inflammation. For this reason, tuberculosis used to be called 'consumption'.

### Reactive hyperplasia of the reticulo-endothelial system

Local or systemic lymph node enlargement commonly accompanies inflammation, while splenomegaly is found in certain specific infections (e.g. malaria, infectious mononucleosis).

### Hematological changes

Increased erythrocyte sedimentation rate. An increased erythrocyte sedimentation rate is a non-specific finding in many types of inflammation.

### Leukocytosis

Neutrophilia occurs in pyogenic infections and tissue destruction; eosinophilia in allergic disorders and parasitic infection; lymphocytosis in chronic infection (e .g. tuberculosis), many viral infections and in whooping cough; and monocytosis occurs in infectious mononucleosis and certain bacterial infections (e.g. tuberculosis, typhoid).

### Anemia

This may result from blood-loss in the inflammatory exudates (e.g. in ulcerative colitis), hemolysis (due to bacterial toxins), and 'the anemia of chronic disorders' due to toxic depression of the bone marrow.

The outcome of acute inflammation varies, and depends on the severity of the insult and the response of the host. Some possibilities include:

Complete resolution with no residual effects

Resolution with fibrosis, scarring

Suppuration, abscess formation

Gangrene, death of tissue in bulk

Death, due to spreading infection or toxicity to vital organs

## Wound Healing

There are two recognized processes:

| PRIMARY INTENTION | SECONDARY INTENTION |
|---|---|
| No loss of tissue and rapid epithelial cover | Loss of tissue with separated edges; granulation tissue appears first, followed by slow epithelial cover |
| Minimal scarring | Much scarring |

Wound healing can be delayed by sepsis, dead tissue, foreign material, dirt and poor blood supply.

## Hemorrhage

Hemorrhage may be arterial (pulsatile, bright red), venous (dark steady stream), or capillary (gentle ooze). It may be external or internal, and is classified in the following ways:

| PRIMARY | REACTIONARY | SECONDARY |
|---|---|---|
| Occurs at the time of injury or surgery | Occurs within 24 hours after primary hemorrhage and is caused by the rise in blood pressure following the hypotension produced by the original injury or anesthesia | Occurs 7-10 days after injury and is usually caused by infection |

## Some Helpful Definitions

Certain pathological conditions have precise definitions, which are often quoted or asked for. Common ones are listed below:

| | |
|---|---|
| Abscess | collection of pus in a cavity lines by granulation tissue |
| Adenocarcinoma | malignant tumor of glandular epithelium |
| Adenoma | benign tumor of glandular epithelium |
| Anemia | hemoglobin below normal for age and sex |
| Aneurysm | localized abnormal dilatation of an artery |
| Carcinoma | malignant tumor of epithelium |
| Cyst | abnormal cavity with well-defined lining |
| Diverticulum | a pouch arising from the wall of a viscus consisting of all layers (true diverticulum) or simply the mucosa (false diverticulum) |
| Embolism | transit within the bloodstream and impaction in some part of the vascular system of abnormal undissolved material |
| Empyema | collection of pus within a viscus or closed cavity |
| Fistula | abnormal communication or track between two epithelial surfaces |
| Gangrene | death of tissue in bulk |
| Goiter | enlargement of part of whole of the thyroid gland |
| Hamartoma | tumor containing a mixture of adult tissues normally present in the organ of origin |
| Hernia | abnormal protrusion of an organ through a defect in the wall of the cavity in which it lies |
| Infarction | an area of ischemic necrosis caused by blockage to the blood supply |
| Necrosis | cell death |
| Sarcoma | malignant tumor of mesodermal connective tissue or its derivatives |
| Sinus | abnormal blind track in tissues that communicate with an epithelial surface |
| Teratoma | tumor containing tissue derived from all three germinal embryonic layers occurring at a site where those three layers are not normally found |
| Thrombosis | formation of a solid mass from the constituents of blood within the bloodstream during life |

| | |
|---|---|
| Tumor | an abnormal mass of tissue, the growth of which exceeds and is uncoordinated with that of normal tissues; can be benign or malignant |
| Ulcer | a localized loss of continuity of an epithelial surface (e.g., skin) |

## Surgical Practice

Having reached a diagnosis using clinical skills and knowledge of pathology, it is necessary to consider whether surgery is indicated. Are the risks and complications acceptable? Does the patient consent to undergo the procedure (if elective)?

### Indications for Surgery

All operations are potentially dangerous and may have unwanted side effects or complications. Surgery, therefore, is indicated only when the potential risks are outweighed by the potential benefits. Indications for surgical treatment can be divided into:

| ABSOLUTE | RELATIVE |
|---|---|
| Situations or conditions in which surgery is the only available treatment option | Situations or conditions in which surgery has the potential to improve the quality of life or reduce the risk of death to a greater extent than any other treatment |

Surgery is also used extensively for diagnosis (biopsy or laparoscopy) before instituting suitable treatments. It can also be used as an adjunct to medical therapy where this has failed or complications have occurred.

### Fitness for Surgery

A patient must be physically capable of withstanding the trauma of any proposed surgical intervention and associated anesthetic. In this context, pre-existing medical illness is an important consideration. In America, the American Society of Anesthesiologists have established some basic criteria:

| CLASS | CONDITION OF PATIENT |
|---|---|
| Class 1 | Otherwise fit patient |
| Class 2 | Mild to moderate systemic disturbance (controlled diabetes or mild asthma) |
| Class 3 | Severe systemic disturbance (ischemic heart disease) |
| Class 4 | Severe life-threatening disorder (recent MI) |
| Class 5 | Moribund patient, unlikely to survive |

### Classification of Operation

From the patient's perspective, any operation is highly significant. However, operations are generally classified by surgeons an anesthesiologists according to a scale which relates to the risks involved and the degree of physiologic disturbance:

| | |
|---|---|
| Minor | skin lesions |
| Intermediate | hernia repair |
| Major | cholecystectomy |
| Complex major | colectomy and anastomosis |
| Complex major plus | heart surgery |

Operations can also be classified by their degree of urgency:

| | |
|---|---|
| Emergency | immediate operation; resuscitation simultaneous with surgery; usually within 1 hour (eg, severe trauma) |
| Urgent | delayed operation; as soon as possible after resuscitation; usually within 24 hours |
| Scheduled | operation at time to suit both surgeon and patient (routine) |

### Complications of Surgery

These can be classified as:

| | |
|---|---|
| Early | within 24 hours |
| Intermediate | up to 3 weeks post-operatively |
| Late | anytime thereafter |

Complications can be:

| | |
|---|---|
| Local | to do with the operation site itself |
| General | affecting other systems of the body |

*Example:*

| COMPLICATIONS OF AN APPENDECTOMY | | |
|---|---|---|
| | **LOCAL** | **GENERAL** |
| Early | Bleeding | Anesthetic: cardiac, urinary retention |
| Intermediate | Wound infection | Chest infection, DVT, PE |
| Late | Incisional hernia | Adhesions, scar, fistula |

Wound infection remains the most common post-operative complication.

Chapter

2

# Pre-Operative Preparation

*"You must remember not only to make surgery safe for the patient, but also to make the patient safe for surgery"*

## Tobacco Use

When discussing tobacco use, the specific referral is to cigarette smoking, however, the advice to give up tobacco pertains to those who indulge in pipe, cigar, or smokeless tobacco products as well. The use of tobacco has no beneficial effects on health. Its use has been associated with an increased risk of lung cancer, emphysema, head and neck cancer, esophageal cancer, stomach cancer, heart attack, stroke, high blood pressure, and hardening of the arteries. There is no disputing these facts. Nicotine, which is a major component of cigarette smoke, is an addictive drug; and breaking the nicotine habit is admittedly very difficult. It is not unusual, nor is it shameful to need medical or psychological assistance in quitting a tobacco habit.

Smoking cigarettes causes a change in the makeup of the cell layer that lines the trachea (windpipe) and major airways. This change eliminates the ciliated cells that remove mucus and dust from the lungs. As a consequence, mucus, dust, smoke particles, and bacteria that would otherwise be removed from the lungs remain within the airways. To clear this debris from the lungs, smokers develop a cough. Under anesthesia, this cough is suppressed, and the result is a plugging up of the airways with potentially infectious mucus. This results in a higher incidence of pneumonia and airway collapse (called atalectasis) in smokers. It also results in a violent awakening from anesthesia due to an intense stimulus to cough. Smokers have been known to rip all of their stitches out when they wake up from anesthesia due to the massive increase in abdominal and thoracic (chest) pressure generated by postoperative coughing fits. Fortunately, it takes only two smoke free weeks for most of this danger to be eliminated. If the patient cannot make the major step of quitting cigarettes completely, encourage them to take off two weeks before surgery.

## Alcohol Use

Behind smoking, alcohol consumption is the next most important cause of preventable postoperative complications. It is not necessary to be an alcoholic for alcohol consumption to present a problem in the postoperative state. Ethyl alcohol is another drug that causes a physical dependence, just like the nicotine in cigarettes. Anyone who drinks alcoholic beverages on a daily basis risks developing an alcohol withdrawal syndrome postoperatively. This means that even a single glass of wine with dinner or a glass of brandy before bed each night, puts the patient at risk of developing a potentially life-threatening complication. To prevent this possibility, advise patients to give up all forms of alcohol for two full weeks before any operation.

For most social drinkers, this poses no problem and carries virtually no risk. Heavy drinkers, who might have ever experienced the symptoms of anxiety, tremor, rapid heartbeat, or confusion when they tried to stop drinking, should not be advised to stop without medical assistance. First, it needs to be emphasized that people who are dependent on alcohol can die when they stop drinking. The brain can become dependent on ethyl alcohol. If this happens and a person just stops drinking alcohol, he/she will experience alcohol withdrawal, ultimately leading to seizures and death. Therefore, anyone scheduled for elective surgery that drinks alcohol on a daily basis should discuss this with his or her surgeon. Unrecognized alcohol withdrawal in the postoperative state carries with it a 50% chance of death, even in America's best intensive care units.

## Bowel Preparation

Those about to have surgery involving the abdomen or any portion of the digestive tract (including the esophagus, stomach, duodenum, liver, pancreas, intestines, rectum, and anus) should be given instructions on taking a bowel (colon) preparation. This preparation is undoubtedly one of the least favorite parts of the preoperative experience but also one of the most important. Bowels are full of a variety of ingested material, and as it makes its way from the stomach toward the anus, the character of this material changes from a watery, green liquid to nearly solid stool. As the digested food makes its way toward the anus, the amount of bacteria in this liquid material increases dramatically. The goal of a bowel preparation is twofold: one is to empty the digestive system of this ingested material, and the other is to decrease the amount of bacteria living within the bowel. This is important for a number of reasons. First, if the operation involves the removal of a segment of bowel, it is technically much easier to reconnect clean and empty intestine than it is to reconnect an intestine that is oozing contaminated material. Second, probably the greatest source of potentially infectious bacteria in the human body is the large bowel. Eliminating these bacteria before they are released from an opening in the bowel greatly decreases the chance of developing an infection postoperatively. This is especially true in operations in which prosthetic (man-made) material is going to be placed permanently in the body (as in large hernia

repairs and abdominal aortic aneurysm repairs). Third, if the goal is to locate a mass present within the bowel, it is much easier to feel it from the outside if no other material exists within the bowel to fool him or her. The bottom line is that a good bowel preparation makes most operations much easier. Conversely, a poorly prepped bowel can make the surgery more difficult and increase the risk for postoperative complications.

## Antibiotic Usage

It is important to know if the patient has used any type of antibiotic in the 6 months prior to surgery. Even if the antibiotics were prescribed to treat a urinary tract infection, bronchitis, or some other infection not related to the planned surgery, antibiotics could alter the composition of the bacteria that reside on and within the body.

## Aspirin and Other Non-Narcotic Pain Killers

These drugs, which are closely related to aspirin in that they control pain by reducing inflammation, are typically referred to as anti-inflammatories. They are used to treat a wide variety of disease processes and all have one common property, the inhibition of platelet function. Platelets are the cells in circulation that are responsible for the clotting of blood. Obviously if surgery is planned, blood should clot as well as possible; therefore, it is essential that patients be advised to avoid all anti-inflammatory drugs for a period of 1 to 2 weeks prior to surgery. Failure to stop taking these drugs may lead to excessive blood loss at surgery, which may necessitate the administration of blood or platelet transfusions. Even patients who are taking an aspirin a day to prevent heart attack or stroke should stop in the week prior to surgery.

## Coumadin

A common blood thinner in use today is Coumadin (warfarin). This is another drug whose use must be discontinued prior to surgery. If a patient is taking this drug, they should be advised to stop taking it a week before surgery. Both Coumadin and heparin act to inhibit blood clot formation independent of the function of platelets. They work by blocking the proteins in your blood that help hold the platelets in a blood clot together. There are many differences between the two drugs, but the important point is that Coumadin takes a long time to take effect, and once stopped, it takes days to weeks for its effect to be reversed. In contrast, heparin is effective immediately, and its effect is terminated within hours of its discontinuation. Therefore, sometimes it is necessary to allow the effect of Coumadin to wear off while preventing blood clot formation by the use of heparin. Just prior to the operation, the Heparin will be

stopped, your blood will clot normally during surgery, and once the operation is over, the heparin (and/or the Coumadin) can be restarted.

## Narcotic Pain Killers

The use of narcotics on a regular basis induces a tolerance to their effect. Higher doses of pain medication may be needed postoperatively to control the pain.

## Shaving the Operative Site

People frequently ask if they should shave the area, which is to be operated on before coming to the hospital. Shaving tends to release bacteria that normally reside in your hair follicles; this increases the incidence of wound infections postoperatively.

## The Night Before Surgery / Patient Instructions

Patients should be asked to eat or drink nothing after midnight the evening prior to surgery. This is done so that the stomach will be completely empty anesthesia is given. Rarely, vomiting complicates anesthesia and endotracheal intubation. While sedated, patients are unable to protect their airway and lungs, and run the risk of inhaling the stomach juices into the lungs (known as aspiration). If this happens, they run a high risk of developing pneumonia; and this particular type of pneumonia can be hard to treat. The simplest way to prevent it is to have an empty stomach prior to administering anesthesia. The only exception to this rule is that certain medications may be taken with a small sip of water the morning of surgery.

Diabetics run the same risk of aspiration as the rest of the population, and must follow the directive to have nothing to eat or drink after midnight on the evening prior to surgery. It is important, therefore, that the evening insulin dose the night before surgery and the morning dose the day of surgery be adjusted.

## Managing Patient Expectations

Before undergoing surgery, patients should have a very clear understanding of the goal of the operation. Beyond that, it is important to understand the risks and benefits associated with the operation.

Some helpful pointers:

- Discuss what the natural course of the disease if surgery is not performed.

- Discuss what will be accomplished during surgery.

Chapter

3

# Fluid and Electrolyte Balance

*This section is designed as both a tutorial and reference on the subject of fluids and electrolytes. In a healthy individual fluid volume and electrolyte concentrations are maintained within strict homeostatic limits through the interaction of several organ systems. Before considering pathology affecting this delicate balance, the basic physiology of fluid and electrolytes is reviewed here.*

OBJECTIVES

Discuss common fluid and electrolyte abnormalities in surgical patients.

Describe body fluid compartments, their content changes with aging and how they relate to clinical management.

Describe fluid management principles in surgical patients.

## The Basics

The maintenance of water and electrolyte balance requires that the quantities of these substances entering the body equal the quantities leaving it. Altering the water balance necessarily affects the electrolyte balance.

### Fluid compartments

The intracellular fluid compartment includes the fluids and electrolytes cell membranes enclose. The extracellular fluid compartment includes all fluids and electrolytes outside cell membranes. Interstitial fluid is outside of the blood vessels and within tissue spaces. Plasma is the "water" within blood. Lymph is found within lymphatic vessels. Transcellular fluid is located within body cavities.

**find drawing of this**

### Composition of body fluids

Extracellular fluids have high concentrations of sodium, chloride, and bicarbonate ions, with less potassium, calcium, magnesium, phosphate, and sulfate ions. Plasma contains more protein than do either interstitial fluid or lymph.

Intracellular fluid contains relatively high concentrations of potassium, phosphate, and magnesium ions; it also contains a greater concentration of sulfate ions and lesser concentrations of sodium, chloride, and bicarbonate ions than does extracellular fluid.

### Movements of fluid between compartments

Hydrostatic and osmotic pressure regulates fluid movements. Fluid leaves plasma because of hydrostatic pressure and returns to plasma because of osmotic pressure. Osmotic pressure drives fluid into lymph vessels. Osmotic pressure regulates fluid movement in and out of cells.

**drawing of osmotic pressure

Sodium ion concentrations are especially important in fluid movement regulation.

### Water Balance

Water Intake = Water Output

*Water Intake*

The volume of water taken in varies from person to person. Most water comes from consuming liquid or moist foods. Oxidative metabolism produces some water. The thirst mechanism is the primary regulator of water intake. Drinking and the resulting stomach distension inhibit the thirst mechanism.

*Water Output*

Water is lost in a variety of ways. It is excreted in the urine, feces, and sweat. Insensible loss occurs through evaporation from the skin and lungs. Urine production regulates water output.

*Regulation of water balance*

The distal convoluted tubules and collecting ducts of the nephrons regulate water balance. ADH from the hypothalamus and posterior pituitary gland stimulates water reabsorption in these segments. The mechanism involving ADH can reduce normal output of 1,500 milliliters to 500 milliliters per day. If excess water is taken in, the ADH mechanism is inhibited.

*Example 1:*

| WATER INTAKE | | WATER OUTPUT | |
|---|---|---|---|
| Drink | 1500 mL | Insensible perspiration | 500 mL |
| Food | 750 mL | Feces | 150 mL |
| Metabolic water | 250 mL | Sweat (variable) | none |
| | | Lungs | 350 mL |
| Total intake | 2500 mL | Total output | 1000 mL |
| INTAKE – OUTPUT = 1500 ML URINE | | | |

*Example 2:*

| WATER INTAKE | | WATER OUTPUT | |
|---|---|---|---|
| Drink | 1500 mL | Insensible perspiration | 500 mL |
| Food | 750 mL | Feces | 150 mL |
| Metabolic water | 250 mL | Sweat (variable) | 800 mL |
| | | Lungs | 350 mL |
| Total intake | 2500 mL | Total output | 1800 mL |
| INTAKE – OUTPUT = 700 ML URINE | | | |

Electrolyte Balance

**Electrolyte Intake = Electrolyte Output**

*Electrolyte intake*

The most important electrolytes in the body fluids are those that release ions of sodium, potassium, calcium, magnesium, chloride, sulfate, phosphate, and bicarbonate. These ions are obtained in foods and beverages or as by-products of metabolic processes.

*Regulation of electrolyte intake*

Electrolytes are usually obtained in sufficient quantities in response to hunger and thirst mechanisms. In a severe electrolyte deficiency, a person may experience a salt craving.

*Electrolyte output*

Electrolytes are lost through perspiration, feces, and urine. Quantities lost vary with temperature and physical exercise. The greatest electrolyte loss occurs as a result of kidney functions.

*Regulation of electrolyte balance*

Concentration of sodium, potassium, and calcium ions in the body fluids are particularly important. The regulation of sodium ions involves the secretion of aldosterone from the adrenal glands. The regulation of potassium ions also involves aldosterone. Calcitonin from the thyroid gland and parathyroid hormone from the parathyroid glands regulate calcium ion concentration.

The mechanisms that control positively charged ions secondarily regulate negatively charged ions. Chloride ions are passively reabsorbed in renal tubules as sodium ions are actively reabsorbed. Some negatively charged ions, such as phosphate ions, are reabsorbed partially by limited active transport mechanisms.

Acid-Base Balance

Acids are electrolytes that release hydrogen ions. Bases combine with hydrogen ions.

| SOURCES OF HYDROGEN IONS | |
|---|---|
| Aerobic | Aerobic respiration of glucose produces carbon dioxide, which reacts with water to form carbonic acid. Carbonic acid then dissociates to release hydrogen ions and bicarbonate ions |
| Anaerobic | Anaerobic respiration of glucose produces lactic acid |
| Other | Incomplete oxidation of fatty acids releases acidic ketone bodies |
| | Oxidation of sulfur-containing amino acids produces sulfuric acid |
| | Hydrolysis of phosphoproteins and nucleoproteins gives rise to phosphoric acid |

*Strengths of acids and bases*

Acids vary in the extent to which they ionize. Strong acids, such as hydrochloric acid, ionize more completely. Weak acids, such as carbonic acid, ionize less completely.

Bases vary in strength also. Strong bases, such as hydroxyl ions, combine readily with hydrogen ions. Weak bases, such as chloride ions, combine with hydrogen ions less readily.

*Acid-base buffer systems*

Buffer systems are composed of sets of two or more chemicals. They convert strong acids into weaker acids, or strong bases into weaker bases. They include the

bicarbonate buffer system, phosphate buffer system, and protein buffer system. Buffer systems also minimize pH changes.

### ACID/BASE BUFFER

H2CO3/NaHCO3 (Buffer Pair) 1:20 ratio results in pH 7.4

| STRONG BASE CONVERTED TO WEAK BASE: | STRONG ACID CONVERTED TO WEAK ACID: |
| --- | --- |
| H2CO3 + NaOH ---> NaHCO3 + H2O | NaHCO3 + HCl ---> H2CO3 + NaCl |

### RESPIRATORY CENTER

It helps regulate pH by controlling the rate and depth of breathing. Increasing carbon dioxide and hydrogen ion concentrations stimulate chemosensitive areas associated with the respiratory center, breathing rate and depth increase, and carbon dioxide concentration decreases. If the carbon dioxide and hydrogen ion concentrations are low, the respiratory center inhibits breathing.

### H2CO3/NAHCO3 BUFFER SYSTEM

Decrease in NaHCO3 causes metabolic acidosis; e.g. from ingestion of acids or retention of acids. Increase in H2CO3 causes respiratory acidosis; e.g. respiratory depression or emphysema

Increase in NaHCO3 causes metabolic alkalosis; e.g. loss of acid from the stomach Decrease in H2CO3 causes respiratory alkalosis; e.g. hyperventilation

### KIDNEYS

Nephrons secrete hydrogen ions to regulate pH. Phosphates buffer hydrogen ions in urine. Ammonia produced by renal cells helps transport hydrogen ions to the outside of the body. Chemical buffers act rapidly. Physiological buffers act more slowly.

## Intravascular Volume Assessment and Fluid Replacement: Pre- and Peri- Operative Periods

## Oxygen Transport

The primary objective of peri-operative fluid management is maintenance of adequate tissue perfusion. In a single term, oxygen delivery, symbolized by $DO_2$, combines cardiac index and arterial oxygen content, symbolized by $CaO_2$.

Normally, $DO_2$ is regulated through dilation and constriction of vascular beds in response to changes in regional and systemic oxygen consumption, symbolized by $VO_2$. In normal situations, $VO_2$ becomes $DO_2$-dependent when $DO_2$ is reduced to critical levels, <330 ml $O_2$/min/m2. This is when oxygen extraction reaches a maximum. Further reductions in $DO_2$ result in a reduction in $VO_2$.

In certain conditions such as ARDS, sepsis, and severe burns, $VO_2$ will be dependent on $DO_2$ at levels that are higher than the normal $DO_2$. In these high-risk patients, survival has been shown to be associated with supernormal $DO_2$, >600 ml $O_2$/min/m2. Studies suggest that treatment of the components of $DO_2$ may improve survival.

$$DO_2 = \text{Cardiac Index} \times CaO_2 \times 10 = SV \times HR \times [(Hb \times SaO_2 \times 1.34) + (0.003 \times PaO_2)]$$

## Clinical Signs of Adequacy of Perfusion:

- Urine output
- Mental status
- Capillary refill
- Skin color
- Temperature
- Pulse rate

- Frank Starling Curve
- Acid-base status
- Lactate Levels
- Oxygen consumption
- Mixed Venous / Oxygen Saturation

In approaching the surgical patient who exhibits signs of low perfusion, such as oliguria or hypotension, the most common etiology is insufficient intravascular volume.

## Quantitative Assessment

- The amount of fluid to be administered is best quantitated by continuous evaluation of the response to that which is infused.

- A good starting point is the use of formulas for depletion, blood loss, third spacing, and burns. These simple approximations of fluid requirements are sufficient in many patients. However, in patients who have severe chronic diseases or those at high risk of perioperative organ failure, more precise management of intravenous fluid therapy is necessary. In such situations, use of central venous and pulmonary artery pressure monitoring and the determination of cardiac performance with thermodilution or echocardiography may be indicated.

It is important to realize that even after minimal tissue manipulation there is still sequestration of fluid into the interstitial space. Surgery involving moderate trauma or hemorrhage requires more careful assessment of preoperative deficits and ongoing fluid requirements.

### Determining Preoperative Fluid Deficits

Increments of Weight in kg Maintenance fluid requirements per hour in ml
1-10 → 4
11-20 → 2
21+ → 1

### Usual Intraoperative Fluid Infusion Rates

Minimal Trauma 4 ml/kg/hr
Moderate Trauma 6 ml/kg/hr
Severe Trauma 8 ml/kg/hr

> **Example:** 20 kg child, NPO for 6 hours, undergoing one hour hernia repair.
>
> Deficit = 40 cc/hr for 1st 10 kg BW + 20 cc/hr for 2nd 10 kg BW = 60 cc/hr
>
> 6 hours of NPO X 60 cc/hr = 360 preop deficit
>
> Intraop losses = 6 cc/kg/hr X 20 kg X 1 hr = 180 cc plus blood loss

- An estimate of the expected effects of fluid infusion on plasma volume (PV) requires knowledge of the sodium and colloid concentrations of these solutions and how they will distribute in the body.

PV = volume infused x (PV/Vd)

where Vd = distribution volume.

For example, assume that acute blood loss of 500 cc is to be replaced with D5W, which contains no sodium. After cellular uptake of glucose, the remaining water would distribute throughout total body water (TBW).

500 = volume of D5W infused x 3/42 volume infused to replace 500 cc blood loss = 7 liter

Another example is using colloids, assuming membrane permeability is normal. Each gram of albumin holds about 14-15 cc of water in the PV.

Each gram of starch holds about 16-17 cc of water in the PV

Therefore, 500 cc of 5% albumin, containing 25 gm of albumin, would expand the PV by 375 cc, three quarters of the infused volume.

- Conclusion:

  - Trends are more important than absolute values.
  - Titrate fluid challenge with adequate clinical assessment.

Qualitative Assessment

The choice of fluid to be used to correct hypoperfusion is of much less consequence than adequate amounts. The primary defect in hemorrhage and trauma is a decreased intravascular volume. The choice of which fluid to use is based on the clinical situation.

COLLOID

5% Albumin (Na 145 meq/L)
25% Albumin (Na 145 meq/L)
Plasma Protein Fraction (Plasmanate\xac )
6% Hetastarch (Na 154 meq/L) (hydroxyethyl starch, Hespan\xac )
Dextran 40 (in either normal saline or D5W)

ISOTONIC CRYSTALLOID

Normal Saline (0.9%) (Na 154 meq/L, Cl 154 meq/L)

Lactate Ringer's (Hartman's solution) (Na 130 meq/L, Cl 109 meq/L, K 4 meq/L, Lactate 28 meq/L, Ca 3 meq/L)
Normosol-R (Na 140 meq/L, Cl 90 meq/L, K 5 meq/L, Mg 3 meq/L)

Choices within crystalloid group should be based on clinical situation as to need for intravascular volume and electrolyte imbalance.

### HYPERTONIC SALINE AND HYPERTONIC SALINE WITH DEXTRAN (HSD)

Solutions containing sodium in concentrations far exceeding those in serum have been extensively investigated in humans. Hypernatremic fluids increase PV both by osmotic attraction of water from the intracellular and extracellular spaces and by transient translocation of interstitial fluid into the PV. At equilibrium, after infusion of 7.5% saline to treat hypovolemic shock, PV should be expanded by a volume equal to the infused volume. Addition of hyperoncotic colloid to the hypernatremic solution can increase the PV increment further, up to 7 times the infused volume.

### COLLOID VS. CRYSTALLOID DEBATE

Replacement of blood loss with crystalloid fluids at a rate of 3-4 cc crystalloid is sufficient if blood loss is moderate (<7 ml/kg), in part because interstitial fluid is rapidly transferred into the intravascular volume in response to decreases in capillary hydrostatic pressure. However, as large blood losses are replaced with crystalloid solutions, the ratio of extravascular to intravascular volume progressively increases, so that a larger amount of crystalloid is needed to restore intravascular blood volume. In such situations, rations of 8:1 cc crystalloid to cc blood loss may be needed.

No data has established the risk of worsening lung injury with colloid in ARDS. Colloid infusion may actually improve oxygen delivery without impairing $PaO_2$ in septic patients.

In conclusion, when debating the use of colloid and crystalloid, it is imperative to think about all of the components of the Starling equation:

$$Q = k[(Pcapillary-Pinterstitial)-s(capillary-interstitial)]$$

where: Q = fluid movement out of the intravascular space
k = permeability coefficient
P = hydrostatic pressures
s = oncotic coefficient

## Blood and Blood products

Surgical teams, usually the anesthesiologist, administer over 1/2 of the blood given to patients in the United States. The following is a list of blood and blood products available in the United States:

- Whole blood
- Packed red blood cells
- Washed red blood cells
- Leukocyte depleted red cells
- Plasma, single donor, fresh-frozen
- Cryoprecipitate
- Platelet concentrate

## Storage of Blood

**get stats on this**

## Ordering Blood Products

- Never put blood into an unlabeled sample tube.
- Sample should be typed, screened for common antibodies (if available)
- Crossmatch
- 1:10,000 chance of an Ab present in recipient that is potentially dangerous

**what are VN stats**

- Emergency request for uncrossmatched blood

If an acceptable sample is available, ABO identical units should be available. 1:1000 patients with an unexpected Ab. Otherwise, Group O, Rh Neg packed red cells should be available, or for women of child bearing age. Otherwise Group O, Rh Positive should be used. It is the responsibility of the individual transfusing the blood to make absolutely certain the recipient's identification matches the identification on the unit of blood. If there is any doubt, transfusion should be delayed or, if absolutely necessary, only group o packed cells should be transfused. Severe hemolytic transfusion reactions are almost always caused by clerical or identification error leading to an ABO incompatible transfusion

## Complications of Blood Transfusion

1. $O_2$ Transport: Shift to left in $O_2$-Hb dissociation curve so RBC's have increased affinity for oxygen and there is less available to tissues. Warm

blood and avoid other things that shift O2-Hb dissociation curve to the left such as alkalosis (bicarb) and hypothermia.

2. Transfusions Reactions

3. Citrate Intoxication and Hyperkalemia

4. Hypothermia

5. Acid-Base Disturbances

6. Microaggregates

7. Infectivity-Hepatitis, HIV, CMV, Syphilis

8. Dilutional Coagulopathy

### Alternatives to Transfusion

- Minimize blood loss - controlled hypotension
- Tolerate a lower HCT

$$EBL = BV \times \frac{Hi - He}{Havg} = 70 \times 60\ cc/kg \times \frac{(40-20)}{30} = 2520\ \text{blood loss}$$

where: EBL = estimated blood loss
BV = blood volume
Hi = initial hematocrit
He = final hematocrit

- Transfuse Autologous Blood

1. Preoperative donation and storage - 72 hours to normalize plasma proteins.

2. Acute preoperative phlebotomy and hemodilution - inexpensive compared to preop donation. Progressively decreases the Hct of units saved.

3. Perioperative blood salvage from the surgical site

#### STUDY QUESTIONS

What is the difference between a Type and Screen and a Crossmatch?
What are maximum surgical blood ordering schedules?
What tests are performed on donor blood?
Which IV Solutions are compatible with red cells?
What filters are sued to administer blood products?
When should blood warmers be used?
What are the advantages and disadvantages of designated donations?
When is salvage and reinfusion of shed blood employed?
What is acute normovolemic hemodilution?

## Electrolyte Replacement

### Potassium

Potassium balance depends on the interaction of internal and external homeostatic mechanisms. Only when one or both systems are disturbed acutely or impaired chronically does plasma K+ change markedly.

### Internal Balance

#### ACID-BASE

With increasing extracellular H+ concentration (acidosis), K+ moves from the intracellular to the extracellular compartment in exchange for H+. The increase in plasma K+ concentration is small at first, but increases for a time, as the acidosis continues. However, K+ is lost in the urine, and one sees a lessening of the effect of acidosis on serum K+. The K+ changes seen with metabolic alkalosis are not well understood and are complicated by the kaliuresis that occurs. Some intracellular shift of K+ does occur, but the decrease in serum K+ is mainly due to renal loss.

#### INSULIN

Insulin stimulates K+ uptake by muscle and hepatic cells.

#### MINERALCORTICOIDS

Aldosterone makes cells more receptive to the uptake of K+ and increases renal excretion of K+.

CATECHOLAMINES

Epinephrine initially increases plasma K+ because of combined alpha and beta receptor stimulation, which releases K+ from the liver. The response is followed by a decrease in plasma K+ caused by beta-receptor stimulation, which enhances K+ uptake by muscle and liver. The end result is a decrease in serum K+.Propranolol impairs cellular uptake of K+.

### External Balance - Renal Potassium Excretion

Potassium Intake - An acute or chronic increase in K+ intake leads to increased secretion in the distal convoluted tubule.

Sodium Intake and Distal Tubular Flow Rate - A sodium load will increase flow past the distal tubule and cause K+ wasting. The converse is true too.

Mineralcorticoids - A mineralcorticoid deficiency leads to K+ retention and Na+ wasting, just as excess leads to opposite changes.

### External Balance - GI Potassium Excretion

Fecal excretion of K+ normally is small, but with diarrhea disorders, K+ loss increases significantly.

### Potassium disorders

#### Hypokalemia

The serum potassium is only a fair reflection of total body potassium.

WORK UP

- Urinary K+ and Cl
- Arterial pH and HCO3
- History and PE
- Current medications

TREATMENT

The treatment of hypokalemia includes repletion of K+ and removal of the cause of hypokalemia. In an emergency situation, in the presence of arrhythmias, K+ can be replaced intravenously by a solution containing 40 to 60 meq/l, infused at a

rate of no more than 40 meq/hour. Any magnesium deficiency must be corrected in order to correct the hypokalemia.

### Hyperkalemia

Potassium is released from cells at times of stress, injury, acidosis; but the kidney is able to regulate potassium well, and hyperkalemia is rarely a problem. However, in the presence of renal failure hyperkalemia becomes a common problem.

It is generally treated if there is an abrupt rise from normal to >6.5 meq/liter or if any level is associated with EKG changes. Clinical features involve neuromuscular abnormalities, with weakness, paresthesias, paralysis, as well as GI complaints of nausea, vomiting, colic, and diarrhea.

Cardiac abnormalities are the most feared sequelae of hyperkalemia and include conduction defects as well as dysrhythmias. Hyponatremia and acidosis potentiate the adverse effects of hyperkalemia on the heart.

SOME SIGNS

- Peaked T waves
- Flattening of P waves
- Prolonged PR interval
- Widening of the QRS
- Sine Wave pattern
- V Fib/cardiac arrest

TREATMENT

1. Restrict Exogenous K+
2. Calcium gluconate - 10 to 30 ml of 10% solution over 3 to 5 minutes
3. NaHCO3 - 50 to 100 ml of 7.5% solution
4. Hyperventilation will also create an alkalosis and drive K+ into cells
5. Avoid hypoventilation
6. Glucose - insulin - 500 ml of 10% dextrose plus 10 units regular insulin or 50 - 100 gm with 10 -20 units regular insulin

7. Lasix, ethacrynic acid, or bumex
8. Oral or rectal sodium or calcium polystyrene with sorbitol
9. Peritoneal dialysis or hemodialysis
10. Transvenous pacemaker

## Sodium

### Sodium Physiology

Sodium and its anions make up about 90% of the total extracellular osmotically active solute.

Serum osmolality (mOsm/kg H20) = 2 X [Na+] + [glucose]/18 + [BUN]/2.8

For practical purposes, twice the Na+ concentration equals serum osmolality because urea and glucose ordinarily are responsible for less than 5% of the osmotic pressure.

### Disorders of Sodium and Water metabolism - Hyponatremia

*"Definition of the Cause Defines the Treatment"*

DETERMINE SERUM OSMOLALITY TO DETERMINE IF IT IS ISOTONIC, HYPERTONIC, OR HYPOTONIC HYPONATREMIA

Isotonic hyponatremia occurs when plasma solids dilute the Na+. This occurs with hyperproteinemia and hyperlipidemia.

Hypertonic hyponatremia occurs with uncontrolled diabetes and with the use of mannitol. Treat by correcting the fluid deficit initially with isotonic saline, then give insulin to decrease glucose and hypotonic saline to correct free water deficit.

True hypotonic hyponatremia is characterized by hypovolemic, hypervolemic, and isovolemic. Differentiation is done by assessing ECF volume: blood pressure, skin turgor, edema, ascites etc.

TREATMENT

You are worried about the patient's CNS state with neuromuscular disorders, convulsions, and even death. How rapidly one corrects the hyponatremia is unclear. Reports describe death or severe neurologic sequelae with both rapid and slow correction of hyponatremia. When severe hyponatremia exists with CNS symptoms, it is recommended to correct at a rate of 2 meq/liter/hour with hypertonic saline and a diuretic. But the concern over central pontine myelinolysis has been linked to the rapid correction of hyponatremia. Which is worse, the treatment or the disease? Don't use hypertonic solutions. Asymptomatic patients are treated more conservatively with water restriction, salt intake, and intravenous normal saline.

### Hypernatremia

Less common than hyponatremia, usually iatrogenic. Hypernatremia occurs with either pure water loss, hypotonic fluid loss, or salt gain. Most commonly we see patients with both water and sodium loss, but water loss exceeds salt loss. Water loss is usually from increased insensible losses, fever, burns, or diabetes insipidus.

#### DIABETES INSIPIDUS

Symptoms include polyuria and polydipsia due to deficient production of vasopressin or ADH by the posterior lobe of the pituitary gland, called pituitary DI or central DI. Etiologies include:

- head trauma
- cranial surgery - specifically post-pituitary surgery
- infectious - meningitis, encephalitis,
- granulomatous - sarcoid, TB, histoplasmosis
- vascular - aneurysms, thrombosis

Treatment is Desmopressin (dDAVP) 0.1 - 0.4 cc intranasally or intrabucally every 8 to 12 hours. Remember polyuria without Hypernatremia is not DI.

#### NEPHROGENIC DIABETES

Characterized by renal tubular unresponsiveness to endogenous ADH. Etiology is primarily kidney disease and lithium toxicity. Primary method of treatment involves removal of the offending drug. Thiazide diuretics paradoxically decrease urine volumes.

### Treatment of hypernatremia

Hypotonic fluid loss is the most common form of hypernatremia. It is caused by gastroenteritis, osmotic diuresis. Signs of intravascular depletion are evident. Treatment involves replacement volume with normal saline, followed by correction of the free water deficit.

Chapter

4

# Shock

*Shock is a reduction in blood flow by diminished cardiac output or maldistributed output such that potential irreversible tissue damage occurs. The body's attempt to restore circulating blood volume with adequate delivery of oxygen to tissues is what causes the symptoms of shock.*

OBJECTIVES

Describe the three basic kinds of shock.

Be able to propose immediate therapy and maintenance for each type.

Understand common mistakes that can be made in the diagnosis and treatment of shock.

## Systemic Pathophysiology of Shock

One of the initial compensatory mechanisms of the body is to release endogenous substances in attempt to counteract the insult. Catecholamine release causes increased:

- Heart rate, cardiac output and contractility
- Preload, venous and arteriolar tone

Additional neuroendocrine response is mediated by the hypothalamic secretion of glucocorticoid, GH, and aldosterone which have varied end-organ effects. Renal interaction with ADH, renin and angiotensin can help quickly regulate and mobilize fluids as well.

Microvascular sphincters to non-essential areas such as the skin, fat and muscles constrict in attempt to shunt necessary fluids to vital organs. Kidneys, splanchnic organs and intestines are amongst the last organs to be deprived and disruption of their function can be a late sign of shock.

## Pathophysiology on a Cellular Level

Failure of the compensatory response can lead to pooling of blood, which results in blood flow stagnation, sludging, and roleux formation. Fluid shifts into and out the cell cause swelling and mitochondrial disruption, leading to the inevitable cell death. Changes in osmotic pressure of the cell and the extracellular spaces cause the movement of interstitial fluid from the lymphatic system into the bloodstream. Much of the cellular waste and debris is circulating in the lymphatic system, which means that additional unsavory substances are pouring into the bloodstream. If shock remains uncompensated, intracellular fluids and ions begin to leak into the blood stream, potentially introducing a number of highly reactive substances into an already overstressed system. Adding further insult, the increase in metabolic demand that the shock causes, cells are depleted of their immediate energy sources and are forced to shift from aerobic to anaerobic metabolism.

| NET RESULTS OF UNCOMPENSATED SHOCK |
| --- |
| Systemic lactic acidosis |
| Decreased myocardial contractility |
| Decreased vascular tone |
| Decreased blood pressure, preload and cardiac output |

## Clinical Presentation of Generalized Shock

### History

Several elements of a patient's social and medical history may help identify risk factors for shock:

- Cardiac disease
- Illness
- Injury
- Use of drugs or alcohol

### Factors Influencing Shock

Other factors may help predict clinical course, outcome, or response to therapy:

- Age

Sex

Nutritional status

Health of patient

Air temperature, Humidity, Altitude

Fear, Anxiety, Pain

Magnitude of insult

Evaluation of the Shock Patient

VITAL SIGNS

Initial recording of vital signs will generally support the following findings:

- Blood pressure: usually low or low normal (< 90 systolic, 50-60 diastolic), occasionally normal
- Pulse: fast, weak, thready, narrow pulse pressure (60-120 bpm)
- Respirations: rapid (air hunger), especially with major hemorrhage (12-24 per minute)

It is important to record the initial vital signs, however, it is crucial to re-take them at regular intervals. There is significant variance in the population and absolute values of vital signs may not be as significant as trends.

PHYSICAL EXAMINATION

- Skin: Cool, pale, moist (hot and dry in hyperdynamic shock)
- Veins: full, flat or collapsed (variable with type of shock)
- Mental status: anxious, dull, obtunded or comatose
- Urine output: low to none

LABORATORY TESTS TO OBTAIN

Again, absolute values are not as important as trends. Carefully record them at regular intervals. Some of these tests may not be available.

- CBC
- Electrolyte panel (including glucose)
- Electrocardiogram

- Chest X-ray
- Arterial blood gases
- Type and cross match 2-5 units of blood
- Optional: cardiac enzymes, pulmonary artery catheter

**WARNINGS**

Most studies are done on healthy adult males (70 kg) whose blood volume is assumed to be 70 cc / kg or 4900 cc. Few patients in practice qualify for this. In females, older patients and the very obese blood volume is likely to be 60 cc / kg or less.

Example 1

50 kg elderly female might have blood volume of only 3000 cc.

1000 cc blood loss = 33% of effective blood volume

1000 cc blood loss in 70 kg male is 20% of effective blood volume

Example 2

90 kg male might have a blood volume of 7000 cc.

1000 cc blood loss = 14% and of minimal concern (if bleeding has stopped).

***Remember:***

- Use of colloid: there is no proof that it is of any value in treating shock.
- Healthy adults over age 65 have impaired systemic function, regardless of physical function
- Try to keep patients as warm and dry as possible
- Use warm fluids during resuscitation if they are available
- Use a Foley catheter for monitoring
  Urinary output must be maintained approximately on 50 ml/h in the adult patient, while 1 ml/kg/h is adequate in the pediatric patient. For infants under one year of age 2 ml/kg/h should be maintained.

# Types of Shock

## Cardiogenic shock

| CARDIOGENIC SHOCK | |
|---|---|
| **BLOOD PUMP PROBLEM** | |
| Intrinsic cardiac problems | Acute myocardial infarction, myocarditis, cardiomyopathy, myocardial contusion |
| Inflow difficulty | Pericardial tamponade, tension pneumothorax, mitral or tricuspid stenosis |
| Outflow trouble | Pulmonary embolism, aortic and pulmonic stenosis, mitral insufficiency (esp. in the setting of AMI with rupture of papillary muscle), VSD, air embolism |

### Clinical Presentation of Cardiogenic Shock

It is important to remember that the same mechanisms of compensation are at work with cardiogenic shock, however the clinical manifestations may be different because patients with cardiac histories may be taking medications that alter vital signs. For example, tachycardia can be masked by beta-blocker therapy. A key piece of information from the patient history is use of medications and cardiovascular history.

Other findings that may be present include:

- Abnormal ECG
    - Low voltage and / or electrical alternans can be seen in pericardial tamponade
    - ST segment elevation or pathologic Q waves can be seen in acute myocardial infarction (AMI)
    - New bundle branch blocks
- Decreased blood pressure
    - This varies, however it is important to keep in mind that a minimum mean arterial pressure (MAP) of 70 mm Hg is required to maintain coronary perfusion.
- Chest findings
    - Pathologic S3 (ventricular gallop)

- Pathologic S4 (atrial gallop)
- Muffled heart tones (tamponade)
- Decreased or absent breath sounds (tension pneumothorax)
- New murmur

| COMMON HEART MURMUR TABLE | |
|---|---|
| SYSTOLIC MURMURS | DIASTOLIC MURMURS |
| Mitral regurgitation | Aortic regurgitation |
| Aortic stenosis | Mitral stenosis |

- Neck abnormalities
  - Distended neck veins (tamponade)
  - Tracheal deviation (pneumothorax = PTX)

Management of Cardiogenic Shock

**AIRWAY – VENTILATION – OXYGEN**

REVERSIBLE CAUSES

Once the diagnosis has been made, all efforts should be made to treat the reversible causes. Of course, the two most critical are tension PTX and tamponade. Treatment for a tension PTX should be managed emergently using a large bore needle or a chest tube, while tamponade is most effectively treated by performing pericardiocentesis.

OPTIMIZATION OF PUMP FUNCTION

Aggressive airway management is the key delivery of oxygen to the already ischemic system. This may involve insertion of an endotracheal tube or may be as simple as a facemask with constant oxygen. Invasive monitoring can be helpful if it available. Fluid management should be undertaken judiciously, since it is likely that additional quantities of fluid would only add further pump stress. Pressors can be given, and there has been much discussion as to which ones to use. In general, dobutamine should be used if the patient is tachycardic with an increased SVR. Dopamine is the preferred pressor in the patient with less tachycardia and a low SVR. Morphine should be given as needed for anxiolysis and pain. Some experts advocate the use of a short-acting beta-blocker (esmolol) for refractory tachycardia.

Some centers offer angioplasty and thrombolytic therapy, although this type of therapy should only be undertaken by a trained interventional cardiologist.

## Vasogenic Shock

| VASOGENIC SHOCK |
| --- |
| **BLOOD VESSEL PROBLEM** |
| Sepsis |
| Anaphylaxis |
| Neurologic injury |
| Pharmacologic injury |

### Specific Pathologies for Vasogenic Shock

Sepsis

Definition: perfusion embarrassment secondary to dilated vascular bed in response to bacteria and their products circulating in the blood

| Risk factors | Mechanism | Notes |
| --- | --- | --- |
| Immunocompromised host, GU / respiratory tract manipulation, asplenia, chronic disease, major burns, hospitalization, abdominal surgery | Endotoxin-mediated effects such as fever, hypotension, increased capillary permeability, DIC, complement activation, leukopenia, leukocytosis | Can have cardiac effects secondary to depressed myocardial contractility |

Anaphylaxis

Definition: hypotension that develops as a part of the immune system response to an antigen (IgE mediated)

| Risk factors | Mechanism | Notes |
|---|---|---|
| Patients will sometimes have other allergies.<br><br>Most will know what their specific allergy is and be aware of treatment. | Antigen exposure → body stimulated to produce IgE antibodies to specific antigen→ antigen binds to mast cells and basophils → anaphylactic response | Increased mucus production, bronchoconstriction |

Neurologic Insult

Definition: hypotension as the result of the loss of sympathetic vascular tone below the level of spinal injury, or generalized (in the case of head trauma)

| Risk factors | Mechanism | Notes |
|---|---|---|
| Spinal or head trauma, certain types of myelomas, tumors affecting the spinal cord, vascular insults along spinal territories | Unknown | May lose sympathetic tone to the heart.<br><br>Usually transient, lasting 3 – 7 days.<br><br>May occur in response to particular general anesthetics. |

Pharmacologic Insult

Definition: hypotension resulting from medication side effects or pharmacologic effects in overdose

| Risk factors | Mechanism | Notes |
|---|---|---|
| Any drug decreasing blood pressure is a possible etiologic agent. Some common examples include:<br><br>Opiates, Sedative-hypnotics, Anticholinergics, Antidepressants, Nitrates, | Overexposure to antihypertension-inducing medication or pharmacologically active agent. | Most likely in drug overdose patients and the elderly population. |

## Specific Clinical Presentations of Vasogenic Shock

### Sepsis

Patients are considered septic when they are shown to be febrile, with or without evidence of bacteremia, fungemia, or viremia and with labile blood pressure and an altered mental status. Typically, septic shock is not surprising in light of the clinical course of patients, however there are many manifestations of septic shock which are considered to be atypical but not uncommon:

- Mental status changes alone
- Fever alone
- Respiratory alkalosis
- Metabolic acidosis
- Unexplained hypotension

### Anaphylaxis

This type of shock is characterized by an almost immediate response to the inciting agent. There can sometimes be cutaneous manifestations such as urticaria, erythema, pruritis or angioedema as well. Respiratory compromise is the first symptom, and it begins as stridor and develops into complete respiratory arrest if not managed appropriately. Circulatory collapse can ensue at any stage, and is initially recognized as tachycardia. This should not be mistaken for 'anxiety' because it will progress to vasodilation and severe hypotension.

### Neurogenic

As previously mentioned, this is generally a diagnosis of exclusion. The degree of hypotension is usually only moderate and sometimes even labile. Relative bradycardia will endue, however it is important to classify injuries appropriately since sympathetic denervation to the heart can have a permanent effect on cardiac rate. Flaccid paralysis may be present below the level of injury but may initially be difficult to assess based on the mechanism of injury and the presence of distracting injuries.

### Phamacologic Insult

Findings will be specific to drug toxidromes, and below are a few examples:

Narcotic toxidrome →
Anticholinergic toxidrome →
Antidepressant toxidrome →
Digitalis toxidrome →

Management of Vasogenic Shock

**AIRWAY – VENTILATION – OXYGEN**

### SEPTIC SHOCK

Parenteral antibiotics, antifungals, or antivirals appropriate for presumed source of sepsis. Remember that it is important to draw blood cultures *before* administering these medications as to draw a pure sample. Once the inciting organism is identified, definitive treatment should be undertaken (i.e., drainage of abscess). Continue to support A-B-Cs.

### ANAPHYLACTIC SHOCK

Recognize clinical entity early! Airway support is the initial concern, and a surgical airway may be necessary. Epinephrine should be given subcutaneously immediately upon recognizing the clinical symptoms of anaphylaxis, and some experts recommend giving intravenous antihistamines (such as diphenhydramine) as well. Corticosteroids may be needed to prevent rebound or delayed reactions. Some patients have significant experience with treating their allergies and their histories may be helpful in planning therapy. Intravenous fluids should be given judiciously since pulmonary edema can develop secondary to leaky capillaries as part of the immune response. If severe and late stage, pressors can be given to maintain critical organ perfusion during resuscitation.

### NEUROGENIC SHOCK

Shock in the trauma patient is *always* hypovolemia / hemorrhage until proven otherwise. Once the patient has been evaluated for head injury, the position of the bed should be adjusted to trendelenberg. Crystalloid fluid boluses can be helpful although in many cases, the patient will not respond as expected. If perfusion remains inadequate after fluid infusion, alpha agonist therapy is recommended as follows:

Dopamine at alpha doses → > 10 mcg / kg / min
Ephedrine → 12.5-25 mg IV q 4 h

Bradycardia can be treated with Atropine 0.5 – 1.0 mg (to max 3mg), but keep in mind that these patients may need temporary pacing.

## PHARMACOLOGIC SHOCK

Specific reversal agents are available for many forms of pharmacologic insult. It is important to determine from the patient history which substance they may have ingested, what their medication regimen consists of, and what / when was their last meal. Drug removal can be attempted with gastric lavage, charcoal adsorption, and enhanced elimination. Once the insult has been identified and appropriately eliminated, supportive care is usually all that is needed.

### Hypovolemic shock

| HYPOVOLEMIC SHOCK |
| --- |
| **BLOOD VOLUME PROBLEM** |
| Dehydration |
| Trauma |
| Visible, non-traumatic blood loss |
| Occult blood loss |

#### Specific Pathologies of Hypovolemic Shock

Dehydration

Intravascular fluid loss can be caused by an increase in fluid losses (such as vomiting or diarrhea) or a decrease in fluid intake. In either case, the result is a severe decrease in volume and a fluid shift that causes electrolytes imbalances as well.

Hemorrhage

Trauma is the most dramatic form of hemorrhage, although it is important to remember that there are several other ways to loose large amounts of blood very rapidly:

- External hemorrhage from a post-operative wound

- Vaginal bleeding
- GI bleeding
- Hemoptysis
- GU bleeding
- Aneurysms
- Ectopic pregnancy
- Hemorrhagic cysts
- Retroperitoneal bleeding

### Clinical Presentation of Hypovolemic Shock

| |
|---|
| Tachycardia and tachypnea |
| Hypotension and decreased pulse pressure |
| Skin changes |
| Decreased mental status |
| Decreased urine output |

The American College of Surgeons proposes the following categorization scheme for hemorrhagic shock:

| CLASS | PERCENT BLOOD VOLUME LOSS | SYMPTOMS |
|---|---|---|
| **I** | 15 % | This is a minimal hemorrhage; uncomplicated generally no changes occur in heart rate, blood pressure or respiratory rate. |
| **II** | 15-30 % | Tachycardia, tachypnea, anxiety, decreased pulse pressure are present. Because the systolic pressure changes minimally in the early phases of shock, pulse pressure evaluation becomes important . Urinary output is |

| | | |
|---|---|---|
| | | only minimally affected. |
| **III** | 30 – 40 % | Classic signs of shock are present with changes in mental status and significant fall in systolic blood pressure. Transfusion is indicated. |
| **IV** | > 40 % | No urinary output is detected. Deep depression of the mental state. Immediate surgical intervention is necessary. |

Another simple scheme that attempts to quantify hemorrhagic shock is the Alghevar Scheme. This considers the systolic blood pressure to heart rate ratio. If this ratio is more than 1, the patient is not yet in manifest hypovolemic shock. If the ratio is less than 1, the patient is in a clear shock condition. Care must be taken not to underestimate the degree of shock.

Alghevar Scheme: Systolic blood pressure / Heart rate

Management of Hypovolemic Shock

**AIRWAY – BREATHING – VOLUME**

CONTROL HEMORRHAGE

Any obvious external source of hemorrhage is controlled by direct pressure and not by tourniquets. Avoid blind clamping as means of control. On-going internal hemorrhage requires a laparotomy.

RESTORE CIRCULATING VOLUME

- Bolus with 20 mg / kg NS or LR
- Give blood if no improvement after 2 boluses
- Blood should be typed and cross-matched if there is time
- Type-specific blood or type O if patient is exsanguinating

The selection of appropriate fluid in resuscitation is important. Because the elevation of serum glucose above the renal threshold may induce diuresis, solutions containing glucose should be avoided: The increased urine output may be taken as an indication that effective circulating blood volume and renal perfusion have been restored. In addition, greater volumes of fluid are required for similar effect. Normal saline solutions may lead to a dilutional acidosis. Colloidal solution are excellent plasma expanders when capillary membrane integrity is intact, but there is some evidence that they may cause greater problems with interstitial edema than crystalloids in the

presence of capillary leakage. In addition both dextran and hydroxyethyl starch interfere with platelet function if more than 1 litre is used. The use of hypertonic saline solutions can result in kaluresis, hypernatriemia and hyperosmolarity (cerebral complications may occur when the serum sodium exceeds 165mmol/l). Lactated Ringer's solution is widely used in the initial resuscitation of the injured patient. It is a common error to replace blood loss only with crystalloids. If a patient remains hypotensive after the rapid infusion of 2 liters of LR solution or if has been estimated that more than 30% of the circulating blood volume has been lost, transfusion with warmed packed red blood cells should be started (if necessary type 0 Rh negative for female and Rh positive for males).

OPTIMIZE OXYGEN DELIVERY

- airway management and supplemental oxygen as needed

## Decompensation

Intrinsic mechanisms and therapeutic intervention are usually helpful in the case of shock. Unfortunately, some patients decompensate and rapid deterioration can ensue:

- dilatation of systemic arterioles and precapillary sphincters
- failure of cell membrane function (fluid shifts)
- pooling of blood in capillaries and veins (65-85% of TBV)
- vascular endothelial dysfunction
  -microembolic (WBC and platelet) occlusion of capillaries, especially in lungs and liver
  -systemic inflammatory response syndrome (SIRS) with release of toxic factors such as free radicals and kinins with resulting fluid extravasation

multi-system organ failure (MOF) → death

Chapter

5

# The Acute Abdomen

*Accurate diagnosis and rapid action are two hallmarks in the treatment of an acute abdomen. This chapter will describe the surgical approach in detail, from the physical examination to the appropriate surgical intervention. A flowchart approach has been taken in order to facilitate the often-overwhelming clinical scenario. Although many of you are seasoned experts in the treatment of the acute abdomen, this chapter will serve as a refresher for your knowledge, and a way to organize your critical thinking pathway for the acute abdomen.*

OBJECTIVES

Know the criteria for diagnosis of the acute abdomen

Describe a differential diagnosis for each quadrant

Be able to utilize supporting tests and studies

## An Approach to the Acute Abdomen

There are few situations in clinical medicine, which demand prompt and decisive action as frequently as does acute abdominal pain. Acute conditions of the abdomen are produced by inflammatory, obstructive, or vascular mechanisms and are manifested by sudden onset of abdominal pain, gastrointestinal symptoms and varying degrees of local and systemic reaction. They require urgent treatment, often including emergency operation. Their urgency usually precludes prolonged investigation and there are few specific tests or examinations, which may be relied upon to give clear-cut answers as to the exact cause of the acute condition.

If surgery carried no risk and did not adversely affect the course of some diseases, it would be safe to say "if in doubt, operate." Unfortunately, laparotomy itself carries risks and the course of some disorders such as acute pancreatitis and paralytic ileus is adversely influenced by anesthesia and surgery.

The diagnosis of acute conditions, therefore, frequently resolves itself into arriving at a fairly immediate judgment derived from an accurate and detailed history, a careful physical examination and a few selected lab tests and x-ray studies. While gathering the

evidence, changes should be evaluated in terms of pathophysiologic alterations rather than specific diagnoses, and attention must be given to the need for supportive measures while investigation is underway.

### Definition of the Acute Abdomen

The acute abdomen may be defined generally as an intra-abdominal process causing severe pain and often requiring surgical intervention. It is a condition that requires a fairly immediate judgment or decision as to management. General causes of the acute abdomen may be divided into six large categories:

1. inflammatory
2. mechanical
3. neoplastic
4. vascular
5. congenital defects
6. traumatic

Each of these categories has many typical examples, of which only a few of the more common conditions will be discussed in this course.

### Inflammatory

The inflammatory category of causes may be divided into two subgroups: 1) bacterial, and 2) chemical. Some common examples of the bacterial causes would include acute appendicitis, diverticulitis, and some cases of pelvic inflammatory disease. An example of a chemical cause would be a perforation of a peptic ulcer, where spillage of acid gastric contents causes an intense peritoneal reaction.

### Mechanical

Mechanical causes of an acute abdomen include such obstructive conditions as incarcerated hernia, post-operative adhesions, intussusception, malrotation of the gut with volvulus, congenital atresia or stenosis of the gut. The most common cause of large bowel mechanical obstruction is carcinoma of the colon.

### Vascular

Vascular entities producing an acute abdomen include mesenteric arterial thrombosis or embolism. When the blood supply is cut off, necrosis of tissue results, with gangrene of the bowel.

### Congenital

Congenital defects can produce an acute abdominal surgical emergency any time from the minute of birth (with conditions such as duodenal atresia, omphalocele or diaphragmatic hernia) to years afterward in conditions such as chronic malrotation of the intestine.

### Traumatic

Traumatic causes of an acute abdomen range from stab and gunshot wounds to blunt abdominal injuries producing such conditions as splenic rupture. History or evidence of trauma should make this diagnosis fairly obvious.

## The Emergency Management of the Patient with an Acute Abdomen

### Immediate Assessment

Every patient who presents with acute abdominal pain needs to have the following recorded on arrival:

- pulse rate
- blood pressure
- respiratory rate

Any of the following alerts the attending doctor to an underlying serious condition.:

- Pulse rate > 100/min
- Systolic blood pressure < 90mm Hg
- Respiratory rate > 25/ mm

## The Three Most Common Life-Threatening Conditions

1. Ruptured abdominal aortic aneurysm
2. Ruptured ectopic pregnancy

3. Septic shock secondary to generalized peritonitis

These patients require resuscitation and treatment while their assessment is still going on. These conditions are easily overlooked if the examining doctor does not specifically consider them.

### Ruptured abdominal aortic aneurysm

(A problem worldwide, although not extremely common in Vietnam)

#### History

Most commonly in patients >50 years

Sudden onset of pain in the abdomen, back or loin

Pain often radiates to the groin (not unlike renal colic)

Pain usually very severe

Associated with vomiting and sweating

May be a history of hypertension or ischemic heart disease

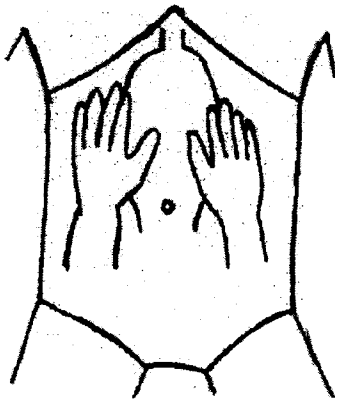
#### Examination

Appear acutely ill and distressed

Tachycardic

May still have a normal blood pressure or even be hypertensive on arrival

Key to diagnosis is careful palpation of the abdomen



An aneurysm can usually be felt by gentle palpation with the hand resting in the midline just above the umbilicus. The pulsatile mass can be confirmed as an aneurysm by bimanual palpation to detect the transverse nature of the pulsation.

### Treatment

Imaging should be reserved for stable patients with diagnostic uncertainty. Immediate surgery is required for control of the hemorrhage and aortic grafting. As the patient is being transferred to the operating theatre the following should be carried out:

- Type and cross match 6 units of blood
- Complete blood count
- BUN and electrolytes

Resuscitation should begin but should not delay transport to the operating room. The blood pressure should not be elevated above 100mm Hg systolic, if possible, to avoid aggravating hemorrhage.

## Ruptured Ectopic Pregnancy

Ruptured Ectopic Pregnancy should be considered in any female of childbearing age who presents with acute abdominal pain. In its most dramatic form these patients present collapsed with pallor, hypotension, and generalized abdominal tenderness.

### History

Sudden onset of lower abdominal pain

Pain may become generalized

Often associated with a syncopal episode

Pain is constant and made worse by moving and coughing

Pain may radiate to either shoulder especially while lying down

A missed menstrual period is usual but not universal

Often vomiting is absent.

### Examination

Patients with significant bleeding will be pale, tachycardic, tachypneic and hypotensive

Guarding at least involving the lower abdomen

Rebound tenderness that is often more marked than the tenderness would suggest

Shifting dullness

Vaginal examination - bulky uterus and cervical excitation.

### Treatment

IV normal saline should be started immediately

- Correct hypotension

*Send blood for:*

- Type and cross match 6 units
- Complete blood count
- BUN and electrolytes
- Pregnancy test

The on call Gynecologist should be informed immediately and ultrasound urgently requested.

### Septic Shock

The common causes are:

- generalized peritonitis, secondary to a ruptured viscus
- appendix
- diverticulitis
- peptic ulcer
- biliary sepsis
- ischemic bowel.

### History

Abdominal pain of at least 6 hours (often of several days duration)

Pain often begins focally then becomes generalized

Urine output is reduced

No bowel motion

Relatives often alarmed at patient's condition

### Examination

"Hot shock"- warm periphery, bounding pulse

"Cold shock"- cold, clammy periphery, feeble pulse (serious prognostic sign)

Tachycardia

Hypotension and tachypnea

Abdomen tender and guarded in all areas

### Treatment

- Urgent resuscitation before any surgery is undertaken
- IV line with normal saline
- Oxygen via mask
- IV antibiotics
- Correction of blood pressure while the remaining assessment is going on
- Involve anesthetist early and contact intensivist where available

## Essential History

Abdominal pain is the main, presenting symptom of patients with an acute abdomen. A carefully taken history describing the pain is the most powerful diagnostic tool.

The following features must be determined:

Onset

## How did it start?

*(Acuteness)*

sudden (minutes) → ruptured viscus, aneurysm or ectopic pregnancy

over 1-2 hours → pancreatitis, bowel obstruction, renal colic

over several hours → cholecystitis, appendicitis, PD

## When did it start?

*(Duration)*

>6 hours severe pain → suspect significant illness

## Where did it start?

*(Initial site)*

has it shifted? → appendicitis(first central then RLQ)

Nature

COLIC

| COLIC FEATURES: | CAUSED BY: |
|---|---|
| Severe pain | Bowel |
| Poorly localized | Gall bladder |
| Restlessness | Ureter |
| Sweating | |
| Reflex vomiting | |

INFLAMMATION:

| INFLAMMATION FEATURES: | CAUSED BY: |
|---|---|
| Constant | Appendix |
| Aggravated by movement or coughing | Gall bladder |
| Localized | Colon |

### Severity

Ask to assess on 1-10 scale. Severe pain is more likely to be significant illness.

### Location

Where is the pain? (generalized, upper, lower, right, left, central)

Does it go anywhere? (radiation)

> renal colic radiates loin to groin
>
> gall bladder pain to right scapula

### Aggravating and Relieving Factors

> coughing increases pain of peritoneal irritation
>
> eating relieves peptic ulcer pain

### Previous History

*(Medical and surgical history)*

> Is this a recurring problem?
>
> What were previous diagnoses?
>
> Any previous surgeries?

### Associated Symptoms

### Nausea/Vomiting

follows onset of pain in surgical causes may be: reflex (in colic), or from obstructed bowel is often absent in gynecological causes

### Constipation
common but obstipation (no stool or flatus) indicates a bowel obstruction

### Diarrhea
1 or 2 loose stools common at onset of peritonitis; profuse in gastroenteritis and inflammatory bowel disease (IBD)

### Blood in stool
IBD

intussusception

ischemic bowel

### Fever
mild in early appendicitis

if high consider abscess, pneumonia, systemic illness

### Urinary Symptoms
DYSURIA

urethral "burning" during micturition

→ UTI

→ bladder stone

abdominal pain during micturition

→ pelvic peritoneal irritation

FREQUENCY

→ UTI

→ stone in lower ureter

HEMATURIA

→ UTI

→ stone

→ tumor

### Menstrual Symptoms
alert you to gynecological causes

LMP date

menstrual irregularity

PV discharge

### Past History

Ask about:

Previous surgery

Medical conditions, medications

Recent trauma

## Examination

### General

pulse

blood pressure

respiratory rate

pallor

jaundice

general condition

### Abdomen

Inspection

Palpation

Percussion

Auscultation

### Further Examinations

Rectal examination

Vaginal examination

## Abdominal Examination

### INSPECTION

distention → bowel obstruction, ascites

visible bowel loops → bowel obstruction

scars → adhesions, recurrent illness, incisional hernia

hernia sites → don't miss a strangulated hernia!!

### PALPATION

start away from the site of pain; watch the patient's face.

guarding, rigidity, rebound tenderness → peritonitis

localized tenderness → appendicitis, cholecystitis, diverticulitis

search for enlarged organs → gall bladder, uterus, bladder, aorta

identify abnormal masses → tumor, abscess, strangulated bowel

check femoral pulses, hernia sites, scrotum

### PERCUSSION

gentle percussion detects rebound tenderness without pain!

shifting dullness identifies fluid → ascites, blood, pus

loss of liver dullness → perforated bowel

dullness lower chest → pleural effusion

### AUSCULTATION

bowel sounds

active, high pitched → bowel obstruction

absent → peritonitis, paralytic ileus

bruits → aorta, iliac, femoral- peripheral vascular disease

breath sounds → look for basal pneumonia

### Rectal Examination

peri-anal disease → IBD

rectal mass → may cause large bowel obstruction

abdominal pain on palpating pelvic peritoneum → pelvic peritonitis

blood → IBD, ischemic bowel

impacted stool → constipation

### Vaginal Examination

cervical excitation → Pelvic inflammatory disease (PID), ectopic pregnancy

purulent discharge → PID

bulky uterus → pregnancy

adnexal mass → ectopic pregnancy, ovarian cyst, tumor

## Pain and the Acute Abdomen

Since pain is the most prominent presenting complaint in a patient with an acute abdomen, it is important to know the origin, location, radiation and character of abdominal pain in order to understand its significance.

The perception of abdominal pain is first visceral and then becomes somatic. The abdominal viscera and the visceral peritoneum receive sensory fibers via the sympathetic chain from T5 through L3. The sensory supply to the viscera is sparse and visceral pain is vague and poorly localized. The alimentary tract from the esophagus to the anal canal is insensitive to many stimuli, which produce intense pain in other structures. The gut can be biopsied, crushed or cauterized without pain.

If the bowel or any other hollow viscus is distended or if its muscle coat goes into spasm, however, pain is felt. The cause of visceral pain is tension in the muscle fibers produced by stretching of the wall, spasm of the muscle or stretching of the capsule of the organ. Violent peristaltic contractions occur in an attempt to force lumenal contents through an obstruction. Pain associated with obstruction is severe and cramping in nature, but intermittent, with pain-free intervals and is called colic. Ischemia of visceral muscle gives rise to pain because the gut loses motility and

becomes distended. Visceral pain of ischemic origin is caused most often by strangulation of the bowel in hernia or volvulus. A less frequent cause is acute mesenteric thrombosis.

The parietal peritoneum, which lines the abdominal cavity and the interior surfaces of the diaphragm, derives sensory fibers from the somatic nerves T6 through L1. When the parietal peritoneum is irritated, somatic pain results. Somatic pain is with localized tenderness and spasm of the muscle groups supplied by the dermatome of origin of the pain stimulus. For example, the right lower quadrant (RLQ) pain, tenderness and muscle spasm associated with appendicitis is caused by inflammation of the contiguous RLQ parietal peritoneum. The abdominal signs in perforated peptic ulcer, on the other hand, are generalized because diffusion of highly acid fluid throughout the peritoneal cavity causes intense irritation of all the parietal peritoneal surfaces.

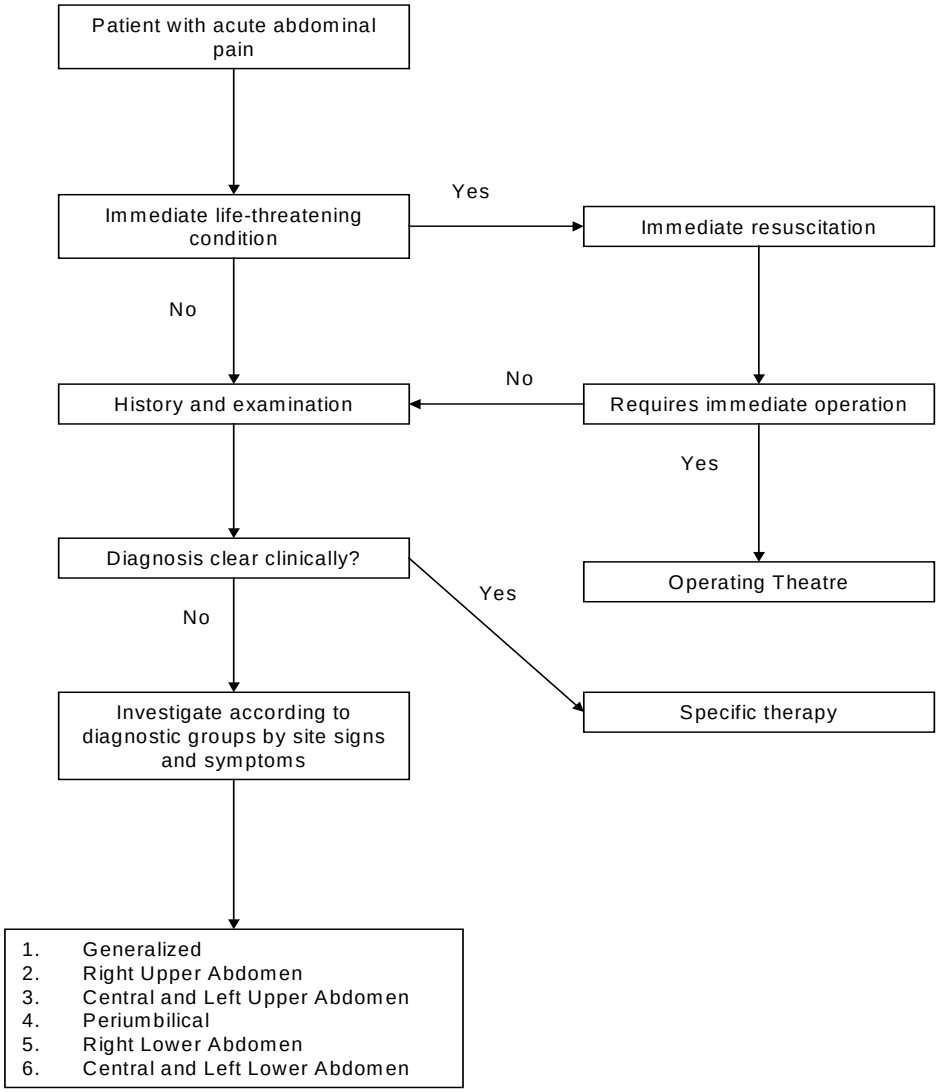
Pain experienced at a site other than that stimulated but in somatic zones supplied by the same or adjacent segments of the spinal cord is called referred pain. Visceral pain is referred to three zones located in the midline of the abdomen. The localization of abdominal pain indicates which organs may be involved. Epigastric pain is associated with structures innervated by T6-T8, the stomach, duodenum, pancreas, liver, biliary tree and associated parietal peritoneum. Periumbilical pain is related to innervation from T9 to T10 and includes the small intestine, appendix, and upper ureters. Hypogastric pain has its origin in structures innervated by Tll and T12, the colon, bladder, lower ureters and uterus.

The pattern of radiation of pain may provide important clues as to its origin. For example, pain, which initially is located in the periumbilical area and then moves to the RLQ occurs with appendicitis, whereas pain in the epigastrium that radiates to the tip of the right scapula is frequently found with acute cholecystitis. Such shifting or radiation of pain to a localized site with local tenderness and muscle spasm denotes local inflammation of the parietal peritoneum and suggests a circumscribed inflammatory process. The pain of renal colic usually is felt in the flank and radiates towards the groin on the same side.

Pain that involves the entire abdomen almost immediately after onset is usually due to flooding of the peritoneal cavity with an irritating fluid from a perforated ulcer, or from blood and chorionic tissue in a ruptured ectopic pregnancy.

A general rule to follow is that the majority of severe abdominal pain occurs in patients who have enjoyed fairly good health and which persists as long as six hours is caused by diseases requiring surgical intervention. Obviously, there are always exceptions to any rule.

# Acute Abdominal Pain Flowchart



# Generalized Abdominal Pain

### Investigations

Erect Chest X-ray

Erect and Supine Abdominal X-ray

Serum Amylase, Creatinine, Urea, Electrolytes

Full Blood Count

Urinalysis

ECG (if> 50 years)

### Initial Management

IV fluids (normal saline) may need bolus to correct hypovolemia.

Pain relief (Injectable NSAIDs and Opiates)

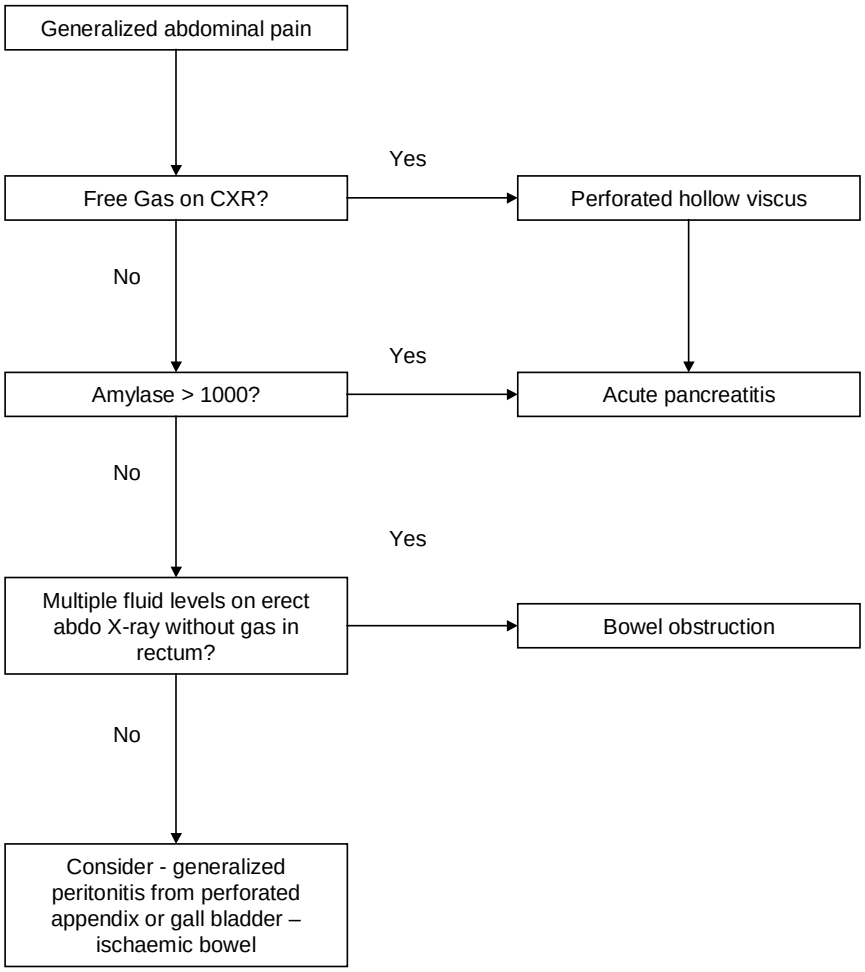
Nasogastric suction (esp. if perforation suspected)

Urinary catheter to monitor urine output

Consider Antibiotics

Surgical referral for definitive treatment

| COMMON CAUSES | DISTINGUISHING FEATURES |
|---|---|
| Perforated viscus with peritonitis e.g. perforated peptic ulcer diverticular disease | Sudden onset, free air |
| Acute pancreatitis | Alcohol, gallstones, amylase > 1000 |
| Bowel obstruction +1- stran2ulation | Colicky pain, vomiting, obstipation |
| Bowel ischemia | Severe pain, tenderness less marked than expected |


Generalized abdominal pain
Free Gas on CXR?
Yes
Perforated hollow viscus
No
Amylase > 1000?
Yes
Acute pancreatitis
No
Multiple fluid levels on erect abdo X-ray without gas in rectum?
Yes
Bowel obstruction
No
Consider - generalized peritonitis from perforated appendix or gall bladder – ischaemic bowel

# Right Upper Quadrant Abdominal Pain

### Investigations

Erect Chest X-ray

Erect and Supine Abdominal X-ray

Serum Amylase, Creatinine, BUN, Electrolytes

Complete Blood Count

Urinalysis

ECG (if > 50 years old)

### Initial management

IV fluids (normal saline) may need bolus to correct hypovolemia.

Pain relief (Injectable NSAIDs and Opiates)

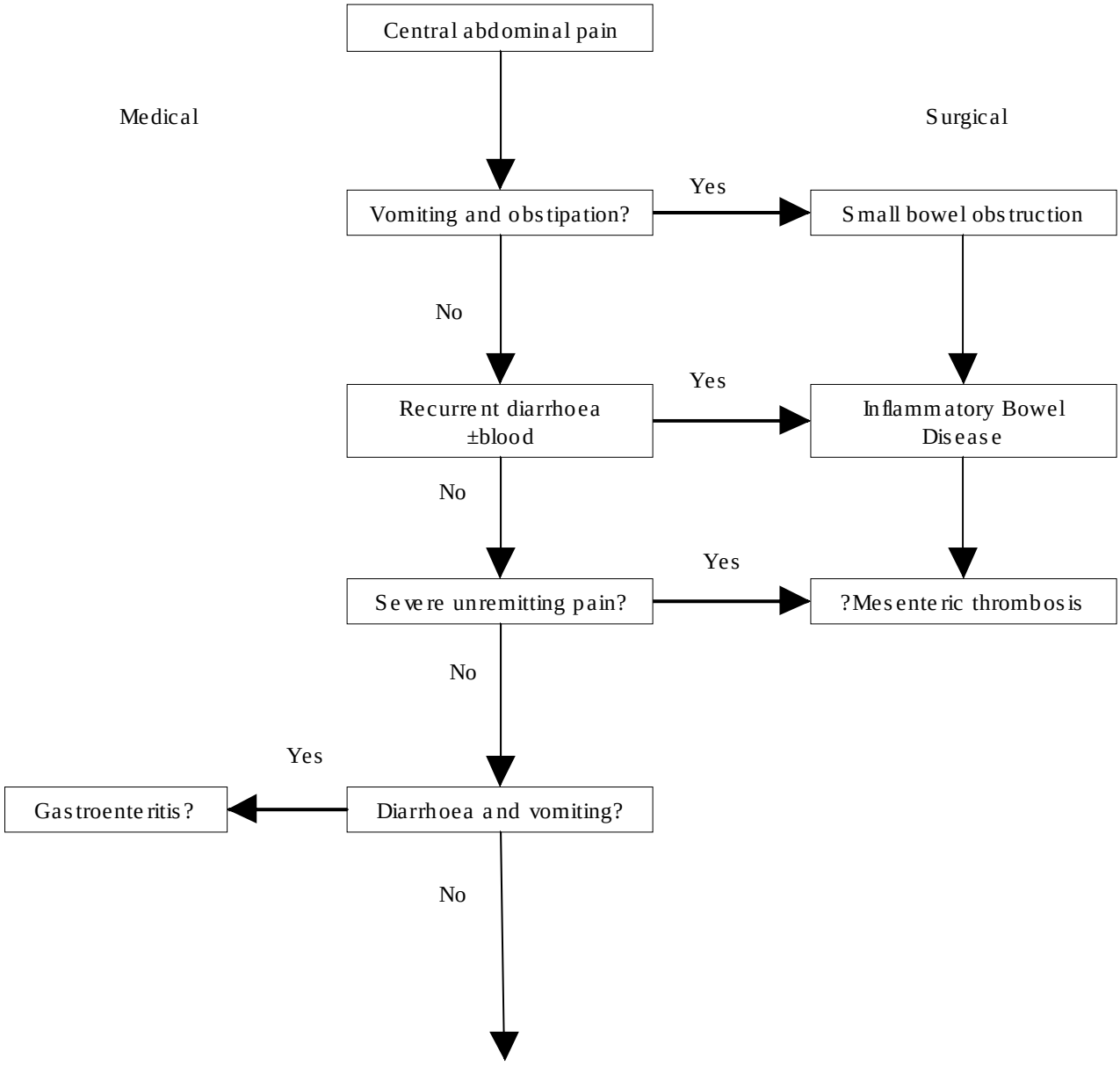
Nasogastric suction (esp. if perforation suspected)

Urinary catheter to monitor urine output

Consider Antibiotics

Surgical referral for definitive treatment

| COMMON CAUSES | DISTINGUISHING FEATURES |
|---|---|
| Biliary colic, acute cholecystitis | Recurrent attacks, tender over gall bladder area |
| Acute hepatitis | Alcohol, medication, jaundice |
| Right pyelonephritis | Dysuria, fever, CVAT |
| Congestive heart failure | Edema, dyspnea, elevated JVP etc |
| High retrocecal appendicitis | Shift of pain, tenderness laterally Fever, tachypnea, bronchial breathing |
| Right lower lobe pneumonia | |




Many patients will not fall into any of these groups and will resolve without diagnosis.
Most cases of appendicitis will localize in the right iliac fossa within six hours.

## Periumbilical Pain

Many patients with central abdominal pain will not require urgent surgery but require exclusion of more acute problems.

### Investigations

*Depends on the severity of symptoms*

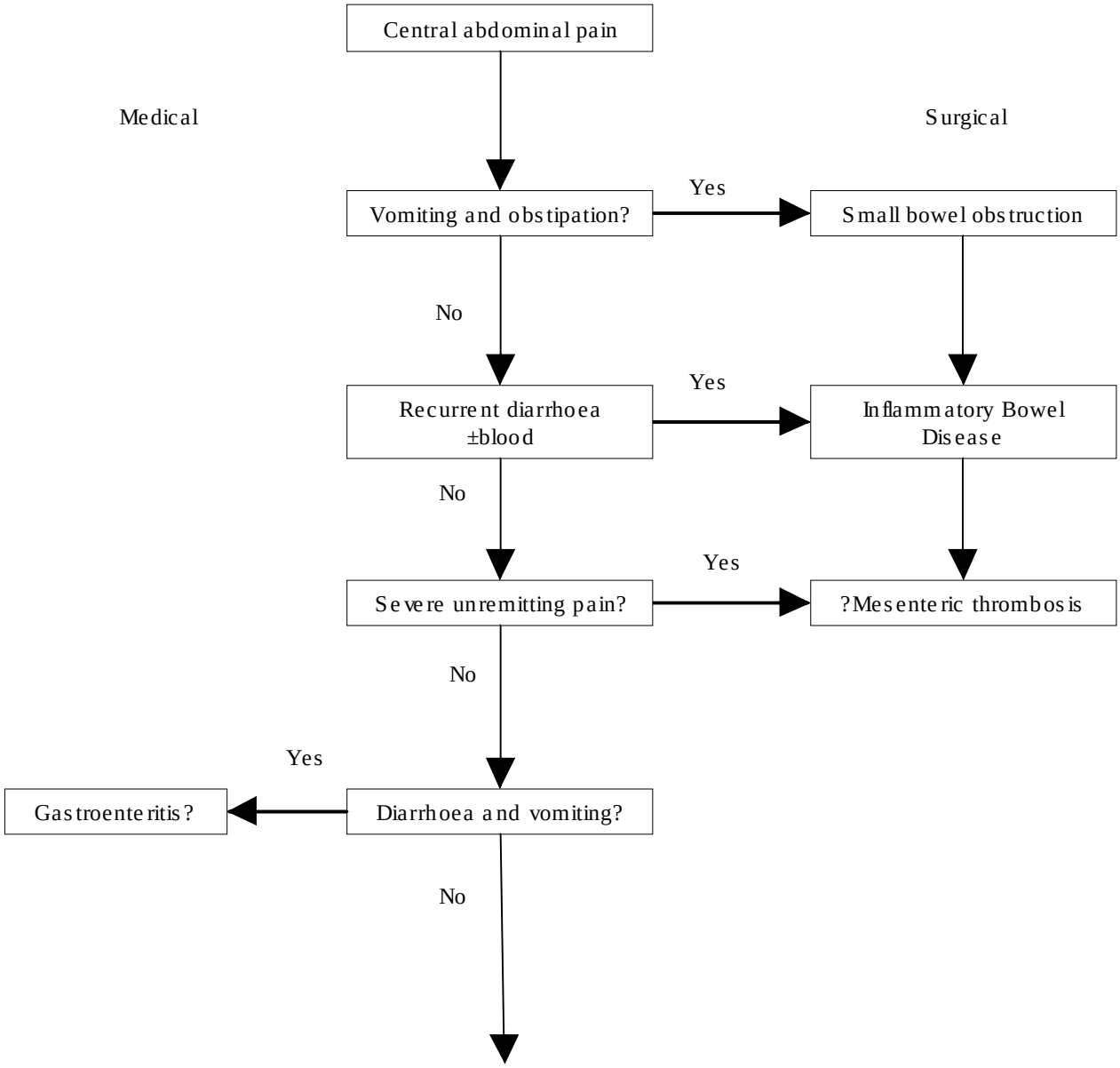
if severe unrelenting pain → urgent surgical referral

if pain colicky and no flatus → erect and supine abdominal X-ray

if diarrhea and vomiting → stool test

### Initial management will depend on specific diagnosis

| COMMON CAUSES | DISTINGUISHING FEATURES |
|---|---|
| Gastroenteritis | Vomiting and diarrhea |
| Constipation | Colicky pain, hard stool |
| Inflammatory bowel disease | Recurrent, diarrhea (+ mucus, blood) |
| Early appendicitis | Nausea, short history, later shift of pain |
| Small bowel obstruction | Colicky pain, vomiting, no flatus |
| Ischemic bowel | Severe pain, tenderness less marked, PR bleeding |




Many patients will not fall into any of these groups and will resolve without diagnosis.
Most cases of appendicitis will localize in the right iliac fossa within six hours.

# Right Lower Quadrant Pain

Investigations
*(Most of these diagnoses can be differentiated clinically)*

Urinalysis → to exclude an unsuspected urinary cause.

Pregnancy test → in women of childbearing potential

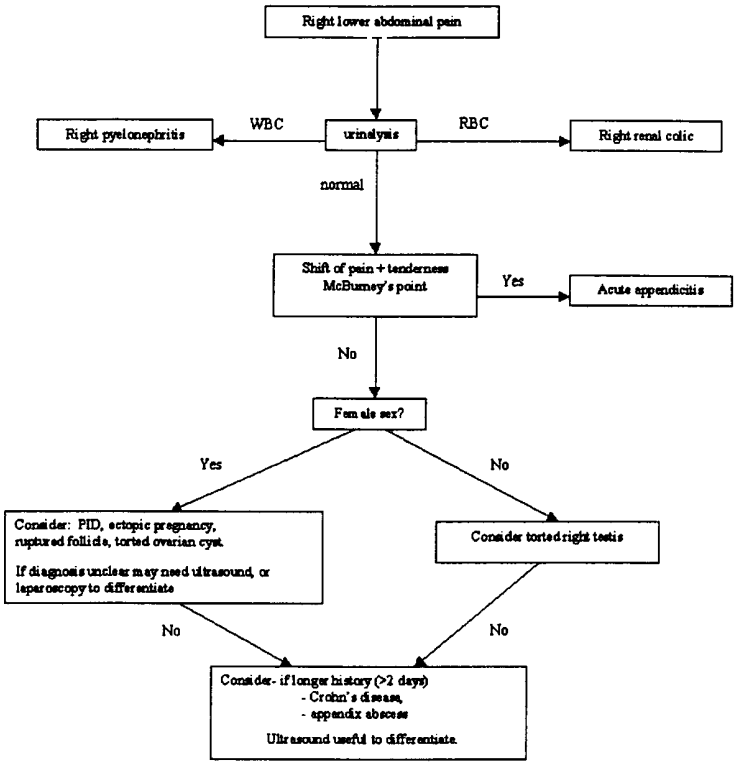
Ultrasound → if gynecological cause is suspected

Full blood count →of limited diagnostic value

Initial management will depend on specific diagnosis

| COMMON CAUSES | DISTINGUISHING FEATURES |
|---|---|
| Acute appendicitis | Shift of pain, localized tenderness, anorexia |
| Mesenteric adenitis | Fever, inconstant signs, children |
| Right renal colic | Colicky pain, radiation, hematuria |
| Toiled right testis | Tender swollen testis, young age |
| Crohn's disease | Several days history, recurrent |

| GYNECOLOGIC CAUSES | |
|---|---|
| Ruptured follicle | Fever, cervical excitation, PV discharge |
| Torsed ovarian cyst | Midcycle, sudden onset |
| Ruptured ectopic pregnancy | Severe pain, vomiting |
| PID | Sudden onset, amenorrhea, shock, cervical excitation |


Right lower abdominal pain
Right pyelonephritis
WBC
urinalysis
RBC
Right renal colic
normal
Shift of pain + tenderness
McBurney's point
Yes
Acute appendicitis
No
Female sex?
Yes
No
Consider: PID, ectopic pregnancy,
ruptured follicle, torted ovarian cyst.
If diagnosis unclear may need ultrasound, or
laparoscopy to differentiate
Consider torted right testis
No
No
Consider- if longer history (>2 days)
- Crohn's disease,
- appendix abscess
Ultrasound useful to differentiate.

# Left Lower Quadrant Abdominal Pain

### Investigations

*(Most of these diagnoses can be differentiated clinically)*

Urinalysis to exclude an unsuspected urinary cause.

Pregnancy test if appropriate

Ultrasound if gynecological cause is suspected

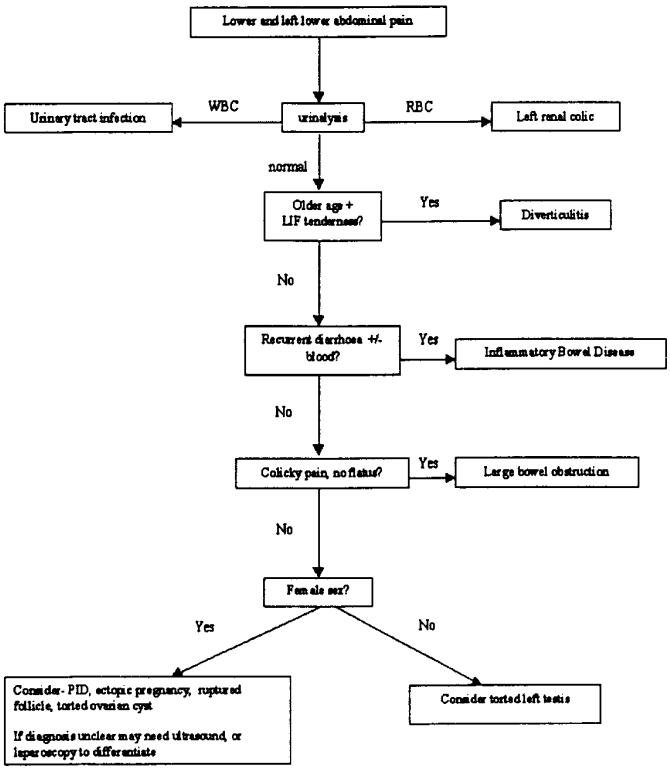
Full blood count

Erect and supine abdominal X-rays

CT scan if complication of diverticular disease is suspected

### Initial management will depend on specific diagnosis

| COMMON CAUSES | DISTINGUISHING FEATURES |
|---|---|
| Diverticular disease | Elderly, recurrent |
| Acute urinary retention | Palpable bladder, difficulty passing urine |
| Urinary tract infection | Dysuria, frequency |
| Inflammatory bowel disease | Recurrent attacks, diarrhea (mucus / blood) |
| Large bowel obstruction | Colicky pain, obstipation |
| Left renal colic | Colicky pain, hematuria |
| Torted left testis | Tender swollen testis, young age |
| GYN causes, as for RLQ | |


Lower and left lower abdominal pain
Urinary tract infection
WBC
urinalysis
RBC
Left renal colic
normal
Older age + LIF tenderness?
Yes
Diverticulitis
No
Recurrent diarrhoea +/- blood?
Yes
Inflammatory Bowel Disease
No
Colicky pain, no flatus?
Yes
Large bowel obstruction
No
Female sex?
Yes
No
Consider- PID, ectopic pregnancy, ruptured follicle, torted ovarian cyst
If diagnosis unclear may need ultrasound, or laparoscopy to differentiate
Consider torted left testis

# Topics

## Acute appendicitis

### History

- Shift of central pain to right iliac fossa
- Pain precedes anorexia and nausea
- Worse on moving and coughing

### Examination

- Modest fever
- Tenderness, guarding and rebound in right iliac fossa

### Diagnosis

- Essentially clinical
- Exclude urinary cause by urinalysis
- Ultrasound in selected cases

### Treatment

- Nothing by mouth
- Intravenous fluids
- Pain relief
- If history < 48 hours early appendectomy with antibiotic cover
- If history > 48 hours and mass palpable
- Nothing orally, IV fluids and antibiotics
- If abscess on Ultrasound - drainage
- Interval appendectomy after 6 weeks

## Acute cholecystitis

### History

Commoner in middle-aged women

Often have a history of previous attacks

Pain in right upper quadrant

May radiate to right scapula

### Examination

Moderate fever

Patient lies still

Tenderness and rebound right upper quadrant ± tender mass

Positive Murphy's sign (arrest of inspiration while palpating over gall bladder)

### Diagnosis

Ultrasound shows gallstones and edematous wall

### Treatment

Nothing by mouth

Intravenous fluids

Pain relief

Antibiotics

Surgery on the first available elective list

Occasionally necrosis or perforation of gallbladder → urgent operation

## Acute pancreatitis

### History

Alcohol intake or gall stone history

Severe epigastric pain, may be generalized

Vomiting

### Examination

Appears very unwell

Tenderness with guarding and rebound upper abdomen

Cardiovascular collapse or respiratory distress in severe cases

### Diagnosis

Amylase> 1,000 in absence of GI perforation makes diagnosis very likely Ultrasound for gallstones, bile duct dilatation, and pancreatic swelling

### Assessment of Severity

RANSON'S CRITERIA: For Determining Prognosis of Acute Pancreatitis

| UPON ADMISSION: | AFTER 48 HOURS: |
|---|---|
| Age > 55 | HCT decrease > 10% |
| Blood Glucose > 200 mg/dl | Serum Ca2+ < 8mg % (mg/dl) |
| WBC count > 16,000 / cu mm | Base Deficit > 4 meq/l |
| Serum LDH > 700 IU% | BUN increase > 5mg % (mg/dl) |
| SGOT > 250 SF units % (56 units/dl) | est. fluid retention > 6L |
| < 3 signs - better prognosis, &LT 1% mortality. | |
| > 3 signs - serious acute pancreatitis - may be 25% mortality. | |

### Treatment

*Initially non-operative*

Nothing by mouth

Naso-gastric suction

IV fluids

Antibiotics

Most settle without surgery but if complications of pancreatic abscess or pseudocyst arise surgery may be indicated.

### Small bowel obstruction

#### History

Colicky, central abdominal pain

Patient writhes during attacks of pain

Bilious vomiting

No flatus

Severe unrelenting pain suggests strangulation

Previous surgery suggests adhesions

Groin lump suggests obstructed hernia

#### Examination

Central abdominal distention

Operation scars, hernia, masses

Bowel sounds active in high-pitched runs

Rebound tenderness suggests strangulation

#### Diagnosis

Clinically obvious if tender irreducible hernia present

Erect and supine abdominal X-rays show fluid levels on erect film with colon not dilated

Rebound tenderness, guarding, rising pulse suggest strangulation

#### Treatment

- Naso-gastric aspiration

- Intravenous fluids (resuscitate if hypovolemia)
- Pain relief
- Early surgery if:
    - Irreducible hernia
    - Strangulation suspected

## Acute diverticulitis

### Background

Incidence of diverticular disease increases with age, > 50% if age> 80 years

Usually involves sigmoid colon

20% of patients develop diverticulitis

### History

Constant, severe left iliac fossa pain

Fever ± chills

Recurrent attacks

### Examination

Localized left iliac fossa guarding + rebound tenderness (generalized if free perforation)

May be mass palpable (phlegmon, abscess)

Tenderness rectally

### Diagnosis

Clinically suspected

CT scan to identify complications

### Treatment

*Initially medical*

Intravenous fluids

Nothing orally

NG aspiration if distended

Intravenous antibiotics

If free perforation and peritonitis- laparotomy and Hartmann's procedure

If abscess- percutaneous drainage

## Large bowel obstruction

### Background

The commonest cause of large bowel obstruction is carcinoma of the colon.

Other causes include volvulus and chronic diverticular disease.

### History

Colicky, central or lower abdominal pain

Obstipation (no stool or flatus)

### Examination

Abdominal distention especially in flanks

Active, high-pitched bowel sounds

Rectal examination may detect tumor

### Diagnosis

Erect and supine abdominal X-rays show dilated colon

Sigmoidoscopy may reach lesion

Urgent barium enema is usually diagnostic

### Treatment

- Intravenous fluids to correct hypovolemia and dehydration Laparotomy under antibiotic cover

## Perforated peptic ulcer

### History

Previous history of epigastric pain

Sudden onset, severe upper abdominal pain

### Examination

Patient lies still

Rigid abdomen

Liver dullness may be lost

### Diagnosis

Free gas under diaphragm on erect chest X-ray (negative in 20% of cases)

Gastrografin meal if required

### Treatment

- Nasogastric suction
- Intravenous antibiotics
- Intravenous normal saline to correct hypovolemia
- Urgent laparotomy

## Medical causes of acute abdominal pain

- Myocardial infarction
- Lobar pneumonia
- Drugs (e.g. digoxin, warfarin)

- Acute hepatitis
- Diabetic ketoacidosis
- Herpes Zoster
- Red cell abnormalities (Sickle cell disease, porphyrias, congenital spherocytosis)
- Lead poisoning
- Campylobacter and Yersinia infections
- Spurious abdominal pain (Munchausen's syndrome)

## Rectal Bleeding

### Scope

The aim of this review is to outline management recommendations for the assessment and treatment of rectal bleeding that presents as an emergency to a secondary or tertiary level medical institution. It does not deal with the elective investigation of intermittent rectal bleeding that is commonly seen in the outpatient setting.

### History and examination

A careful directed history and examination are essential.

From the history determine the following:

1. Rate and volume of blood loss
2. Abdominal pain (presence, location and character)
3. History of recent or previous surgery or colonoscopy (polypectomy)
4. Past history of bowel disease, aortic surgery or upper GI disease
5. Family history of inflammatory bowel disease, or colorectal cancer
6. Drug history especially of use of aspirin, NSAIDs or anticoagulants

On examination look for:

1. Shock (pale, tachycardia, hypotension, sweaty, tachypneic, cool peripheries)
2. Stigmata of chronic liver disease / telangectasia / pigmentation
3. Abdominal tenderness / aortic aneurysm / abdominal mass / hepatomegaly
4. Anal and rectal examination for polyps, carcinoma or pelvic masses.

### Causes and natural history

The majority of patients (—80%) admitted with rectal bleeding will settle spontaneously without the need for surgery during the presenting admission. In those who continue to bleed after admission imaging is important to determine the site of bleeding provided the patient's condition is stable. Older patients with cerebrovascular or coronary artery disease are intolerant of episodes of hypotension and require regular clinical review and may require early surgery.

Common causes of rectal bleeding are:

1. Colonic diverticular disease
2. Colonic angiodysplasia
3. Colorectal cancer (rarely lymphoma)
4. Colonic polyps (adenomas, hamartomas, juvenile, inflammatory and malignant polyps)
5. Inflammatory bowel disease (ulcerative colitis / Crohn's colitis / indeterminate colitis)
6. Ischemic colitis
7. Meckel's diverticulum and other small bowel causes
8. Infective causes (esp shigella, salmonella, amoebiasis)
9. Aortoenteric fistula (nearly always after aortic grafting)
10. Hemorrhoids (may be heavy and persistent) / fissure / anal tumor
11. Secondary hemorrhage after hemorrhoidectomy / polypectomy
12. Coagulopathy

13. Radiation proctitis

In young adults think of:

- Inflammatory bowel disease
- Meckel's diverticulum
- Hamartomatous (Peutz-Jegher /juvenile) polyps
- Familial adenomatous polyposis (FAP)

Assessment and immediate management

In common with other surgical emergencies attention in the potentially shocked and bleeding patient is first directed at the *Airway, Breathing and Circulation*.

***Airway*** It is unusual for the Airway to be compromised except in the grossly shocked patient. An oropharyngeal airway is indicated if the patient is unable to maintain a patent airway.

***Breathing*** Adequacy of respiratory effort is assessed and high flow oxygen via a facemask is mandatory in all shocked patients.

***Circulation*** is assessed by the pulse, blood pressure, postural drop in blood pressure and the peripheral perfusion.

Action

1. Insert two large peripheral IV (16g or >) and administer high flow 02
2. Resuscitate with crystalloid
3. Take blood for CBC, cross match 4-6 units of packed cells, clotting studies
4. Insert urinary catheter
5. Perform rectal examination, proctoscopy and rigid sigmoidoscopy
6. If the above do not demonstrate a cause of bleeding arrange urgent gastroscopy (if available)
7. If an upper GI cause of bleeding is excluded admit the patient to an HDU or if stable to a surgical ward.

Subsequent management depends on:

- cardiovascular stability of the patient
- presence of ongoing hemorrhage
- availability of special investigations

**Passage of a nasogastric tube and the aspiration of bile stained (i.e. non blood stained) fluid may be used to exclude an upper GI source of hemorrhage but this test has a 10-15% false negative rate*

Role of surgery

Surgery is only required in a minority of patients with rectal bleeding as most will stop spontaneously. Surgery is indicated in those patients who continue to bleed after admission to hospital or in those who re-bleed while in hospital. Serious consideration is given to surgery on any patient requiring more than four units of blood or in the unstable patient who shows evidence of ongoing bleeding with hypotension and tachycardia.

Therefore surgery is required if:

1. significant bleeding continues after admission (>4 units blood)
2. the patient has a major re-bleed while in the hospital
3. localization of the site of bleeding is helpful to the surgeon but not essential

Operative strategy

→ if the bleeding point has been accurately localized segmental resection (usually a hemicolectomy) is performed after a laparotomy has been undertaken

→ if the bleeding site is unknown a careful search of the whole of the gut is made.