Case 1
Clavicle Fracture
(Broken Collarbone)

Anatomical Region and Surface Anatomy of the Clavicle.

The clavicle is located in the pectoral girdle. It is located also in the anterior and superior portion of the thorax, placed almost horizontally above the first rib. Medially the clavicle articulates with the manubrium of the sternum, at what is known as the sternoclavicular joint. Laterally the clavicle articulates with the acromion of the scapula at the acromioclavicular joint. Because the clavicle is the only skeletal connection of the pectoral girdle to the trunk of the body, it serves as a strut which maintains the upper limb at a suitable distance from the thorax, so as to allow for maximum movement of the arm. The clavicle also serves to protect underlying neurovascular bundles – the subclavian vessels, the transverse scapular vessels and the brachial plexus of nerves. The clavicle presents a double curvature – medially it presents an anterior convexity, while laterally it presents an anterior concavity. The lateral end of the bone is somewhat flattened, the medial end is more rounded or prismatic.

Superior Surface of left Clavicle

The lateral third is seen to have two borders and two surfaces. The anterior border is concave, while the thicker posterior border is convex. Both are marked with impressions for the attachment of the deltoid muscle on the anterior border and the trapezius muscle on the posterior border. The superior surface is flat, and between the points of attachment of deltoid and trapezius lies an area of subcutaneous bone. On the posterior border of the inferior surface lies a rough eminence known as the coracoid tuberosity (conoid tubercle) which is the point of attachment of the conoid ligament. Running anterolaterally from this is the oblique or trapezoid ridge, which is the point of attachment of the trapezoid ligament. Together these ligaments make up the coracoclavicular ligament.

The medial two-thirds of the clavicle has three borders and three surfaces. The anterior border, continuous with the anterior margin of the lateral third, is the point of attachment of the pectoralis major. The superior border, continuous with the posterior border of the lateral third, is the point of attachment of the sternocleidomastoid muscle. The posterior or subclavian border runs from the coracoid tuberosity to the costal tuberosity. This represents the posterior boundary of the groove for subclavius muscle. The anterior surface lies between the anterior and superior borders. The posterior, or cervical border lies between the posterior, or subclavian border and the superior border and faces toward the root of the neck. This surface is in contact with the transverse scapular vessels, the subclavian vessels and the brachial plexus of nerves. It also contains the oblique foramen, which is the point of entry of the chief nutrient artery to the bone. The inferior or subclavian surface lies between the anterior and subclavian borders. Features include the costal tuberosity (rhomboid impression) for attachment of the costoclavicular ligament, and the subclavian groove, which is the point of attachment of the subclavius muscle.

Inferior Surface of left Clavicle

The sternal extremity of the clavicle is triangular in form, and faces
anteriorly, inferiorly and medially. It articulates with the manubrium of the sternum at the sternoclavicular joint. It presents an articular facet, which through the intervention of an articular disc allows this articulation. The lower part of the articular facet extends on to the inferior surface and articulates with the cartilage of the first rib. The circumference of the articular surface is rough, as it is here that the ligaments supporting the sternoclavicular joint attach. These include the costoclavicular and sternoclavicular ligaments.

The acromial (lateral) extremity is smaller and somewhat oval-shaped. It is directed obliquely and inferiorly, and articulates with the acromion of scapula. The circumference of the articular surface of this extremity is also rough, again due to it being the point of attachment of ligament (coracoclavicular ligament).

Because the clavicle is so well anchored at both extremities, it is unlikely to fracture in the medial or lateral thirds. In 80% of cases the fracture will take place in the middle third of the shaft. It is common for the fragments of bone to be displaced following a fracture. This is due to the action of the muscles attached to the bone. The sternocleidomastoid causes the medial fragment to be displaced upwards. The trapezius is unable to hold up the lateral fragment, which is consequently displaced by the pull of the deltoid muscle and the weight of the arm.

**Definition:**
A clavicle fracture is a break in the clavicle bone (also called the collarbone). It connects the sternum (breastplate) to the shoulder.

The clavicle can fracture in three different places:
- **Middle Third** – the middle portion of the clavicle, which is the most common site for a clavicle fracture.
- **Distal Third** – the end of the clavicle connecting to the shoulder
- **Medial Third** – the end of the clavicle connecting to the sternum

On each side, the clavicle extends horizontally from the manubrium of the sternum medially to the acromion of the scapula laterally. It is typically 13-15 cm in length. The medial two-thirds is convex anteriorly and roughly quadrilateral in cross-section. The lateral one-third is concave anteriorly and flatter.

The anterosuperior surface of the bone is smoother than the inferior surface and lies subcutaneously. It is crossed by the supraclavicular nerves and occasionally a communication between the external jugular and cephalic veins.

**Understanding the Anatomy**
Physicians need to understand the anatomy of the injured region and the mechanics of the injury to make the proper diagnosis and suggest appropriate treatment. The clavicle is an S-shaped bone that connects the shoulder girdle to the trunk. It is a rigid strut that maintains the shoulder in a functional position in relationship to the axial skeleton and allows varied hand positions in sports. The deltoid, pectoralis major, trapezius, sternocleidomastoid, and subclavius all insert or originate on the clavicle and can cause deforming forces after an injury.

In addition to its structural function, the clavicle protects major underlying neurovascular structures as they pass from the neck to the axilla. This fact is especially important because clavicle fractures—especially of the medial aspect—can compromise the costoclavicular space and injure neurovascular structures (2,3). Fortunately, clavicle injuries with associated significant neurovascular injuries are quite rare.

The clavicle articulates with the acromion to form the acromioclavicular (AC) joint and with the sternum to form the sternoclavicular (SC) joint (figure 1: not shown). The AC joint is a diarthrodial joint between the lateral end of the clavicle and the medial margin of the acromion. Several ligaments reinforce the joint capsule to maintain stability. The AC ligament and the capsule provide anterior-posterior and medial-lateral stability, while the coracoclavicular ligaments provide vertical stability. Only when the coracoclavicular ligaments are completely torn is the distal clavicle prominent in a shoulder separation.

The SC joint depends on the surrounding capsule and ligaments for stability. Ligamentous structures include the SC ligament (or capsular ligament), an intra-articular disk ligament, an interclavicular ligament, and an extra-articular costoclavicular ligament (rhomboïd). The SC ligament is the strongest and most important structure preventing upward displacement of the medial clavicle (4). The costoclavicular ligament is an important stabilizer if a proximal resection is necessary.

Of particular interest is the close proximity of the great vessels and pericardial contents just posterior to the SC joint. These vessels can be compromised by a posterior SC dislocation. Also, the proximal clavicular epiphysis is the last growth plate in the body to fuse (at approximately 22 years). Therefore, many SC injuries in young athletes are probably physeal injuries.
Causes:
A clavicle fracture is caused by trauma to the clavicle bone. The trauma is usually caused by:
- Direct blow to the clavicle
- Falling on an outstretched arm
- Newborn babies can break a clavicle passing through the birth canal

Mechanism of Clavicle Injury:
The mechanism of injury can be particularly helpful in diagnosing clavicle injuries. Any significant trauma causing elevation, depression, retraction, or anterior or posterior translation of the shoulder will transmit forces to the axial skeleton via the clavicular complex anteriorly or the periscapular muscles posteriorly. Since the clavicular complex is the more rigid of the two, failure along the clavicular complex is much more common than injury to the periscapulars.

The actual mechanism of injury can be direct or indirect (figure 2: not shown). Direct injuries are associated with contact sports and stick sports (such as football, hockey, or lacrosse) in which the athlete sustains a direct blow to the clavicle (5). Fractures are the common result of direct injuries. Blows that direct a downward force at the superior aspect of the acromion may lead to AC injury or a shoulder separation.

Indirect injuries are due to falls onto the outstretched arm or onto the lateral border of the shoulder. Fractures of the middle third of the clavicle are most frequently seen in falls onto outstretched arms, while fractures of the distal third are most often associated with loads transmitted to the lateral border of the shoulder (6). If an athlete falls or is hit on the lateral border of the acromion, the force is transmitted first to the site of impact, then to the AC joint, then to the clavicle, and then to the SC joint. Failure can occur at any one of these sites.
Symptoms:
- Pain, often severe
- Sagging shoulder, down and forward
- Inability to lift the arm because of pain
- A lump or visible deformity over the fracture site
- Tenderness and swelling of the affected area
- Loss of pulse below fracture

The list of symptoms mentioned in various sources for Fractures includes:

- Local bleeding
- Symptoms of associated nerve damage:
  - Numbness
  - Paralysis

*Numbness* usually arises from damage or disease of nerves. Numbness is loss of sensation, preceded by abnormal pain-like sensations often described as pins-and-needles, prickling or burning sensations; these are called *paresthesias*. Any numbness or abnormal sensation symptoms need prompt professional medical advice.

*Paralysis* usually involves both the loss of the ability to move the area and loss of sensations.

Physical:
- Tenderness
- Crepitus
- Edema
- Deformity
- Ecchymosis, especially when severe displacement causes tenting of skin
- Bleeding from open fracture (rare)
- Decreased breath sounds on auscultation, indicating possible pneumothorax
- Decreased pulses or evidence of decreased perfusion on vascular exam, suggesting vascular compromise
- Diminished sensation or weakness on distal neurovascular exam, suggesting neurologic compromise
- Nonuse of arm on affected side in neonates

Diagnosis:
Physicians need to be familiar with the anatomy of the region and the most common mechanisms of injury to be able to expeditiously diagnose and treat such injuries. Diagnosis is often straightforward, and conservative. The doctor will ask about your symptoms, physical activity, and how the injury occurred, and will examine the injured area.

Examination of the clavicle should be carried out standing behind the patient. Commence at the sternoclavicular joints and palpate laterally in a sliding motion along the smooth, subcutaneous anterosuperior surface. This surface is traversed by the thin sheet of platysma but occasionally, the supraclavicular nerves can be palpated.

Moving laterally, compare the bilateral symmetry of the convex medial two-thirds and concave lateral third. Swelling, crepitation or loss of continuity may indicate a fracture of the clavicle.

Tests may include:

**X-rays:** A test that uses radiation to take a picture of structures inside the body, especially bones. An x-ray is used to look for a break in the bone.

**Imaging Studies:**
- Routine clavicle radiography
- Fracture is usually demonstrated on an anteroposterior (AP) view.
- Apical lordotic views may be required to define degree of displacement.

**Other Tests:**
Other tests may be required when clinically indicated to assess possibility of life-threatening associated injuries.
- Chest radiography, if pneumothorax suspected
- Angiography, if vascular injury suspected

**Complications of Fractures**
Complications of Fractures are secondary conditions, symptoms, or other disorders that are caused by Fractures. In many cases the distinction between symptoms of fractures and complications of Fractures is unclear.
Complications list for Fractures: The list of complications that have been mentioned in various sources for Fractures includes:

- Bleeding
- Hemorrhage
- Shock
- Death
- Circulatory problems
- Embolism

Underlying Condition Misdiagnosis
An underlying condition is a second condition that you may have in addition to the first diagnosed condition, and this underlying condition is believed to cause the first condition. In this case, the underlying condition is the true primary condition, since it occurred first, and the originally diagnosed condition is actually called the "secondary" condition.

Treatment:

Treatment will depend on the severity of the injury. Treatment involves:

- Putting the pieces of the bone back in position, which may sometimes require anesthesia and more rarely surgery
- Keeping the pieces together while the bone heals itself

Brace or Sling. Most clavicle fractures can be treated with either a figure-of-eight strap, which is wrapped around the body and the shoulders, or with the arm in a sling. These devices help hold the shoulder in place while the clavicle heals. The doctor may prescribe pain medication.

Surgery. Very rarely, surgery may be needed to set the bone. The doctor may insert pins or a plate and screws in the bone to hold it in place while it heals. You will still need to wear the sling or figure-of-eight strap while you heal.

Exercises. When your doctor decides you are ready, start shoulder range-of-motion and strengthening exercises. You may be referred to a physical therapist to assist you with these exercises. Do not return to sports activity until your clavicle is fully healed.

Healing Time
- A child may heal as quickly as 3-4 weeks.
- An adolescent may take 6-8 weeks to heal.
- An adult who has stopped growing may require 8-10 weeks to heal.

Common Injuries and Treatment:

Clavicle fractures. Clavicle fractures are classified by the location of the fracture (middle, distal, or proximal thirds) and further described by the amount of angulation, comminution, and displacement at the fracture site. Most clavicle fractures associated with athletic activities are caused by relatively low-energy impact and have little comminution (figure 4). During clavicle displacement, the proximal segment is drawn superiorly by the sternocleidomastoid muscle, while the distal segment droops inferiorly from the force of gravity and the pull of the pectoralis major. Occasionally, the fracture may be significantly angulated, thereby tenting the skin superiorly, compromising the neurovascular structures inferiorly, or even puncturing a lung.

Nondisplaced or minimally displaced fractures of the medial clavicle or midclavicle can be treated symptomatically with a sling or a figure-of-eight harness for 4 to 6 weeks in young adults and 6 weeks or longer in adults. Andersen et al (7) found no difference in healing, functional, or cosmetic results whether a sling or figure-of-eight harness was used.

Nondisplaced fractures of the distal clavicle are best treated with a sling and early rehabilitation. A standard figure-of-eight harness often lies directly over the distal fracture and thus exacerbates the pain. A modified figure-of-eight harness with increased padding in conjunction with a sling may be effective, but if significant displacement persists, open reduction and internal fixation may be indicated.

For moderately displaced midshaft fractures with no neurovascular compromise and no violation of skin, a figure-of-eight harness serves to reduce the fracture by longitudinal traction.

Young and Rockwood (8) recommend open reduction for any fracture site if complete displacement persists for longer than 3 weeks of figure-of-eight splinting. They further recommend primary open reduction and fixation if the patient has severe skin
tenting, a failed closed reduction, or neurovascular injury. However, non-union of fractures is significantly more common with open than with closed treatment (6).

Athletes should not be allowed to return to play until the fracture is clinically and radio graphically healed. Noncontact and throwing athletes should have a full, painless range of motion and at least 90% strength compared with the uninjured arm. Contact sports should be avoided for 4 to 6 months.

**Acromioclavicular injuries.** AC injuries are classified by the severity of ligament injury. Type 1 injuries consist of a mild sprain of the AC ligament and are treated conservatively with ice, a sling for comfort, early motion, and NSAIDs. Athletes can return to play when they have no pain and a full range of motion (2 to 3 weeks).

Type 2 injuries involve a complete tear of the AC ligament and a mild sprain of the costoclavicular ligament. Radiographic evaluation will show minimal displacement at the AC joint and no separation of the costoclavicular space. Since the AC joint remains stable, treatment is conservative, the same as for type 1 injuries.

Type 3 injuries consist of a complete AC dislocation with complete tears of the costoclavicular and AC ligaments (figure 3b: not shown). Treatment is based on the severity of associated injury. If the distal clavicle is completely displaced superiorly, the deltoid and trapezoid insertions may be avulsed and require surgical repair. If the distal clavicle is dislocated inferiorly below the coracoid, surgical repair is also indicated. Fortunately, these injury types are rare, and most type 3 AC injuries can be treated conservatively with 90% to 100% satisfactory results (9). Return to sports, however, may take as long as 10 to 12 weeks.

**Sternoclavicular injuries.** SC injuries occur once for every five AC injuries reported (10). Like AC injuries, SC injuries can be classified into three types, with type 1 being a mild sprain, type 2 a moderate sprain, and type 3 a complete dislocation. Dislocations are classified as anterior or posterior, depending on whether the medial clavicle is anterior or posterior to the sternum. Anterior dislocations are much more common. Associated injuries must be ruled out.

For type 1 and 2 injuries, treatment is symptomatic and conservative. A sling or figure-of-eight harness can reduce stresses on the SC joint and prevent subluxation. After 7 to 10 days, range-of-motion exercise is initiated (10). Return to sport is allowed when the athlete has no pain and full range of motion and can perform sport-specific movements without limitation.

Type 3 injuries warrant closed reduction. Anterior dislocations can be easily reduced with the patient supine and a rolled towel placed between the scapulae. Traction and abduction on the arm with direct pressure on the clavicle will generally reduce the dislocation. The reduction is often unstable, but an anterior dislocation can be left unreduced after an initial attempt with little functional deficit.

Posterior dislocations are worrisome because they can cause shortness of breath, dysphagia, and pressure on the great vessels. If the patient is skeletally mature, closed reduction should be attempted in the operating room under regional or general anesthesia. Occasionally, conversion to an anterior dislocation is the treatment of choice. If that fails, open reduction may be indicated. In patients who have an open proximal physis (those younger than 22 years), surgery may not be needed because of bony remodeling potential. Following reduction, a figure-of-eight splint is used to allow healing of the ligaments.

**Clavicular osteolysis or degeneration.** Long-term, repetitive, medial forces can lead to chronic changes at the AC or SC joint. The most common changes are degenerative. The athlete will usually complain of pain at the AC joint or distal clavicle but may not remember a specific traumatic event. The pain is usually exacerbated by crossing the upper extremity over the chest, overhead activities, or "locking out" a bench press when the arms are fully extended over the body. Radiographic studies show joint-space narrowing, sclerosis, degenerative cysts, and osteophyte formation. AC involvement is much more common than SC involvement.

Osteolysis is characterized by symptomatic resorption of bone over weeks to many months. A single traumatic event or repeated microtrauma may precipitate the condition (11). It seems to be linked with sports that require repeated shoulder-to-shoulder or shoulder-to-wall impact, such as rugby, hockey, football, ice hockey, racquetball, or handball. Cyclists who have fallen onto the point of the shoulder and have developed osteolysis have also been reported. Cahill (12) noted that most of his cases were associated with weight lifting.

Initial treatment is conservative, consisting of rest, ice, and NSAIDs. Steroid injections are of debatable benefit. Ultimately, after 3 to 4 months of failed conservative treatment, resistant cases may require excision of the proximal or distal clavicle.

**Excellent Prognosis**

Optimal treatment of clavicle injuries stems from accurate diagnosis based on knowledge of the anatomy of the region and the biomechanics of injury. Fortunately, conservative treatment of these injuries usually leads to an excellent prognosis and ultimately a full return to sports.

**Risk Factors:**

A risk factor is something that increases your chance of getting a disease, condition, or injury.

- Advancing age, because of the increased risk of falling
- Osteoporosis
- Certain congenital bone conditions
- Participating in contact sports
- Violence

**Prevention:**

To help prevent clavicle fractures:

- Do not put yourself at risk for trauma to the clavicle bone.
Eat a diet rich in calcium and vitamin D.
Build strong muscles to prevent falls and to stay active and agile.

**SOURCES:**
- "Clavicle Fractures." eMedicine, June 1, 2001.
- American Orthopaedic Society for Sports Medicine
  - http://www.sportsmed.org/
- http://www.gpnotebook.co.uk/simplepage.cfm?ID=328859652&linkID=9743&cook=no
- www.emedx.com

**Case 11:**

A 50-year-old smoker presents to his physician with weight loss and a persistent cough. He is referred to a cardiothoracic surgeon following the discovery of a large mass in the lung field on a chest radiograph. There is no evidence of metastasis and the surgeon recommends removal of the left lung in the hope of improving the patient’s survival. The procedure for the removal of a lung is known as a pneumonectomy. Why is the left bronchus difficult to close during a left pneumonectomy? What structures need to be protected in case of right or left pneumonectomy?

Our patient presents with weight loss and a persistent cough. The physician conducts a thorough physical examination and takes the patient’s history. Patient is then sent to the hospital where further tests are carried out and a definitive diagnosis of lung cancer is obtained. The treatment most suitable is decided to be removal of the left lung.

**Cancer** is by definition any malignant tumour. It arises from abnormal and uncontrolled division of cells that then invade and destroy the surrounding tissues. Spread of cancer may occur via the bloodstream or the lymphatic channels or across body cavities such as the pleural spaces thus setting up secondary tumours. Our patient however shows no signs of metastases. Development of lung cancer is a multistage process. There has to be an acquisition of events that allows for normal cells to escape normal growth regulation and proliferate. There must be up to 8 abnormalities that would have to arrive at a single cell before it can escape normal growth regulation.

In the surgery the doctor notes the patient’s age (the most common age of diagnosis of cancer is 70-74). Also the fact that the patient is a smoker is noted. The patient should be asked that if he has not been making a conscious effort to lose weight are there any other factors in his life that may be causing him to eat less.

It is important that the patient’s cough is examined, also any sputum that may be being produced, the colour of it and also the quantity of sputum being produced.

The fact that our patient is a smoker is of huge significance and cannot be ignored. Smoking accounts for 80-90% of all pulmonary malignancies. Cigarette smoke
is a carcinogen. A **carcinogen** by definition is any substance that when exposed to living tissue may cause the production of cancer. They cause damage to the DNA of the cells that may persist if the cell divides before the damage is repaired. Other examples of carcinogens are asbestos and radon. Radon is a significant one to note as it is believed to account for approx 200 deaths in Ireland annually due to cancer. Galway is a high risk county for radon levels.

The risk of developing lung cancer due to smoking is duration dependent and cumulative. The more you smoke the higher your risk of developing lung cancer, and the longer you smoke for the higher your risk for developing lung cancer. This risk then interacts with all other risk factors. If you are male and smoke 1 pack of cigarettes daily your risk of developing lung cancer is 22 times that of the normal population. If you are female and smoke 1 packet of cigarettes daily your risk is 12 times that of the normal population.

It is interesting to note that it is almost impossible to obtain a clear control group for these statistics as everyone has been exposed to second hand smoke. One statistic that has been calculated however is that if you are a non-smoker married to a smoker your risk developing lung cancer increases by 33%.

The question then must arise how common in lung cancer? Cancer in all of its forms accounts for ¼ of all deaths in Ireland. It is the largest single cause of death for the Irish population. Lung cancer is the most common cancer killer among Irish males and is the 2nd most common cancer killer among Irish females after breast cancer. Lung cancer has a lower incidence than other cancers but significantly has a higher mortality. Just 8.1% of Irish men diagnosed with lung cancer are alive 5 years after diagnosis. To put that statistic more simply and to include females, 4 out of 5 people die within one year of diagnosis and 1 out of every 20 is alive 5 years after diagnosis.

Another question that arises is that of how lung has this lung mass been in our patients lung? Lung cancer can be divided into two main divisions – SCLC, small cell lung cancer, and NSCLC, non-small cell lung cancer. NSCLC may then be further divided into squamous cell carcinoma, adenocarcinoma, and large cell lung cancer.

SCLC grows very rapidly, and is very likely to spread to other organs. A patient can die within 2-4 months after development if this form of cancer is not treated. Chemotherapy is the usual treatment.

NSCLC is a very silent disease and it is one of these forms of cancer that out patient has. This type of cancer grows very slowly and can go unnoticed for years. Just to give some statistics from the Philadelphia Lung Project – for lung cancer to be clinically detectable on a chest x-ray it must be 1cm in diameter. At 1cm you have approx 30 volume doubles. Knowing that the doubling time for lung cancer is between 3-6 months that means that this cancer when it becomes visible has to have been there for between 8-15 years.

### DIFFERENTIAL DIAGNOSIS;

Weight loss  
Persistent coughing  
Large lung mass

### POSSIBLE DISEASES

1/Alveolar Hydatid Disease (AHD)

### TESTS

X-ray  
M.R.I.  
Antibody blood test

2/Lung Sarcoidosis (Boek's disease)
The presence of a parasitic cyst in the lung. It can be quickly ruled out by assessing the patient's environmental surroundings i.e. Occupation (veterinarian), tourist contact with foreign animals, foreign uncooked meat e.g. fish.

Weller and co-workers (1987), discussed a case of echinoccosis in a 16-year-old student exposed to the disease during a year spent in Bolivia. He developed both hepatic and pulmonary cysts and whose diagnosis was based on travel history and physical exam, x-ray, ultrasound and CT findings.

Lung Sarcoidosis  (Boeck's Disease)
The cause of this disorder is unknown. It leads to the lymph nodes in many parts of the body including the lungs which become enlarged. Small fleshy nodules develop in the lungs.

It has features similar to T.B. Recovery is complete with minimal after effects in two-thirds of cases. In Boecks disease, statistics state that it occurs in young adults in the age-group of 20-40. It rarely occurs in children under ten years or in adults over 60 years. Another point is that anyone can get it. It occurs in all races and in both sexes.

The greater risk factor occurs in black adults, and in those of Scandinavian, German, Irish and Puerto Rican origin.

Preliminary diagnosis of sarcoidosis is based on the patient's medical history, routine tests, physical exam and chest x-ray. No single test can be relied on for correct diagnosis. In most cases, however, x-rays and blood tests are the first procedures that the physician requests.

Tuberculosis
T.B. is a chronic bacterial infection, which causes nodule formation in the lungs. It causes more deaths worldwide than any other infectious disease. T.B. is spread through the air. One-third of the world is infected with T.B. The infecting bacteria are mycobacterium tuberculosis/ bovine.

T.B. has a range of definitive symptoms that differentiate it from the other diseases thus far. E.g. Pyrexia, chest pain, shortness of breath, fatigue, weight loss, persistent coughing.

The most common method and one that is usually very accurate is the T.B. skin test or mantoux test. It can identify most people infected with tubercle? six to eight weeks after exposure. A substance called Purified Protein Derivative (PPD) under the skin of the forearm and examined 48-72 hours later. A red welts forms at the site of injection. The person may have beeninjected with M. Tuberculosis but does not necessarily have the active disease.

Lung Cancer
Aoibheann has already explained the types and causes for lung cancer. I'm now going to discuss in detail the types of tests used and how we arrived at our definitive diagnosis for lung cancer.

To help find the cause of symptoms, we have in this case evaluated all the factors Aoibheann has said. The physician must now perform a physical exam, may order an x-ray, CT, MRI. Lung cancer has been suspected. Sputum cytology was used which is 20% diagnostically effective in patients.

To confirm lung cancer, one must examine tissue from the lung. A biopsy removal produces a small tissue sample for pathological examination. This is, and can be done with a number of other procedures. E.g. Bronchoscopy, needle aspiration, thonalentesis (removes fluid from around the lung).Thoracotomy-open chest-major operation.

Additional blood tests; PTH (Parathyroid Hormone), CEA (Carcinogen Antigen).

Surgery

When the man is confirmed to be of sufficient lung function and overall health for a pneumonectomy, it can be recommended to him. It is essential to be very informative, for he may well feel complications occurring before the surgeon does and in so can help too.

At 50 years of age, the man may not be entirely intrigued by the idea of changing his lifestyle when his prognosis is at best 'guarded.' However with a tumour in his left lung, ignorance is far from bliss.

Essential points of information include the implications of not having surgery. The tumour can only grow and if cancer cells get into the circulatory or lymphatic drainage, death will soon follow! The involvement of the mediastinal lymph chain is associated with poor prognosis.

The proportion of blood volume passing through the lungs is approximately 300% as it seeks oxygenation. Cancer cells that penetrate this system will easily metastasize in the body to other sites where tumours will subsequently develop. The supraclavicular lymph gland and the brain are prime targets, as is the recurrent laryngeal nerve, penetration of which will paralyze the vocal fold as the recurrent laryngeal nerve supplies all but 1 of the laryngeal muscles.

The entire tumour has to be removed from his lung in the hope of improving his chance of survival. It is essential both to remove the entire tumour to minimize risk of metastasis, and to maintain all the healthy tissue, as the guy’s breathing capacity will be halved therefore the function of the right lung must be optimal. Thus, mediastinal structures such as the great vessels and the heart, as well as the trachea and all structures pertaining to the function of the right lung, must be protected during surgery.

Pneumonectomy is not the least invasive form of lung volume reduction surgery but it is the most suitable for this case, as the tumour is obviously not just restricted to one lobe or bronchopulmonary segment, in which cases lobectomy and wedge resection would be performed, respectively. Furthermore the tumour probably involves a significant portion of the pulmonary artery and veins, therefore the entire lung must be eradicated so to speak. A pneumonectomy could
also be required in emergency cases of a severe chest wound, where irreparable damage has been done to major blood vessels or the main bronchus.

The standard way of approaching the lungs and major bronchi is through a posterolateral thoracotomy, whereby the patient has to lie on the unaffected side in the lateral position. The patient will be under a general anaesthetic for the operation. A double lumen tube helps to ventilate the unaffected lung and compress the diseased lung, while protecting it from pus and blood.

Incision is usually near the inframammary fold in women, and the nipple in men. It begins below the tip of the scapula and extends posteriorly and superiorly between the medial border of the scapula and spine, where it deepens to the level of the latissimus dorsi, which is then divided with coagulating diathermy. A periosteal elevatior is used to lift the periosteum off the superior border of the rib. This reveals the pleura which may be entered by blunt dissection. A rib spreader is inserted between the ribs and opened gently to prevent fracture. Sometimes a portion of the rib is removed to provide better access to the thoracic cavity. (Interestingly, this portion of the rib could be used again perhaps, for example, in autogenic bone grafting of the mandible following removal of a tumour). The anaesthetist is now able to deflate the affected lung to allow a better view of the intrathoracic structures. Elastic in nature, the lung recoils to a third its size when the thoracic cavity is opened, making it much easier to access and remove.

Vascular structures are identified and ligated i.e. the main pulmonary artery and pulmonary veins on the left side of the mediastinum. The left bronchus is clamped and cut as close to the trachea as possible. The position of the heart imprinting the cardiac impression of the left lung is significant in the arch of the aorta - a continuation of ascending aorta, arches posteriorly on the left side of trachea and esophagus and superior to left main bronchus. This makes the left bronchus difficult to close after surgery. A cut is made at the hilum, the lung is clamped and removed.

Video Assisted Thoracoscopic Surgery is less invasive, using smaller incisions and precise technology, and the hospital stay is much shorter and less complications arise, but the incision needs to be bigger in order to access the lung etc. (apparently)

Other bronchial complications must be avoided, such as bronchopleural fistula, for example, which is an abnormal connection of the bronchial stump with the pleural space. After Pneumonectomy, the space is filled with air, which is gradually displaced by tissue fluid. The fluid level then rises, but a bronchopleural fistula will cause an infection of this tissue fluid and its subsequent effusion. This is a potentially lethal condition and may provoke other diseases e.g. empyema.

Intercostal drains are usually inserted at the end of the procedure. Haemostasis is actually very important due to the large space left after pneumonectomy. Eventually, the mediastinum, in this case will shift towards this space and right lung hypertrophy will occur with the proliferation of alveolar type 11 cells to increase the number of gaseous exchange units to try and compensate for the reduction in lung volume, therefore breathing capacity. Chest radiographs from now on will appear lob-sided due to this mediastinal shift.

Sometimes the mediastinal shift is acute, provoking the rare post pneumonectomy syndrome whereby as the mediastinum shifts so suddenly, the heart and great vessels undergo clockwise rotation, which may well impair venous return, impinging on the contractility of the heart e.g. cardiac arrhythmia my result.

Paraplegia i.e. intercostals arterial bleeding at the costovertebral junction may also occur, but there is a 0.08% risk of this.
When it comes to it, it’s his choice. The five year survival rate is at best 14%, but he is a smoker, therefore basal collapse and hypoxaemia may well ruin the lung function of the remaining lung and worsen the prognosis. It is significant that a mass in the lung field needs to exceed 1cm diameter to be detected in the chest radiograph. By this stage it may well have spread to other areas in the body, they simply aren’t detectable yet as these metastases are microscopic. The project ACOSOG Z0040 ongoing in the American college of surgeons in southern California, attempts to understand the significance of such ‘occult microscopic metastases’ and how to detect them early. The fact is, it is pointless to remove the lung tumour if other cancer cells are just waiting to grow elsewhere. Immunohistochemical lymph node and rib bone marrow studies are therefore ongoing to try to understand such complexities of the big killer itself.

Would it simply cost the government too much to fund the development of screening for cancer?

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Grand Clinical Rounds: Case 13

Anatomy of Pelvis

The pelvis is interposed between the movable vertebrae of the vertebral column it supports and the lower limbs upon which it rests. The pelvic cavity, the space bounded by the bones of the pelvis, contains the organs of the reproductive system, caudal terminations of the alimentary and urinary systems, blood vessels, lymphatics and nerves.

The superior boundary of the pelvic cavity is the pelvic inlet. It is bounded by the pelvic brim. The inferior boundary is the pelvic outlet which is closed by the musclofascial pelvic diaphragm and bounded anteriorly by the pubic symphysis and posteriorly by the coccyx.

The main function of the pelvic girdle is to support and protect the contained viscera. Its most important mechanical function, however, is to transmit the weight of the trunk and upper limbs to the lower extremities.

The pelvis is composed of four bones: two hip bones, each of which is made up three bones – ilium, ischium and pubis – which fuse together during adolescence. The sacrum – formed by the fusion of five sacral vertebrae. The coccyx – formed by the fusion of four coccygeal vertebrae. The hip bones join anteriorly at the pubic symphysis and to the sacrum posteriorly at the sacroiliac joints.

The ilium is the uppermost and widest of the three bones comprising the hip bone. It consists of a flattened plate with a long curved ridge, the iliac crest, along its border. The ala or wing of the ilium represents the spread of the fan and the body of the ilium represents the handle of the fan. It also forms the superior part of the acetabulum – the cup-like depression on the surface of the hip bone with which the head of the femur articulates.
The ischium is the dorsal and posterior of the three principal bones comprising either half of the pelvis. It consists of a body and ramus. The body forms the posterior part of the acetabulum and the ramus forms part of the inferior boundary of the obturator foramen.

The pubis is the smallest pelvic bone. It is the ventral and anterior of the three major bones comprising either half of the pelvis. It consists of two rami diverging posteriorly from the region of the pubic symphysis. The superior ramus extends to the acetabulum of which it forms the anterior part. It unites there with the ilium and ischium. The inferior ramus extends below the obturator foramen where it unites with the ischium.

The pelvis is divided into the greater (or false) and lesser (or true) pelvis. They are separated by using the pelvic brim as the limiting line. The pelvic brim extends from the promontory of the sacrum, arcuate line of the ilium, pectinal line and pubic crest. The greater pelvis is located superior to the pelvic brim and the lesser pelvis inferior to the brim. The greater pelvis is bounded by the abdominal wall anteriorly, the iliac ala laterally and the L5 and S1 vertebrae posteriorly. Abdominal viscera such as the sigmoid colon and some loops of ileum are located in the greater pelvis. The lesser pelvis is located between the pelvic inlet and the muscular pelvic diaphragm. It contains the pelvic viscera.

**Sexual Differences in Pelves**

The sexual differences are linked to function. The female pelvis is distinguished from that of the male by its bones being more delicate and its depth less. The pelvic inlet of the lesser pelvis is larger in the female and is more circular. The size of the female lesser pelvis is important because it is through the bony pelvic canal the foetus passes during birth. The female lesser pelvis has adapted for childbirth.
**Uterus**

The uterus is a thick-walled, pear-shaped, hollow muscular organ. It is located in the lesser pelvis normally with its body lying over the urinary bladder and its cervix between the bladder and rectum.

The uterus is a mid-line organ and is held to the lateral walls of the true pelvis by a double layer of peritoneum, called the broad ligament. It contains the ovaries, uterine tubes and related structures.

The uterus is divided into two parts: the Body, which has two parts- fundus and isthmus, the cervix, cylindrical narrow inferior part of uterus that projects into the vagina.

The wall of the uterus consists of three layers: Perimetrium – outer serous layer, Myometrium – muscular layer and Endometrium – inner mucous membrane.

The non-gravid uterus is intrapelvic, but it loses this protection as it expands out of the pelvis during pregnancy. The uterus increases in weight from 60g to 1000g by the end of pregnancy. At about the 36th week of pregnancy the foetus settles into a head down, or cephalic position. At this time the foetus may soon “engage” or settle into the pelvis. This relieves pressure on the upper abdomen and places more pressure on the bladder.

**Sheath of Rectus Abdominis Muscle**

The Pfannenstiel incision involves the external sheath of the rectus abdominis muscle. The sheath is formed by the aponeurosis of the external oblique, internal oblique and transversus abdominis muscles. At the lateral border of the rectus muscle this sheath divides into an anterior layer which passes ventral to the muscle an a posterior layer which passes dorsal to the muscle. At a point midway between the umbilicus and pubis the posterior layer of the internal oblique aponeurosis and the aponeurosis of the transversus abdominis pass ventral instead of dorsal to the rectus muscle. The line formed dorsal to the rectus abdominis in this region is called the arcuate line. The rectus sheath can be divided into three parts each with a different construction:

1. Above the costal margin
2. Costal margin – Arcuate line
3. Arcuate line - Pubis

It is the third part that the Pfannenstiel incision is concerned with.

The contents of the Rectus Sheath include: the Rectus Abdominis and Pyramid muscles, Superior and Inferior epigastric vessels and the termination of the lower five intercostal nerves (7-11), the 12th (subcostal) thoracic nerve and accompanying vessels.

**Pelvic Fracture**

Pelvic fracture is a disruption of the bony structure of the pelvis. This generally requires substantial force such as that from a motor vehicle accident. Because of the force involved, pelvic fractures frequently involve injury to organs contained within the bony pelvis. Pelvic fractures range widely in severity. In addition, as the pelvis is supplied with a rich venous plexus as well as major arteries, fractures may produce significant bleeding. Pelvic fractures are classified as stable or unstable and as open or closed. A stable fracture is one in which the pelvis remains stable and involves one break-point in the pelvic ring with minimal haemorrhage. An unstable fracture is one in which the pelvis is unstable with two or more break-points in the pelvic ring with moderate to severe haemorrhage. All types of pelvic fractures are further divided into “open” or “closed” depending on whether open skin wounds are present or not in the lower abdomen.

In the case of our patient it is deemed that a vaginal labour would be hazardous to the health of both mother and child. This could be due to the weakening of the pelvic bone and the pressure that a
normal labour would exert, or possibly due to a disruption to the birth canal as a result of the pelvic fracture. The risk of placental abruption and uterine rupture is also great.

**Surgical incisions of the anterior abdominal wall**

Various incisions are used by surgeons to gain access to the abdominal cavity. The incision chosen is based on a number of factors. The incision that allows the adequate exposure of the concerned area and also the incision that achieves the best cosmetic effect is normally chosen. The location of the incision is dependant on the type of operation being performed, the location of the organ, bony or cartilaginous boundaries, maintenance of a normal blood supply and prevention of injury to surrounding muscles, fascia and nerves. In this type of incision, muscles are not transected as this would lead to the death of muscle fibres, the surgeon splits them between their fibres. The muscles and viscera are retracted towards their neurovascular supply. The accidental cutting of a motor nerve would paralyse the muscle fibres it innervates, this would therefore weaken the anterior abdominal wall. However, one or two small branches of nerves may be cut without a noticeable loss of motor supply to the muscles or loss of sensation of the skin. This is due to the overlapping areas of innervation between nerves in the abdominal wall. The most common abdominal incisions include the median or mid-line incision, left paramedian incision (both used for exploratory operations), gridiron (muscle-splitting) incision, transverse (abdominal) incision (both can be used for appendectomy), pfannenstiel (suprapubic) incision (for caesarean section, hysterectomy) and finally the subcostal incision (used for gallbladder removal).

![Diagram of abdominal incisions](images.google.ie)

**Case 13**

**Treatment**

The pelvic ring fracture can be classified into:

**1-Stable fractures:**

The stable fractures is when the pelvic ring is undisturbed, where the fractures do not enter the pelvic ring. An example of that would be a fracture to the iliac wing, the ischiopubic ramus and the floor of the acetabulum.

Complications of the satble fractures can be minor bleeding and pain. The treatment is bed rest and after 2-3 weeks the patient can walk with the aid of crutches.

**2-Unstable fractures:**

The pelvic fracture is when the pelvic ring is disturbed and the fractures do enter the
pelvic ring. An example of the would be An anteroposterior compression, lateral compression (open book) and vertical shear.

Complications that may occur with unstable fractures are major bleeding, injuries to the bladder and sciatic nerve injuries.

**Anterioposterior compression :**

This is where all 4 pubic rami are fractured and the main complication is damage to the bladder. In this case reduction is neither possible nor necessary. The treatment would be rest on the bed for 3-4 weeks.

**Lateral compression :**

In this case the pelvis opens up like a book, usually at the pubic symphysis and the main complication is bleeding. Her reduction is possible but if it fails a metal fixation can be used. There are two types of metal fixation that can be used, external, internal, or both.

**External fixation**

External fixation can be done using the Ganz fixator. When the patient is lying on his back in the supine position a Steinmann pin is inserted on each side of the pelvic girdle. Then the pins are driven 1cm into the bone with a hammer.

The pins are attached to 2 vertical side arms which are then attached to horizontal long arm. Then the bolts are driven inwards with a wrench which will close the gap and stabilize the pelvic ring.

The patient must stay in bed for 4 weeks where the device is removed. He can then walk with crutches and regains full activity within 12 weeks.

**Internal fixation :**

Internal fixation is done by a pfannenstiel incision down to where the pubic symphysis is visible. The gap is reduced by ilium-ilium compression whilst observing the symphysis. A plate is then applied on the superior surface of the pubic bones crossing the pubic symphysis. Two 6.5 mm screws are then applied directed posteroinferiorly.

Rehabilitation starts six weeks after the operation and it includes weight bearing and strengthening.
Cesarean section:

- The umbilical cord is clamped and cut. The placenta will then be removed and the uterus stitched up and put back into place. The closing of the abdominal wall will begin at this time with the muscles over the peritoneum pushed in place and sewn. Then the closure of the incision by staples into the skin.

- There should be no sign of the scar within 3 months of the operation.

Complications:

- Some complications that can occur during or after a c-section include heavy bleeding, major infections of the uterus, opening up of the skin incision, blood clots around the uterus, an inability of the blood to clot, and damage to the uterus that makes future childbirth more dangerous.
CASE 14

The thyroid gland is the largest endocrine gland in the body, weighing on average 25g. This brownish-red, vascular gland is found in the visceral compartment of the neck. The cervical viscera are organised in three layers. From deep to superficial these are; the endocrine layer, containing the thyroid and parathyroid glands, the respiratory layer containing the pharynx, larynx and trachea and the alimentary layer in which the pharynx and oesophagus are located.

The visceral compartment of the neck is surrounded by the pretracheal layer of deep cervical fascia. This layer of fascia extends inferiorly from the hyoid bone into the thorax where it blends with the pericardium of the heart. It includes a thin muscular layer which encloses the infrahoid muscles and a visceral layer which envelops the viscera of the anterior triangle of the neck, including the thyroid gland.

The bilobed thyroid gland is found in the anterior triangle of the neck, where it lies deep to the sternothyroid, the sternohyoid and the omohyoid and just anterior to the larynx and trachea at the level of C5 through T1.

The lateral lobes of the thyroid gland are conical in shape. Their ascending apices extend superiority and laterally to the level of the oblique lines on the laminae of the thyroid cartilage of the larynx. The bases of the lobes are level with the fourth and fifth tracheal cartilages. Each lobe is
approximately 5cm long. Each lobe appears approximately triangular in cross-section, with lateral, medial and posterior surfaces. The lateral (superficial) surface is related to the sternothyroid and sternohyoid. The medial surface lies against the lateral side of the larynx and upper trachea, with the lower pharynx and upper oesophagus immediately behind. The external and recurrent laryngeal nerves approach the gland from above and below respectively. The posterior part of the thyroid gland overlaps the medial part of the carotid sheath which contains the external carotid artery. The parathyroid glands lie in contact with this surface. These are small, yellow-brown oval structures which produce parathormone.

The thin glandular isthmus connects the lobes lower parts. It measures about 1.25 cm transversely and vertically. The posterior surface of the isthmus is firmly adherent to the second and third tracheal rings and the pretracheal fascia is fixed between them. A conical pyramidal lobe often ascends towards the hyoid bone from the isthmus. A fibromuscular band, the levator of the thyroid gland, sometimes descends from the hyoid to the isthmus or pyramidal lobe.

The thyroid gland has a thin capsule of connective tissue whose extensions divide it into masses of irregular form and size. The parenchyma derives mainly from the endoderm of the thyroglossal duct. The thyroid gland contains two cell types. The first and most abundant are the Follicular cells. When activated by TSH or sympathetic stimulation, these cells secrete thyroxin. The second cell type, which accounts for less than 3%, are the Parafollicular or C cells. These cells secrete thyrocalcitonin which regulates calcium metabolism.

Two major arteries supply the thyroid gland. These are the superior and inferior thyroid arteries. The superior thyroid artery is the first branch of the external carotid artery. Accompanied by the external laryngeal nerve, it pierces the pretracheal fascia, and descends to the superior pole of the lateral lobe of the gland. Here it divides into anterior and posterior glandular branches. The anterior branch supplies the superior border of the gland, while the posterior branch passes to the posterior side of the gland. The inferior thyroid artery is a branch of the thyrocervical trunk and is accompanied by the recurrent laryngeal nerve. It ascends to the inferior pole of the lobe of the gland. Here, the inferior thyroid artery divides into an inferior branch which supplies the lower part of the gland and anastomoses with the posterior branch of the superior thyroid artery and the ascending branch which supplies the parathyroid glands.

In about 10% of people, a thyroid ima artery springs from the brachiocephalic trunk of from the aortic arch to supply the thyroid gland. Three pairs of veins drain the thyroid venous plexus. These are the superior and middle thyroid veins which drain into the internal jugular vein and the inferior thyroid vein which drains into the brachiocephalic veins.

The lymphatics follow the arteries. From the upper pole they enter the anterosuperior group of deep cervical lymph nodes. From the lower lobes, they pass with the inferior thyroid artery into the posterioinferior group. A few pass downwards into the pretracheal nodes, following the course of the
thyroid ima artery. The bulk of the sympathetic supply of the thyroid gland is derived from the middle cervical ganglion and enters the gland on the inferior thyroid artery.

Carcinoma of the thyroid gland is a malignancy of the thyroid gland. The large majority of Thyroid cancers are primary. Thyroid carcinoma may be caused by exposure to ionizing radiation in childhood. Some instances of Papillary & Medullary carcinomas are associated with a family history. Metastatic carcinoma of the thyroid is very rare.

The different types of thyroid carcinoma are Papillary(60%) & Follicular carcinomas(20%) which are termed the well differentiated carcinomas, Anaplastic carcinoma(5-8%), Medullary carcinoma(5%) & Lymphoma(5%). Papillary carcinoma is the most common type and can occur at any age the peak incidence is in the third & fourth decades. It is usually slow growing & may metastasize early to regional lymph nodes. The tumour is dependant on thyroid stimulating hormone. Follicular carcinoma has a peak incidence in the 40 – 60 age groups. This disease occasionally spreads with bony secondaries. Anaplastic carcinoma occurs in old age and is usually very rapidly growing. Medullar carcinoma may present at any age, it usually presents with a solitary nodule & may spread to the cervical nodes. This type of carcinoma may be familial, therefore identification of ret-proto-oncogene on ch10 is necessary and when present family screening is indicated. Lymphoma is usually only diagnosed after a thyroidectomy. In our case the cancer is most likely to be either Papillary carcinoma or Medullary carcinoma and considering there is a much higher % of papillary carcinoma it is more likely to be this type.

In this particular case the patient consulted her physician complaining of a swollen neck which had been increasing in size and also some breathlessness. The Swelling was found to be a carcinoma of the thyroid gland and was situated on the right side of the larynx and trachea. The breathlessness occurred as the tumour was located anterior to the trachea, the abnormal mass was pushing on the trachea and partially occluding its lumen. Other symptoms and signs that may indicate thyroid carcinoma include deep cervical lymph nodes being enlarged and the patient may experience local discomfort in the neck, Bone pain may represent secondary disease & in some cases may be the presenting factor, a cough may present if there are lung metastases. It is important to note that patients with carcinoma of the thyroid are usually euthyroid (i.e. Have a normal functioning thyroid gland).

On examination firstly the physician must take a history from the patient, the main aim of this is to determine if there is any past history of thyroid cancer or radiation. Then the physician watches the neck during swallowing water to see if the tumour will move. In our case the tumour moved upwards with swallowing, this is because the thyroid gland is invested in a sheath derived from pretracheal fascia which holds the gland onto the larynx and trachea & the thyroid follows the movements of the larynx during swallowing. Any pathological swelling of the thyroid will therefore move upwards distinguishing it from a swelling in some other part of the neck. The physician should then examine the thyroid from behind feeling it for size, shape, tenderness & mobility. IF a nodule greater than 4cm across is found on
the gland it is more likely to be malignant. The physician will then do an ultrasound on the lump to see if it is solid/cystic or part of a group of lumps. A fine needle aspiration should then be carried out and cytology done on the fluid. If a malignant tumour is suspected the deep cervical lymph nodes should be examined for metastases as the thyroid is primarily drained by these.

The treatment of a carcinoma of the thyroid gland depends on the type. For well differentiated carcinomas total thyroidectomy & excision of all involved nodes is advised. Iodine (123) diagnostic scanning can be done to check for secondary disease. Lifelong thyroxine treatment is required as (a) replacement therapy and (b) to achieve TSH suppression. Prognosis can be good in this case but 9% will die & 30% will need treatment for secondary disease. In anaplastic carcinoma surgery is rarely indicated & prognosis is poor. For madullary carcinoma a total thyroidectomy with removal of all affected lymph nodes is advised. Inoperable tumours should be irradiated. Prognosis without metastases is excellent and cure may be expected, but with metastases less than 50% live 10yrs. In the case of lymphoma treatment depends on type and is a matter for a specialist.

Before undergoing a thyroidectomy the patient must first undergo the following preoperative preparation: The doctor must ensure the patient is euthyroid. A warning should be given regarding Hypocalcaemia (i.e. A serum conc of ca++ below the normal range) this is seen in 30% of cases in total thyroidectomies with 2% still requiring replacement therapy @ 3mths postoperatively. The patient should also be warned of nerve damage as permanent injury occurs in 1-2% of operations. Complications that can arise during & after a thyroidectomy include: Haemorrhage, Laryngeal oedema, Nerve damage, Hypocalcaemia, wound infection (rare) and Hypothyroidism.

Postoperatively, the surgeon carefully ensured that the patient was speaking properly. This is because the damage to the external laryngeal nerve can affect cricothyroid muscle which results in the inability to tense the vocal folds producing weakness of voice. Besides, the bilateral damage to recurrent laryngeal nerves may cause to lose speech completely (aphonia) and encounter difficulty in breathing (dyspnea).

Considering the differential diagnosis of the swollen neck, medical history and physical examination can help to make primary diagnosis. The can then be confirmed via laboratory investigation. There are a few differential diagnosis to be considered in the case of swollen neck. Firstly, regarding inflammatory and infectious conditions, the swellings will usually resolve within 2 to 4 weeks after being treated with antibiotics. The mess arises from acute inflammation would be soft tender and mobile while the one due to chronic inflammation would be non-tender and rubbery.

There are also some swellings related to congenital anomalies. Branchial anomalies such as branchial cyst and branchial sinuses are the most common congenital masses in the lateral neck. Usually, branchial cyst is palpable inferiorly to the angle of mandible while brachial sinuses along the anterior border of sternocleidomastoid. Meanwhile, thyroglossal cyst duct in the central portion of neck,
near the hyoid bone can be examined by asking the patient to protrude his tongue. A definite superior tug on the mass is noted and the swelling

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Case 15

Anatomical Relavance - Inguinal canal

The inguinal region in the inferior part of the anterolateral abdominal wall includes an area of weakness, especially in males, because of the passage of the spermatic cord through the inguinal canal.

Firstly, the inguinal canal in adults is an oblique, inferomedially directed passage through the inferior part of the anterior abdominal wall. It lies parallel and just superior to the medial half of the inguinal ligament. The main occupant of the inguinal canal is the spermatic cord in males and the round ligament of the uterus in females. Also contains the blood and lymphatic vessels and the ilioinguinal nerve in both sexes.

For descriptive purposes and to be able to imagine the inguinal canal, it can be thought as a tunnel or a tube that travels from an 'entrance', which is lateral and deep to an 'exit', which is medial and superficial. The so called entrance of the inguinal canal is formed by the deep (internal) ring. It is an outpouching of the transversalis fascia approximately 1.25 cm superior to the middle of the inguinal ligament and lateral to the inferior epigastric vessels. On other hand, the exit is formed by the superficial (external) ring. It is a slitlike opening between the diagonal fibers of the aponeurosis of the external oblique, superolateral to pubic tubercle. The lateral and medial margins of the superficial ring formed by the split in the aponeurosis are the lateral and medial crura. It is important to put in mind that both the entrance and the exit of the inguinal canal do not overlap each other because of the oblique path of the canal through the aponeurosis of the abdominal muscles.

Besides of having an entrance and exit, the inguinal canal also has two walls (anterior and posterior), a roof and a floor. The anterior wall is formed by the external oblique aponeurosis throughout
the length of the canal, with the lateral part of this wall being reinforced by the fibers of internal oblique. The posterior wall on the opposite side is formed by the transversalis fascia with the medial part of it being reinforced by the merging of the pubic attachments of the internal oblique and transverse abdominal aponeurosis into a common tendon - the conjoint tendon. The roof of the inguinal canal, however, is formed by the arching fibers of the internal oblique and transverse abdominal muscles. Meanwhile, the floor of the canal is formed just by the superior surface of inguinal ligament.

Instead of just knowing the anatomy of the inguinal canal, it is also important to understand any action that would effect the canal. Increases in intra-abdominal pressure will force the posterior wall of the canal against the anterior wall, closing the passageway and thus strengthening any potential defect of the abdominal wall. Contraction of the external oblique also approximates the anterior wall to the posterior wall. Furthermore, the internal oblique and transverse abdominal muscles makes the roof of the canal descends, which constricts the canal. All these events occur simultaneously during acts such as sneezing, coughing, and 'bearing down' (vasalva maneuver).

Another important thing to remember is the innervation of the inguinal region, because injury to the nerves in this area may cause significant problems. The major nerves in the inguinal region are the ilioinguinal, iliohypogastric and genitofemoral nerves. The ilioinguinal nerve traverses the inguinal canal near the external inguinal ring and provides unilateral sensory innervations to the pubic region and the upper portion of the scrotum or the labia majora. This is the nerve most commonly injured during open herniorrhaphy. The iliohypogastric nerve passes superior to the internal inguinal ring and provides sensory innervations to the skin superior to the pubis. The genital branch of the genitofemoral nerve travels within the spermatic cord to provide sensation to the scrotum and the medial thigh. The femoral branch of this nerve supplies sensation to the skin of the anterior thigh.

Related question

What are the boundaries of Hasselbach's (inguinal) triangle?
Those boundaries are inferior epigastric artery laterally, rectus abdominis muscle medially, and the inguinal ligament inferiorly.

What body layers would surround it as it proceeded into the scrotum and what abdominal layers are they derived from?
The body layers would be the skin, dartos muscle, membranous layer of the superficial fascia, external spermatic fascia which derived from external oblique aponeurosis, the cremasteric fascia which derived from internal oblique aponeurosis, and the internal spermatic fascia which derived from transversalis fascia.

EMBRIIOLOGICAL CORRELATION
At 35 years of age, S. T. may not realise it, but the main cause of his hernia occurred when he was developing in the womb. Indirect inguinal hernias are also widely known as congenital hernias, and S.T. was always more likely than others to get an indirect inguinal hernia. However it should be noted that not all indirect inguinal hernias are considered to be congenital (less than 10%). For this case, we will presume it was congenital.
S. T.’s problem would have started when the Processus Vaginalis failed to obliterate. The Processus Vaginalis is a peritoneal diverticulum which is essential in the development of the inguinal canal. It is formed before the descent of the gonads in the third trimester. This diverticulum would have pushed its way through the lower part of the anterior abdominal wall, in an effort to make
way for the descent of S. T.’s ipsilateral testicle. The Processus Vaginalis invaginates the transversalis fascia, and then passes underneath the curved fibres of the transversis abdominus muscle. After this it has one layer of covering, in S. T.’s case the internal spermatic fascia, which came from the transversalis fascia. Now it pierces the lower fibres of the internal oblique muscle, and takes them with it. These form the S. T.’s cremaster muscle. Finally the Processus invaginates the aponeurosis of the external oblique muscle, forming S. T.’s superficial inguinal ring (which is important for clinical examination of the hernia). This invaginated aponeurosis forms the external spermatic fascia. The Processus Vaginalis now has three concentric layers of tissue covering it, which will also cover S. T.’s spermatic cord. These layers are important for the surgical treatment of indirect inguinal hernia.

In males, after the Processus Vaginalis has made its way to the scrotum the testes descend from the level of L1 into the scrotum during the 7th and 8th months of gestation. To do this they follow alongside the Processus Vaginalis. Not inside it, as the gonads are retroperitoneal. The deep and superficial inguinal rings have to distend to allow passage of the testes through them. This helps to explain the 9:1 ratio of indirect inguinal hernias in men:women. The inguinal canal is also more patent in S. T.’s case, than it would be in a women’s, because the spermatic cord which passes through it is larger than the round ligament of the uterus which would pass through it in women.

Once S. T.’s testes reached their destination, the Processus Vaginalis was supposed to obliterate, and leave a small pouch - the Tunica Vaginalis - to protect and envelope the testicle on that side. However we are presuming that in S. T.’s case this didn’t happen properly, and hence he had a patent Processus Vaginalis. This affords omentum and abdominal viscerae the opportunity to herniate into the inguinal canal, and in some cases, depending how much of the Processus Vaginalis has failed to obliterate, into the scrotum.

PHYSIOLOGICAL FEATURES

With Indirect Inguinal Hernias the big physiological factor is intra-abdominal pressure. In this case the increase in intra-abdominal pressure which triggered the hernia was lifting a heavy coffee table. This would have caused S. T.’s abdominal wall muscles to contract vigorously, in an effort to keep his trunk stable. Rectus Abdominus would have played a large role in increasing the intra-abdominal pressure. It is also likely that he held in his breath, meaning that the diaphragm would have been pushing down on the abdominal cavity, thus increasing pressure (Valsalva Manoeuvre). This increased pressure results in the contents of the abdomen pushing out against the internal wall of the abdomen, looking for more space. In S. T.’s case the Deep Inguinal Ring was a weakening in the wall, and probably with a patent Processus Vaginalis creating a passageway for tissue out of the Abdominal cavity, the extra cellular fluid would have lubricated the squeezing of abdominal viscera into the inguinal canal, where the hernia would run posterior to the spermatic cord.

It is also possible that S. T. was a smoker, which may have left him with a chronic smokers cough. In this case the constant chronic coughing (each cough increasing intra-abdominal pressure) would have caused weakening of the deep inguinal ring over time. This would increase the chance of an IIH occurring on lifting a heavy object.

Constipation is also physiologically relevant. It causes increased intra-abdominal pressure, and can cause a hernia. This is very important to bear in mind when the patient is recovering post-op. Constipation can be caused by some analgesics, which the patient would be taking post-op, and hence cause reoccurrence of the hernia while the site of surgery is still healing, like a viscious cycle. On this note, Laparoscopic treatment could be considered to be more advantageous than Open Mesh surgery. A study by Mr. Sudhir Kumar et al. showed that patients who underwent the Lichtenstein procedure for hernial repair required analgesics for 6.4 days post-op, as opposed to the patients treated by Laparoscopy, who only required analgesics for 3.4 days post-op.

BIOCHEMICAL FEATURES

It has been found that people suffering from groin hernias tend to have an abnormal metabolism of connective tissue. The role of protease and antiprotease imbalance in the pathogenesis of groin hernias has lead to a new understanding of the aetiology of groin hernias, and causes of their reoccurrence. Frequency of groin hernias rises with age, and the activity of collagen-degrading enzymes is higher in older patients, presumably due to a reduced inhibition of collagenase, according to LT Sorensen and LN Jorgensen. However, ST is 35, so collagenases are unlikely to have had an effect in our case. They also think that smoking may be a contributor to the creation of groin hernia through inducing a systemic imbalance in levels of protease and antiprotease. Also, biochemical evidence shows that groin hernias
are associated with the impaired hydroxylation of proline, causing a weakening of connective tissue. This factor could have helped to weaken the boundaries of S. T.’s deep inguinal ring, and make it easier for the hernia to occur.

Given all this, suturing this tissue, or using it as an attachment, when it is already inadequate is a breach of the fundamental rules of Surgery, and is a good explanation for the high reoccurrence rates (>40%) of the original surgical treatments, such as the Shouldice technique.

**CLINICAL EXAMINATION**

There are six important points a physician should focus on when they examine for a hernia

1. Position of the hernia
2. Tenderness of the hernia
3. Shape of the hernia
4. Size of the hernia
5. Consistency of the hernia
6. Temperature of the hernia

The physician firstly examines the patient while lying supine. The examination is carried out by invaginating the scrotum with the little finger, and gently palpating the external inguinal ring, and posterior wall of the inguinal canal (which would be the internal oblique/conjoint tendon). Then on asking the patient to cough, the physician would feel for any impulse. If a hernia is present the physician should attempt to reduce it by taxis. If the hernia can be reduced by taxis, the physician should occlude the deep inguinal ring with finger pressure at the mid-inguinal point, and check to see if the hernia reappears during coughing. To distinguish what type of hernia it is, the physician needs to establish the anatomical relationships of the hernia.

In the case of S.T. with his already bulging hernia, the physician would have just tried to reduce it by taxis, and tried to establish if a truss could hold it in place, and how urgent the surgery was. From the path of the hernia, which was reported, “progressed medially for 4cm, and then turned toward the scrotum”, the physician was able to easily distinguish this as an IIH.

Latex gloves should be worn for the digital examination, and of course a history should be taken beforehand, with the history of smoking being a big factor.

**TREATMENT**

With a lifetime risk of 27% for men having an inguinal hernia, and 3% for women, and with over 20 million inguinal hernia repairs worldwide each year, the treatment of inguinal hernias is a big issue which still has plenty of room for improvement. It must be said, however, that the rates or reoccurrence have dropped hugely with the advent of newer surgical techniques. Originally with the first techniques used in the west in the 1890s rates of reoccurrence could be >40%, while now, with the Lichtenstein procedure, they are down to 1.4%.

If ST had been elderly and inactive, he would not have been treated surgically. Instead he would have had the hernia reduced by taxis, and been given a truss. This is a supportive device, which helps to strengthen the abdominal wall. It is worn as an undergarment. It is not considered unsafe to leave hernias untreated, especially in the elderly, as there is only a 2% chance of strangulation for those with an IIH. Seeing as ST needed to be able to be active, and seeing as he had left his hernia unattended for so long, he had to undergo surgery. There are four types of surgical herniorrhaphy which can be carried out. The first of which is not recommended at all.

1. Conventional- Bassini technique (1890) Shouldice technique (1940s)
2. Lichtenstein - anterior tension-free open-mesh method
3. Laparoscopic- Transabdominal Preperitoneal or Total Extraperitoneal
4. Plug technique- also open-mesh

The mesh used in types 2, 3 and 4 is made from polypropylene and is non absorbable.

The technique which ST would have been recommended to undergo would have been the Lichtenstein technique. Although 5% of surgeons favour the Laparoscopic technique (95% favouring the Lichtenstein) it is generally reserved for bilateral, or recurring hernias. The plug technique is still relatively obscure.

The Lichtenstein technique would have involved an incision 3-5cm long, running parallel to ST’s inguinal ligament, and just above it. The surgeon would have had to cut through the skin, Camper’s fascia, Scarpa’s fascia, and the Aponeurosis of the external oblique muscle. A polypropylene mesh is shaped and fitted into the inguinal canal in such a way that it strengthens the deep inguinal ring by distributing the stress-force it experiences during increased intra-abdominal pressure to the surrounding tissues. It is tension free, so there are no sutures pulling structures together under tension.
ST would have needed about 7-10 days before being able to resume normal activity, and full recovery would have taken about 4 weeks. One study suggests he would have had a 4.4% chance of recurrence with this procedure, and he could have had it all done as an out patient.

While it is probable that ST was placed under general anaesthetic, it is helpful for the patient to be conscious, so that the surgeon can ask him/her to cough. This assists in locating the hernia, and seeing if the repair is adequate.

An indirect inguinal hernia comes out through the anterior abdominal wall outside of Hasselbach’s triangle. While a direct inguinal hernia has nothing to do with the processus vaginalis, or the deep inguinal ring, and comes out through the abdominal wall inside the boundaries of Hasselbach’s triangle.

Laparoscopic technique
Transabdominal preperitoneal
www.lapsurgery.com/hernia.htm

Digital examination
medicine.ucsd.edu/clinicalmed/genital.htm

After barium meal
www.med.sc.edu:1000/inguinalhernia.htm

A truss
www.proline-sports.com/acatalog/UK_Hernia_Bel

Right indirect inguinal hernia
medicine.ucsd.edu/clinicalmed/genital.htm

A Hydrocele
(same as previous)
This section of case number 15 is dealing with differential diagnosis. Firstly I would like to begin by reiterating the importance of surgery as a treatment for inguinal hernia. Indirect inguinal hernia is a common but disabling condition and the surgery described previously should be sought as quick as possible because indirect inguinal hernia carries the threat of becoming an acute surgical emergency if the intestine it contains becomes obstructed or ischemic. Indirect inguinal hernia can diminish blood flow to the trapped portion of the intestine, a condition called “strangulation” and can lead to death of the affected bowel tissues. A strangulated hernia is life threatening and hence requires immediate surgery.

In order to distinguish indirect inguinal hernia from another diagnosis, the symptoms and risk factors of indirect inguinal hernia should be made clear. Signs and symptoms include-

- Pain or discomfort in the groin, especially when bending over, coughing or lifting.
- A heavy dragging sensation in the groin.
- Occasionally in men there will be pain and swelling in the scrotum and around the testicles when the protruding intestine descends into the scrotum. Most often it is this bulge that a patient will first recognise. This bulge may disappear when lying down as was the case with this patient but will be especially obvious when standing upright or coughing. An important risk factor relevant to this case is that males are ten times more likely to develop an indirect inguinal hernia than females.

Keeping this symptoms in mind, other diagnoses which could have been made by the physician include:

1. Inflammation or haemorrhage in the subcutaneous tissue may produce a very swollen scrotum, the swelling extending into the pelvis. This swelling will be similar to the one caused by an indirect inguinal hernia. However inflammation or haemorrhage in the subcutaneous tissue would most likely be caused by patients falling astride a bar or similar object or by a direct blow to the testes. For that reason the physician must be very precise when questioning the patient about his/her activity prior to the onset of pain and appearance of the bulge. In this case it occurred while straining to lift a heavy object.

2. An important determinant of indirect inguinal hernia is that it can be followed back to the superficial inguinal ring and it gives a clear pulse when the patient is asked to cough. In some cases fluid may accumulate within tunica vaginalis, preventing one from palpating the testes, such an accumulation is called a “hydrocele.” For this reason it may be difficult to distinguish between a scrotal swelling of testicular origin and a large inguinal hernia. If it is possible to get above the swelling it is not an indirect inguinal hernia but something confined to the scrotum like a hydrocele. Furthermore since the fluid is nearly always clear it is possible to transilluminate a hydrocele with a pocket torch.

3. Symptoms of testicular cancer again show some similarities to those of an inguinal hernia, such symptoms including: dull ache in scrotum and scrotal heaviness. But cancer of the testes will produce a hard non tender testicular mass, so a physician will distinguish between testicular cancer and indirect inguinal hernia based on the tenderness of the testicular mass in inguinal hernia and the painless testicular mass related to testicular cancer.

4. Another somewhat similar condition which still leaves the testes palpable is a cyst arising in a remnant of the peritoneal pouch associated with the descent of the testes. In this case a physician will distinguish between a cyst and an
indirect inguinal hernia by listening to the mass with a stethoscope for intestinal sounds associated with an indirect inguinal hernia but not a cyst. Hernia is not confined to the inguinal canal as other abdominal and pelvic regions are susceptible to herniation. Firstly the femoral ring is susceptible to femoral hernia which protrudes through the femoral ring normally closed by a femoral septum of modified extra peritoneal tissue and is therefore a weak spot. The pubic tubercle is an important landmark in differentiating an indirect inguinal hernia from a femoral hernia as the hernia’s neck is superomedial to it in inguinal hernia and inferolateral to it in the femoral form. The anterolateral abdominal wall can also be affected by hernia. Umbilical hernia is of three varieties: Congenital hernia- due to failure of retraction of the umbilical loop of the gut. Infantile umbilical hernia- due to stretching of umbilical scar tissue usually within 3 years of birth. Acquired umbilical hernia- this is hernia through the linea alba usually just above the umbilicus. Peritoneal folds of the peritoneal cavity are also susceptible to herniation because they may create a fossae or recesses of the peritoneal cavity. A segment of intestine that enters one can become constricted at the entrance of the recess leading to a form of internal hernia. Finally the jejunum and ileum can also be affected by hernia due to an external injury. The rupture of the jejunum and ileum by external injury is due to their elasticity and mobility. The more fixed duodenum particularly its horizontal part across the vertebral column is more vulnerable. Omentum commonly protrudes into the hernia sac, other abdominal viscera may rarely be involved. To finish up here are some suggestions on how to minimise the risk of hernia. Obviously one cannot prevent the congenital factor that may lead to an indirect inguinal hernia but one can take steps to reduce strain on abdominal muscles and tissues by: 1. Maintaining a healthy weight. 2. Emphasizing high fibre foods 3. Not relying on a truss for longterm support. Wearing a truss is not a longterm solution for an inguinal hernia as a truss will not protect against complications or correct the underlying problem but it is suitable as a temporary solution provided it is utilised correctly.

Case # 16 – Small Bowel Obstruction

The Peritoneum is a mesothelial lining of the abdominal cavity having visceral and parietal layers. Parietal - covers the abdominal wall & Visceral - forms surfaces of organs, the serosa.

The small intestine, consisting of the duodenum, jejunum and ileum extending from the pylorus to the ileocecal valve, with a length of 6-7m approximately.
The adult duodenum is 20-25cm long, with the proximal 2.5cm intraperitoneal and remainder retroperitoneal. The duodenum forms an elongated ‘C’ between vertebrae levels L1-L3, with the head of the pancreas lying within the concavity of the ‘C’. Beginning at the pylorus and ending at the duodenojejunal junction, it is described in four parts.

The superior part is short, approximately 5cm long, mostly horizontal, lying anterolateral to the body of L1. The first 2-3cm is called the duodenal cap, due to its triangular homogenous appearance during radiography. From the duodenal cap to midway along the ileum, the internal lumen is characterised by extensive mucosal folds visible in radiographs. The superior relations are similar to those of the stomach with the lesser omentum passing from the upper border and the greater omentum from the lower. It rests on the pancreas inferiorly.

The descending part is longer (7-10cm) and runs inferiorly along the right sides of L2-L3, curving around the head of the pancreas. Initially it lies to the right and parallel to the IVC. Its anterior surface is crossed by the transverse colon, with the coils of the small intestine lie inferiorly. This part of the duodenum contains the major and minor duodenal papilla.

The horizontal part is approximately 6-8cm long at L3 level, lying below the head and uncinate process of the pancreas. Anteriorly it crosses the IVC and aorta. It is crossed anteriorly by the superior mesenteric vessels and the root and mesentery of the small intestine. If the mid portion of the third part is potentially pinched between the SMV and the aorta it may give rise to intermittent obstruction.

The ascending part is short approximately 5cm and begins at the level of L3 vertebrae rising superiorly to the superior border of L2. It lies on the left psoas. It is related anteriorly to the root of the mesentery and the coils of the small intestine. At the inferior border or the pancreas it curves anteriorly to join the jejunum at the duodenojejunal flexure which is supported by the attachment of the ligament of Treitz, composed of a slip of skeletal muscle from the diaphragm and a fibromuscular band of smooth muscle from the duodenum. The ligament is an important landmark in the radiological diagnosis of incomplete and malrotation of the small intestine.

An important transition in the blood supply of the alimentary tract occurs at the duodenal papilla. Proximally the abdominal part of the alimentary tract is supplied by the celiac trunk with parts 1-2 as far as the ampulla supplied by the gastroduodenal artery via the superior pancreaticoduodenal artery. Distally parts 3-4 are supplied by the superior mesenteric artery via the inferior pancreaticoduodenal artery. Venous drainage via the duodenal veins which ultimately drain to the portal vein. Nerve supply is via the vagus and sympathetic nerves via the celiac and superior mesenteric plexuses. Pain from the duodenum is poorly localised and referred to the central epigastrium.

The jejunum begins at the duodenojejunal junction and the ileum ends at the ileocecal junction. Together the jejunum are 6-7m long in cadavers; however tonic contractions make them substantially shorter in a living person. The jejunum constitutes approximately two fifths of the length and the ileum the remainder. Both lie in the free edge of the mesentery, a fan shaped fold of peritoneum attaching the jejunum and ileum to the posterior abdominal wall. The root is directed obliquely, inferiorly and to the right extending from the duodenojejunal junction to the ileocolic junction and the right sacroiliac joint.

The jejunum lies superiorly and to the left in the infracolic compartment and the ileum lies to the right extending over the pelvic brim and into the pelvis. The majority of the jejunum and ileum is covered anteriorly by the greater omentum. The mucosa of the jejunum and ileum are thrown into numerous circular folds which protrude into the lumen, these are more pronounced in the jejunum giving it a characteristic appearance during chest radiography. The submucosa contains aggregates of lymphoid tissue, more obvious in the ileum. The superior mesenteric artery supplies the jejunum and ileum, running between layers of the mesentery and giving off branches. The arteries unite to form loops
(arterial arcades) which give rise to straight arteries the vasa recta. The superior mesenteric vein drains the jejunum and ileum lying to the right of the SMA in the mesentery, ending posterior to the neck of the pancreas where it unites to form the portal vein. The lymph vessels lie between the layers of the mesentery. Nerve supply is via the superior mesenteric plexus, receiving preganglionic parasympathetic elements via the right vagus nerve, with sympathetic fibres from thoracic spinal segments T5- T9 and lesser splanchnic nerves. Pain is poorly localised and is commonly referred to the periumbilical region or central epigastrium.

The large intestine extends from the distal end of the ileum to the anus, a distance of 1.5m, consisting the cecum, appendix, colon, rectum and anal canal.

A characteristic that distinguishes all parts of the large intestine (except rectum) from the small intestine is the way in which the longitudinal muscle is organised. The outer layer of longitudinal muscle of the duodenum, jejunum and ileum is uniformly distributed as a layer of circular muscle. In the large intestine the longitudinal muscle is thickened into three concentrated longitudinal bands called taeniae coli. Their presence causes the walls to form pouch like sacculations, Haustra. The large bore and presence of appendices epiploicae help distinguish the large from small intestine.

Clinical Diagnosis

Small bowel obstruction is the mechanical obstruction of the bowel lumen causing blockage to the bowel contents so that there is a collection of fluid and gasses proximal to the obstruction which will lead in bowel distention. The fluids could be of gastric juice or pancreatic, biliary or bowel fluid, while the gasses are of swallowed air and gasses released from bacterial fermentation. The distention will stimulate the bowel cell’s secretory activity which will worsen the problem. There is hyperactivity proximal to the obstruction and inactivity in the bowel distal to the obstruction.

There are 3 major groups of causes of the small bowel obstruction. The first one is the mechanical cause, which is simply anything that can obstruct the lumen of the bowel, for example gallstones or foreign objects. Intrinsic causes are causes that constrict the bowel from the interior of the bowel itself, such as tumours or inflammatory diseases. And lastly is the extrinsic cause, which are causes that constricts the bowel from the exterior, such as adhesion and hernias.

Adhesion is an internal band of scar tissue that forms between organs as tissues heal from injuries that might be due to infection, inflammation of surgery. It connects organs and tissues that are not normally attached and this will lead to disturbance in functions of the organs, causing abdominal pain and bowel obstruction.
The patient has several signs and symptoms, the first one being colicky abdominal pain. Colicky pain is pain which comes and goes. This is due to the increase in peristaltic action in which the bowel tries to force the blocked contents through the obstruction.

Nausea and vomiting is because of distention of the small bowel results in increased intra luminal pressure. This will compress the mucosal lymphatics which in turn will cause bowel wall lymphedema. Collection of lymph in the bowel will induce vomiting in the patient.

There is also distention of the abdomen. This is due to the accumulation of gasses and fluids in the small bowel that causes the bowel to become distended and will in turn cause the abdomen to be distended.

There are also signs of inflammation as indicated by the fever and tachycardia. These are the signs of a strangulating bowel obstruction, where the mesenteric blood supply of the bowel is cut off, causing the cell to undergo ischemia and, if prolonged, can lead to infarction. This will then lead to bacterial and endotoxin translocation in the normally sterile small bowel. The bacteria and endotoxin can the leak into the peritoneal cavity, if there is a perforation in the gut wall, causing peritonitis.

Our patient also suffers from dehydration, which is indicated by the lowered blood pressure, because she is constantly losing fluid either through oedema in the bowel, or by vomiting.

The elevated WBC count is due to necrosis of the small bowel wall and as a result of the peritonitis.

Upon palpation, the patient demonstrates involuntary guarding, which is spasm of the abdominal muscle wall which causes muscle rigidity that can’t be suppressed and rebound tenderness, extreme localised pain that is felt when pressure that is applied to the abdominal wall is suddenly removed. Both of these symptoms are indicative of the peritonitis.

X-ray findings of the patient show dilated small bowel loops due to accumulation of fluids and gasses in the small bowel. There’s also multiple “air-fluid level”s. These are caused by collection of fluid and air in the small bowel. The air rises above the fluid, creating a smooth surface at the interface between the air and fluid. Lack of air in the distal colon is also another sign the bowel has been obstructed.

Normally, food and liquid course through the intestines by means of peristalsis and these peristaltic movements of the bowel creates bowel sounds. In this case, auscultation of the patient’s abdomen reveals intermittent high bowel sounds due to the abdomen trying to force its contents through the obstruction.

**Differential diagnosis and treatment**

There are many conditions that show a lot of the same symptoms as Small Bowel Obstruction, or SBO, the most important being: Paralytic Ileus, Pseudoobstruction, Pancreatitis, Acute Gastroenteritis, Acute Mesenteric Ischemia, Appendicitis, Cholecystitis and Large Bowel Obstruction.

Ileus, or Paralytic Ileus or non-mechanical obstruction, occurs when peristalsis stops. The symptoms after the bowels stop moving are abdominal cramping and distention, nausea, vomiting and failure to pass gas or stool. Our patient has all these symptoms but Ileus is not diagnosed because in the case of Ileus, when a doctor listens with a stethoscope to the abdomen there will be few or no bowel sounds. On auscultation, our patient had intermittent high-pitched bowel sounds. Also, the x-ray showed the signs of an SBO, therefore Ileus can be ruled out.
With a Pseudoobstruction the intestinal walls are unable to contract normally (hypomotility) to generate wave-like (peristaltic) motion. This condition resembles a true obstruction, but no such blockage exists. Symptoms are again similar and also enlargement of various parts of the small intestine occurs. With the x-ray it was revealed our patient had a SBO with dilated small bowel loops and therefore Intestinal Pseudoobstruction could be ruled out.

Pancreatitis is an inflammation of the pancreas. One important function of the pancreas is to secrete digestive enzymes into the small intestine. In Pancreatitis, these enzymes become active inside the pancreas and start digesting the pancreas. The symptoms of this usually begin with severe pain in the upper abdomen. Some other symptoms are: swollen and tender abdomen, sweating, nausea, vomiting, fever, mild jaundice and rapid pulse. Our patient shows some of these but the condition is ruled out because the patient's abdomen is distended and auscultation reveals intermittent high-pitched bowel sounds. The x-ray further confirms the patient is not suffering from Pancreatitis.

Gastroenteritis is the inflammation of the stomach and the intestines, usually caused by a virus but it can also be caused by bacteria or bacterial toxins. It is Bacterial Gastroenteritis, also known as Acute Gastroenteritis, that would be of more concern to us in this case with the symptoms being similar to those of our patient. But again this condition could be ruled out for the same reasons as Pancreatitis.

Mesenteric Ischemia is a condition in which the mesenteric arteries do not deliver enough blood and oxygen to the small and large intestines. The most prominent symptom is severe abdominal pain in the area of the navel around 30 to 60 minutes after eating. Other symptoms might include diarrhoea, nausea, vomiting, and constipation. The symptoms of Mesenteric Ischemia are common to many other conditions, so lab tests are extremely important in its diagnosis. Blood tests may show an elevated white blood cell count in the case of Mesenteric Ischemia which is seen in our patient. It is important for this case that Mesenteric Ischemia may be a complication due to the intestinal obstruction so ischemia cannot be ruled out by the findings of a SBO in the x-ray.

Appendicitis has similar symptoms to SBO but this is obviously ruled out because the patient’s previous surgical history includes an appendectomy. The appendectomy is important when diagnosing this patient because, as Has has explained earlier, this surgery can have future complications, even decades after the surgery.

Cholecystitis is when the gallbladder becomes infected. Again there is an elevated White Blood Cell count but for the same reasons as before it is ruled out.

Radiologically, SBO can be differentiated from large bowel obstruction. Small bowel is recognised by the presence of valvulae conniventes. Also dilated small bowel loops tend to be central. The colon is recognised by its larger lumen and haustrations and it is more peripheral. From this we can determine where the obstruction is located and we can rule out large bowel obstruction.

The main types of SBO are mechanical obstruction, simple obstruction implies one obstruction point, closed-loop obstruction is a blockage at two or more points and it may develop when the bowel twists around on itself, Paralytic Ileus, I've already explained and strangulating obstruction, which was explained earlier.

The diagnosis of a SBO is made and the patient is sent to surgery for evaluation. Strangulating obstruction is difficult to diagnose preoperatively. Abdominal exploration, also known as laparotomy or exploratory laparotomy, is the surgical procedure that opens the abdomen and explores it for problems. During a laparotomy treatment of problems may also be administered. Some problems inside the abdomen can be easily diagnosed with non-invasive tests, for example abdominal x-rays and CT scans,
but many problems require surgery to explore the abdomen to obtain an accurate diagnosis. Our patient had an elevated white blood cell count, indicating an infection, peritonitis and abdominal tenderness, all signs of a strangulating obstruction. You have to be quick to send the patient to surgery because the blood supply to the gut wall is being cut off. If this is prolonged death will occur.

Case 17

2nd Medicine

ANATOMY CLINICAL CASE
PRESENTATION.

CASE NUMBER;

PRESENTED 16th FEBRUARY
by;
Anatomy and Histology of Stomach

A gastric ulcer is a sore on the lining of the stomach, which, in our case, has invaded the muscular coats of the stomach wall and has become a perforated ulcer.

An ulcer occurs when there is an imbalance between the rate of secretion of stomach acid and pepsin by the gastric cells and the secretion of protective bicarbonate ions into the deeper layers of the surface mucous coat of the stomach lining. Ulceration is most commonly caused by the bacterium Helicobacter Pylori. Helicobacter Pylori helps the stomach acid to erode the walls by weakening the protective mucous lining. This enables the acid to get through to the sensitive lining underneath. If this happens, both the acid and bacteria irritate the lining and cause a sore or ulcer. Another cause is the extensive use of non-steroidal anti-inflammatory agents like aspirin and ibuprofen. In a few cases cancerous tumours in the stomach or pancreas can cause ulcers. Often, spicy foods, excessive alcohol, and stress are claimed to cause ulcers. This is not the case but they do worsen the condition.

In order for an ulcer to perforate the walls in the stomach, it must pass through each of the 4 layers that make up the wall. These are the mucosa which is the innermost protective lining, the submucosa containing the blood vessels supplying the stomach, the muscularis propria where the 3 layers of smooth muscle of the stomach are found, oblique, circular, and longitudinal and finally the adventitia which is the outermost layer of loose connective tissue. The mucosa of the body of the stomach, where the ulcer was found in our case contains glands embedded in the mucosa that secrete acid and peptic gastric juices as well as some protective mucous. The glands open into gastric pits and those pits occupy about one quarter of the thickness of the gastric mucosa. The cells we are concerned with in the glands are the surface and neck mucous cells that secrete the protective bicarbonate ions, the oxyntic cells, which secrete the gastric hydrochloric acid, and zymogenic cells, secreting pepsin. The secretion of acid and pepsin is controlled by the vagus nerves and the hormone gastrin.

The most common location for ulcers is the near lesser curvature, which is where it has occurred in our case. In relation to the rest of the stomach, the lesser curvature forms the right border and extends from the cardiac orifice, which is the opening of the oesophagus into the stomach, and the pylorus, which is
where the stomach opens into the duodenum. The stomach itself is found in the upper part of the abdomen, extending form beneath the left costal margin into the epigastric and umbilical regions. As the perforated ulcer occurred in the posterior, structures related posteriorly to the stomach could also be damaged by the gastric juices. These structures include the pancreas, left suprarenal gland, upper part of the left kidney, diaphragm and splenic arteries. In our case, the diaphragm was irritated as the patient experienced referred pain in her right shoulder. Pain was experienced in here as the phrenic nerves, which arise from the cervical nerves 3, 4, and 5, which also contain the nerves supplying the shoulder, region, innervate the diaphragm. The epigastric pain also experienced by our patient arose because the afferent pain fibres from the stomach ascend in company with the sympathetic nerves and pass through the celiac plexus and the greater splanchnic nerves. The sensory fibres enter the spinal cord at segments T5 to T9 and give rise to referred pain in the dermatomes of these segments on the lower chest and abdominal wall which is the epigastric area. The parasympathetic nerve supply to the stomach is derived from the vagus nerve and it controls the secretion of acid and pepsin. The area where the ulcer occurred, the posterior surface, is supplied by a branch of the posterior vagal trunk, which is formed in the thorax from the right vagus nerve. The trunk enters the abdomen on the posterior side of the oesophagus and then divides to give off branches that supply the celiac and superior mesenteric plexuses as well as the posterior aspect of the stomach.

The celiac trunk gives off the main blood supply of the stomach. The branches of the trunk are the left gastric artery, the hepatic artery and the splenic artery. The left gastric artery passes superiorly and to the left to reach the oesophagus then descends along the lesser curvature of the stomach. This, along with the right gastric artery supplies the area of the stomach where our patient’s ulcer was found. The right gastric artery arises from the hepatic artery of the celiac trunk at the upper border of the pylorus and it runs to the left along the lesser curvature. The 3rd branch of the celiac trunk, the splenic artery gives rise to vessels supplying the other areas of the stomach; the short gastric arteries which supply the fundus, the left gastroepiploic artery which supplies the upper part of the greater curvature. The lower part of the greater curvature is supplied by the right gastroepiploic artery, which is derived from the other branch of the hepatic artery, the gastroduodenal artery.

**Signs and Symptoms of a Perforated Gastric Ulcer**

A gastric ulcer pre-ulceration causes a variation of signs and symptoms ranging from epigastric pain after meals to vomiting and weight loss. Sometimes however the pain and other symptoms are mild and can be ignored or temporarily relieved by antacids. This is how it can progress to the perforation stage. When perforation occurs the signs and symptoms cannot be so easily ignored.

**Signs**

Shock, circulatory and septic.

- Low temp.
- Low b.p.
- Dilated pupils
- Cold, pale, clammy
- Irregular breathing (costal in nature with avoidance of abdominal exertion)
- Weak rapid pulse
- Reduced urinary flow rate

Patient lies very still
Guarding of the abdomen, rigid abdominal muscles. This is a reflex response to abnormal stimuli arising from the infected area and helps to protect against pain.

note: in this case the patient did not present with this “guarding” as the perforation was in the posterior wall of the stomach and so the peritonitis was confined to the lesser sac. Rigidity could occur if the inflammation were to spread to the greater sac via the communicating foramen, the Foramen of Winslow.

**Symptoms**

Epigastric pain

- Severe
- Sudden onset

Pain in the tip of the right shoulder

- Referred pain. Due to common innervations of diaphragm and skin over right shoulder as explained in the anatomy

Our patient presented with both the symptoms mentioned above. She had a history of gastric ulcer and had recently ceased taking her medication. She also has a high powered executive job which is most probably stressful. Given her history and her signs and symptoms we suspected a perforation of a gastric ulcer had occurred. Certain diagnostic tests were then carried out to investigate further.

**Tests**

**Gastroscopy**

A gastroscope is an endoscopic tool for the stomach. It is an illuminated optical instrument used to inspect the interior of the stomach. It is essentially a camera on a stick which allows all areas of the stomach to be seen and photographed and specimens taken for microscopic exam. Care must be taken when performing his exam as gastrosopes can be the cause of perforation.

Above are two endoscopic images of a perforated gastric ulcer.

A perforation was discovered on the posterior wall of the stomach near the lesser curvature. This confirmed the diagnosis.
Plain x-rays of the abdomen with the patient in the upright position have been used in diagnosing perforated ulcer. Some cases have been negative for free air (especially in elderly) but the majority (50-70%) in several case series have been positive for free air in the peritoneal cavity. A left lateral decubitus film, with patient in position for at least ten minutes before hand has been shown to be the most sensitive in detecting pneumoperitoneum. Similarly, use of water-soluble contrast medium with an upper gastrointestinal tract series or computed tomography scan may increase the diagnostic yield.

**Barium meal**

A chalky liquid is drunk and an x-ray is performed, showing the stomach lining. These tests are less common nowadays, but may be useful where endoscopy is unavailable.

**Biopsy**

_A small tissue sample is taken during an endoscopy and tested in a laboratory. This biopsy should always be (but in our case was not) done if a gastric ulcer is found as the two are very commonly associated._

**C14 breath test**

(in a perforation this test is not usually performed as it is a medical emergency but it can be useful in a less urgent scenario)

To check for the presence of _H. pylori_. The bacteria convert urea into carbon dioxide. The test involves swallowing an amount of radioactive carbon (C14) and testing the air exhaled from the lungs. A non-radioactive test can be used for children and pregnant women.

**Differential Diagnosis**

Any condition causing the shock and abdominal pain should be considered.

Important ones to consider are
o Pancreatitis- Check serum amylase levels
o Perforated duodenal ulcer- duodenoscope
o Biliary Colic
o Cholecystitis- Ultrasound and x-rays…… (NB as she may fit the f-profile (Fair Fat Female in her Forties)
o Pleurisy- (high temp not low temp.)
o Ruptured appendix- localised pain in mcburneys poin, high temp, vomiting.
 o MI- SGOT serum levels
 o Renal Colic

http://www.surgical-tutor.org.uk/default-home.htm?system/abdomen/perforated_peptic.htm~right

**TREATMENT**

There are a number of treatments that relieve the discomfort associated with ulcers and, other treatments to actually heal the ulcer, itself.

**ORIGINS**
The current "revolution" in ulcer cause and treatment was first noted by Dr Barry Marshall, who discovered the presence of a small bacterium, under the microscope, attached to the lining of stomach wall samples taken from patients' ulcers, in 1982. These initial findings have been confirmed as a cause of ulcer disease. The bacterium is called Helicobacter Pylori (or H Pylori, for short). Current medical opinion suggests that anyone with ulcer symptoms should, at some point, be checked for the presence of H Pylori. The importance of this finding is that, in many cases of H Pylori positive ulcers, antibiotics can actually heal the ulcer. This is a revolution in thinking.

There are many new ulcer treatment plans available, depending on the type and cause for the ulcer in question.

Ulcers require specific treatment. It is important for you to make every effort to contribute to this process. Smoking cigarettes has consistently been found to slow down the healing of an ulcer. Avoidance of alcohol, aspirin, and other inflammation-reducing drugs is also advisable, especially during a flare-up, because these substances cause increased stomach acid production which weakens the stomach lining and makes it vulnerable to damage. Other parts of a treatment plan may include the following recommendations:

- **Diet** Milk and cream diets were once widely used in the treatment of ulcers, but the theory behind such treatment has since been abandoned. Currently, the main dietary rule is to eat regular, nutritious meals. Otherwise, use common sense and avoid foods that bother you.
- **Alcohol**, nicotine, and tobacco cause the most acid production in the stomach. Other dietary factors may include tomatoes, citrus, spicy foods, chocolate, and mint.
- **Drugs:** There are many different types of drugs available today for treating ulcers. Some are over-the-counter preparations that are used to neutralize stomach acid, and others are prescription medications that promote healing by inhibiting acid secretion, blocking acid
production, eliminating (H Pylori) if present, or providing a protective coating over the ulcer. These medicines will be prescribed according to the likely cause for the ulcer.

**Surgery**

For the majority of people with ulcers, the success of dietary therapy, drug therapy, or a combination of the two makes surgery unnecessary. Indications for surgery usually include perforation, obstruction due to scarring, and/or unrelenting bleeding. Uncontrollable pain may also justify surgery, but usually only after other alternatives have failed. In the case of our patient a perforation was confirmed by means of a gastroscopy and therefore surgery was deemed necessary.

The surgeon would perform a laparoscopy and once inside the abdomen perform a vagotomy. A vagotomy is the surgical cutting of the vagus nerve to reduce acid secretion in the stomach. The Vagus nerve stimulates acid and mucous production in the stomach by secreting acetylcholine and if the vagus is overactive the pH becomes very low. This is not of permanent consequence to the patient as basal cell acid production compensates a few weeks after surgery.

The surgeon repaired the ulcer and this is normally done by a simple oversew of tissue from the greater omentum. However, many malignant gastric ulcers are often large and ragged, making closure impossible or unsafe. In this case many surgeons would perform a partial gastrectomy. Others would excise the ulcer to exclude malignancy and perform a vagotomy. The latter probably occurred in our case.

As previously described, the three branches of the celiac trunk supply different parts of the foregut. In our case study the surgeon had to ligate the left gastric artery during the surgery. If this artery is occluded due to ligation, collateral circulation from the right gastric artery (a branch of the proper hepatic artery), right gastroomental artery (a branch of the gastroduodenal artery), and the left gastroomental and short gastric arteries (branches of the splenic artery) is usually adequate to compensate for loss of flow through the left gastric artery.

Although variations in the celiac trunk are quite common, the particular variation described in our case is quite rare. If the common hepatic artery did originate from the left gastric and the left gastric were ligated, the only blood flow to the stomach would be from the left gastroomental artery from the splenic artery. This may not be sufficient for the stomach's survival. Also, critical blood flow to the liver, duodenum, gall bladder and pancreas would be interrupted, leading to the death of the patient unless blood flow was promptly restored.

The risks, as in all surgery, are infection and bleeding. Some additional risks are changes in gastric activity, diarrhea and dumping syndrome. After surgery the patient is required to remain in hospital for around a week for observation. Nasogastric suctioning is required for the first couple of days.

**Conclusion**

Peptic ulcers are easily treated since the discovery of H. pylori’s role in their formation. Antibiotics and dietary therapy can now effectively treat the vast majority of cases and surgery is now a last resort in cases of extreme neglect.
Case 18

INTRODUCTION

Our case deals with a 48 year-old male diagnosed with alcoholic cirrhosis of the liver. He has a history of alcoholism. Alcoholism is a “disease” with symptoms such as craving, impaired control, physical dependence and tolerance. Excessive alcohol consumption can lead to Alcoholic Liver Disease (ALD). Cirrhosis is the final stage of ALD characterized by scarring of the liver and development of liver nodules. It severely affects liver function.

ANATOMICAL RELEVANCE

The liver is the largest organ in the body. It is situated in the upper right hand corner of the abdomen extending from the right through left hypochondriac regions. The main functions of the liver include The liver is responsible for breaking down harmful substances that could be present in alcohol, drugs or even food, into harmless substances that can be excreted with the body's urine or stool. It also produces compounds, especially proteins that are necessary for bodily functions as well as acting as a store for fats, sugars and vitamins. In cirrhosis these functions are lost.

The portal venous system consists of the portal vein and its tributaries: Splenic vein, Inferior and Superior Mesenteric Veins, Left and Right Gastric Veins and Cystic Veins. Normally portal venous blood traverses the liver and drains into Inferior Vena Cava. In the case of portal hypertension, like in our patient, blood from the Gastro-Intestinal tract reaches the Inferior Vena Cava via several collateral routes. These routes are available because the portal venous system of veins does not contain valves, therefore blood can flow in a reverse direction towards the Inferior Vena Cava. The locations of anastomosis between portal system and systemic system are as follows:

<table>
<thead>
<tr>
<th>PORTAL SIDE</th>
<th>SYSTEMIC SIDE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior rectal vein</td>
<td>Middle and inferior rectal veins - To IVC</td>
</tr>
<tr>
<td>Esophageal tributaries of the left gastric vein</td>
<td>Esophageal veins - To Azygos vein</td>
</tr>
<tr>
<td>Paraumbilical veins</td>
<td>Subcutaneous veins of anterior abdominal wall - To IVC</td>
</tr>
</tbody>
</table>
**What is Cirrhosis?**
Pathologically defined as irreversible, diffuse fibrosis (or scaring) of the liver. The scar tissue replaces normal, healthy tissue, blocking the flow of blood through the liver and preventing it from working as it should. **Other causes of cirrhosis:** Chronic hepatitis C, Chronic hepatitis B and D, Autoimmune hepatitis, Inherited diseases, Non-alcoholic steatohepatitis, Blocked bile ducts, Drugs, toxins and infections. **Early symptoms:** fatigue, weakness, exhaustion, loss of appetite with nausea and weight loss and epigastric pain.

**Symptoms, Signs and Complications**

- **Jaundice** - yellow discolouration of the skin and whites of the eyes which is due to the increased levels of bilirubin in blood. This occurs when the diseased liver does not absorb enough bilirubin excreted.

- **Tachycardia** - This is due to the elevated serum cholesterol and triglycerides levels which then cause atherosclerosis. Heart has to pump blood more rapidly due to and increased demand for oxygen by the cardiac tissue.

- **Low blood pressure** - It is believed that substances circulating in the blood that are normally cleared by a healthy liver can cause a generalized dilatation of the blood vessels throughout the.

- **Spider nevi (hemangiomas)** - These are enlargement of blood vessels that resemble little spiders. A network of dilated capillaries (tiny blood vessels) radiate from the central arteriole.

- **Portal hypertension** - An increase in portal blood pressure due to obstruction of, or increased resistance to blood flow. This forces shunting of blood from portal to systemic venous systems and the engorgement of portocaval anastomoses since that is the path of least resistance.

- **Ascites** - It is the presence of excess fluid in the abdominal cavity. When portal hypertension occurs, the high pressure is transferred to the splenic, superior and inferior mesenteric veins. This causes fluid to ooze out of the capillary beds and accumulate in the abdominal cavity.

- **Hematemesis** - It is the vomiting of blood due to oesophageal varices. Engorgement of the portocaval anastomoses causes varicosities between the mucosa and muscular wall of the bowel and predisposition to rupture.

- **Splenomegaly** - It is the enlargement of spleen. When portal hypertension occurs, the high pressure is transferred to the splenic vein causes engorgement and subsequent enlargement of spleen

- **Caput medusae** - the appearance of distended and engorged umbilical veins which are seen radiating from the umbilicus across the abdomen to join systemic veins. The varicosities of subcutaneous veins in the area of the navel cause this to happen.

- **Internal haemorrhoids** - These are dilated (enlarged) veins which occur in and around the anus and rectum. One-third of rectal veins travel to the liver via the portal venous system. Back flow of blood cause enlargement of normal spongy blood-filled cushions in the wall of anus and may bleed.
Diagnostic Tests

- **Physical Examination** - This includes inspection (looking), palpation (feeling), auscultation (listening) and percussion (producing sounds). A physical examination may reveal an enlarged liver or spleen, distended abdomen, yellow eyes or skin (jaundice), red-spider like blood vessels on skin, reddened palm and/or dilated abdominal wall veins.

- **Liver function tests** - These are actually a group of blood tests performed to check whether or not the liver is functioning normal. However, lab findings can be normal in cirrhosis with slight elevation of blood liver enzymes and bilirubin levels.

- **CAT scan, ultrasound or radioisotope scan** - A scanning procedure using X-rays and a computer to detect abnormalities of the body's organs. This technique provides cross-sectional images of body organs, which is much clearer than those provided by conventional X-rays.

- **Laparoscope** - A tiny camera is inserted through a small slit or incision in the abdomen to view the liver directly. The laparoscope sends images of the liver to a monitor. The physician watches the monitor and uses instruments in the laparoscope to remove tissue samples from one or more parts of the liver.

- **Liver biopsy** - This is a simple, rapid method where a needle is inserted through the ribcage or abdominal wall and into the liver to obtain a sample of the liver tissue. The tissue is then prepared, stained in a laboratory and viewed under a microscope for any abnormal features of the liver tissue which a specific diagnosis and determination of the seriousness of the condition can be made. Biopsy is the only way diagnosis can be 100% certain.

**Treatment of our patient’s alcoholic cirrhosis** should aim to stop progress of the cirrhosis and treat complications that are disabling or life threatening. The most important aspects of treatment are abstinence from alcohol and an adequate wholesome diet. Abstinence from alcohol should be encouraged with help from our patient’s family, psychiatric intervention & also support groups such as Alcoholics Anonymous.

Our patient could also be prescribed with benzodiazepines to prevent alcohol withdrawal phenomena. **Disulfiram** can be given as a deterrent to patients who have difficulty resisting sudden impulses to drink after becoming abstinent. The drug blocks the metabolism of alcohol causing acetaldehyde to accumulate in the body. When alcohol is consumed there follows an unpleasant reaction consisting of headaches, flushing, nausea & laboured breathing. Knowledge that this reaction will occur can provide our patient with an insurance against drinking and even remove craving.

**Our patient’s Portal venous pressure can be reduced in 2 ways:**
1. **Pharmacologically:** Vaso-pressin constricts the splanchnic arterioles & reduces portal blood flow & hence portal pressure. Octreotide, the synthetic form of somatostatin reduces the portal pressure & can stop variceal bleeding.

2. **Transjugular Intrahepatic portosystemic shunt (TIPSS):** This is a technique in which a stent is placed between the portal vein & hepatic vein in the liver to provide a portosystemic shunt to reduce portal pressure. The procedure is carried out under radiological control via the internal jugular vein. Successful shunt placement stops & prevents variceal bleeding. Further bleeding necessitates investigation & treatment (e.g. angioplasty) because it is usually associated with shunt narrowing or occlusion. Hepatic encephalopathy may occur following TIPSS & requires that the shunt diameter be reduced.

_Hematemesis in our patient was caused by oesophageal varices and these can be treated by:_

1. **Sclerotherapy:** This is the most widely used method for preventing recurrent oesophageal variceal bleeding. Varices are injected with sclerosing agent as soon as practicable after bleeding, and injections are repeated every 1-2 weeks thereafter until the varices are obliterated. The treatment is not free of risks as the injection can cause transient chest or abdominal pain.

2. **Banding:** This is a technique in which varicies are sucked into an endoscope accessory, allowing them to be occluded with a tight rubber band. The occluded varix subsequently sloughs with variceal obliteration.

3. **Balloon tamponade:** After endotracheal intubation to protect the airway, a special tube is passed through the mouth into the stomach & a balloon inflated. Traction is exerted on the upper end for up to 4 hours to arrest haemorrhage. Several varieties of tube are in use e.g. Sengstaken–Blakemore tube has separate intragastric and oesophageal balloons & depends for its action upon physiological arrest of bleeding.

_Our patients Ascites can be treated by:_

1. **Sodium & Water restriction:** Conventional treatment aims to reduce body sodium and water by restricting intake, promoting urine output and if necessary removing Ascites directly. Non-steroidal analgesic agents must be avoided. A few patients can be managed satisfactorily on this treatment.
2. **Diuretic drugs**: Most patients require diuretic drugs in addition to sodium restriction. For example, spironolactone is the choice for long-term therapy because it is a powerful aldosterone antagonist—side effects include painful gynaecomastia. Some patients also require powerful loop diuretics—e.g., furosemide, though these can cause fluid, electrolyte & renal function disorders.

3. **Paracentesis**: is the process of drawing off fluid from the body. Paracentesis of 3-5 litres over 1-2 hours has always been used for intermediate relief of cardiorespiratory distress due to gross ascites. Large-volume Paracentesis alone has been regarded as a hazardous treatment; however, Paracentesis to dryness or the removal of 3-5 litres daily is safe, provided the circulation or supported by giving a colloid such as Human albumin solution (6-8 g per litre of Ascites removed) or another plasma expander. Total Paracentesis can be used as an initial therapy or when other treatments fail.

4. **LeVeen shunt**: The LeVeen shunt is a long tube with a non-return valve running subcutaneously from the peritoneum to the internal jugular vein in the neck, which allows ascitic fluid to pass directly into the systemic circulation.

**Treatment of Haemorrhoids (Piles)**: involves measures to prevent constipation & straining. Injection sclerotherapy or band ligation is effective for most, but a minority of patients require haemorrhoidectomy, which is usually curative.

*Surgery*: liver transplantation is indicated for some patients with irreversible, progressive cirrhosis.

**Differential Diagnosis.**

Other diseases that should be considered when making a diagnosis of alcoholic cirrhosis are:

1. **Haemochromatosis**: The disease usually presents in men aged 40 years & over. It is a condition in which the amount of total body iron is increased; the excess iron is deposited in & causes damage to several organs including the liver. Early clinical features particularly tiredness, fatigue & arthropathy, are increasingly recognised.

2. **Hepatocellular carcinoma**: More common in males. A primary malignant tumour of the liver. It is characterized by hepatomegaly, pain, hypoglycaemia, weight loss, anorexia, ascites as well as elevated serum alpha-fetoprotein levels, portal hypertension & jaundice in the plasma. The tumour occurs most frequently in association with hepatitis or cirrhosis of the liver & in those parts of the world where mycotoxin aflatoxin is found. Screening used to identify this form of carcinoma. Can be detected by regular alpha-fetoprotein measurements & ultra-sound examinations undertaken @ 6 monthly intervals.

3. **Budd-Chiari Syndrome**: A disorder of hepatic circulation, marked by venous obstruction, that leads to liver enlargement, ascites, extensive development of collateral vessels & severe portal hypertension.

4. **Viral hepatitis**: Prodromal symptoms may precede the development of jaundice by a few days to 2 weeks. They are the common manifestations of an acute infectious disease and include chills, headache and malaise. Gastrointestinal symptoms may be prominent; anorexia and distaste for cigarettes are frequent and nausea vomiting and diarrhoea may follow. A steady upper abdominal pain,
occasionally severe occurs as a result of stretching of the peritoneum over the enlarged liver. Initially physical signs are scanty; the liver is usually tender though not readily palpable, enlarged cervical lymph nodes may be found and splenomegaly may occur, particularly in children.

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**Case 19**

**Uterine Prolapse**

**Definition:**
Uterine prolapse is a descent or herniation of the uterus into or beyond the vagina.

In first-degree prolapse: the cervix remains within the vagina;
In second-degree prolapse: the cervix is at or near the introitus;
In third-degree prolapse (procidentia): most or all of the uterus lies outside the vaginal opening.
Uterine prolapse always is accompanied by some degree of vaginal wall prolapse. Often accompanied by protrusion of the bladder and front wall of the vagina (cystocele).

**Normal position of uterus in pelvic cavity and relations:**

**Relations**
- **Anteriorly**: vesicouterine pouch and superior surface of bladder
- **Posteriorly**: rectouterine pouch containing loops of intestine and anterior surface of rectum
- **Laterally**: peritoneal broad ligament and fascial transverse (cardinal) ligaments and ureters

**Support of uterus:**
Principal support of uterus holding it in this position are both active and passive.

*Active support*
Active support provided by pelvic diaphragm, which consists of levator ani and coccygeus muscles. Pelvic diaphragm stretches between pubis anteriorly and coccyx posteriorly, and from one lateral pelvic wall to another.
The levator ani is attached to the internal surface of the lesser pelvis and forms most of the pelvic floor.
Levator ani consists of:
- pubococcygeus
- puborectalis
- iliococcygeus

The levator ani also has important functions in voluntary control of urination, fecal continence (via puborectalis) and defecation. Weakness of these muscles could result in urinary incontinence.

**Nerve supply of the pelvis floor:**
- **Levator ani**: nerve to levator ani (branch of S4)
- **Coccygeus**: branches of S4 and S5

*Passive support*
Passive support provided by position of uterus; the way it is normally anteverted and anteflexed
- **Anteversion**: uterus bent forwards on backward sloping vagina
- **Anteflexion**: body of uterus angled further forwards on cervix

Because of this, the uterus ‘rests’ on top of the bladder. Hence, when intra-abdominal pressure increases, the uterus is pressed against the bladder instead of being pushed through the vagina, as would be the case if the uterus were upright over the vagina (as in cases of retroversion and retroflexion).

The cervix is the least mobile part of the uterus because of the passive support provided by the attached condensations of the endopelvic ligaments, which may also contain some smooth muscle.
These ligaments are:
- transverse cervical (cardinal) ligament
- uterosacral ligament

Together, these active and passive supports keep the uterus centered in the pelvic cavity and resist the tendency for the uterus to fall or be pushed through the hollow tube formed by the vagina.
Any weakness of the pelvic support, including musculature, ligaments, and fascia, could result in uterine prolapse.

**Innervation of uterus:**
Sensory fibres from uterus pass back to the central nervous system in autonomic nerves. The pathways are not clearly understood.
Uterine pain is felt in lower back and perineum.

**Causes Of Uterine Prolapse**

- Pregnancy or Childbirth – effect on pelvic floor muscles. Increased incidence in multi-parous women or women who have given birth to particularly heavy children
- Raised Intra-Abdominal Pressure – pressure placed on pelvic viscera, eg. Coughing, sneezing
- Fibroid Tumours – benign tumours of the muscle of the uterine wall, may be intramural, subdermal or submucosal
- Menopause – reduced oestrogen can cause reduction in ligament elasticity
- Congenital Factors – collagen deficiency (eg. Marfan Syndrome), ligaments are composed of collagen type 2.
- Race Associated Risk – more likely in white women than in black women, reason unknown

**Prolapses**

- Uterine – Slipping down of the uterus into the vaginal canal, Scale of 0 to 4 to classify it with 0 being no prolapse and 4 being severe,
- Cystocele – herniation of the bladder into the vaginal canal, usually results in stress incontinence, present either alone or along with other types in 80% of cases of prolapse
- Enterocele – herniation of the bowel through a defect in either the vesicouterine pouch (anterior) or the rectouterine pouch (posterior)
- Rectocele – herniation of the rectum (differs from rectal prolapse which is where the rectum falls out the anus), present in 40% of cases
- Cervicular – the top of the vagina, ie. the cervix, may also undergo prolapse, as seen in 32% of cases

**Symptoms**

- Vaginal pain and heaviness
- Feeling of something ‘coming down’ the vagina (as per case)
- Stress – Incontinence → Involuntary loss of urine due to a compromised urethral sphincter (Sphincter Urethrae and Pubovaginalis part of Levator Ani). Loss of the valve action of the neck of the bladder as it has fallen down and assumed a funnel shape.
- Pain during sexual intercourse
- Lower back pain – due to dermatomal distribution of L2
- Excessive vaginal discharge – due to vaginal infection
- Anal pain or pressure – due to rectocele. Faecal incontinence may also result
- Bladder Infections – due to cystocele. Urinary incontinence may also result
- Symptoms tend to worsen with activity but improve with bed-rest

**Examination / Diagnosis**

- Complete pelvic exam including rectovaginal examination to assess sphincter tone
- Sims Position / Speculum – exaggerated left lateral position with the knees pulled into the chest, C-Shaped speculum inserted into the vagina to enable the prolapse to be seen more clearly
- Empty Bladder / Standing Position – better than full bladder and supine position according to a study carried out in Ohio
- Physical findings may be enhanced by having the patient strain during the exam or stand/walk before examination in order to slightly enhance the degree of prolapse, allowing it to be better seen.
- Mild Uterine Prolapse may only be recognised when the patient strains during bi-manual examination
Imaging Techniques – X Ray Fluoroscopy, dye in vagina, rectum, bladder, enabling the shape of the uterus to be clearly seen.

Evaluate Estrogen status : due to effect on ligament elasticity.

- Signs of Decreased Estrogens: Loss of Rugae in vagina
- Decreased Secretions
- Thin Perineal skin/easily torn

**TREATMENT**

**Vaginal Hysterectomy**

1. Procedure performed to correct uterine prolapse.
2. A Hysterectomy is a surgical removal of the uterus.
3. A Vaginal Hysterectomy means surgical removal of uterus through the vagina.
4. One or both the Ovaries and fallopian tubes may be removed during the procedure.
5. The approach is normally chosen when the uterus is not greatly enlarged (large = approx 12-16 weeks size).
6. The procedure has fewer complications, shorter length of hospitalization and faster recovery compared to Abdominal Hysterectomy.

**Anterior Colporrhaphy**

1. Also known as Anterior Vaginal Wall Repair.
2. Most common utilized procedure for correction of cystocele.
3. An incision is made through the vagina to release a portion of the anterior vaginal wall.
4. The pubocervical fascia is folded and stitched to bring the bladder and urethra in proper position.
5. It can be done using general or spinal anesthesia.
6. A foley catheter may be put in place for 1 to 2 days after surgery.
7. However anterior colporrhaphy by itself does not treat stress incontinence and an additional procedure is required.
TREATMENT

**Pessary**
- Plastic device that fits into the vagina.
- Provides support to the uterus, vagina, bladder or rectum.
- It is often used as a treatment for uterine prolapse.
- It can also help if the patient has cystocele, rectocele, and/or stress urinary incontinence.
- Pessary comes in various shapes and sizes although the only way to fit it is through trial and error.
- Side effects: Increased vaginal discharge, vaginal discharge may develop an odor, and vaginal irritation.

**Pelvic Floor Strengthening Exercise**
- also known as Kegel exercises
- The principle is to strengthen the muscles of the pelvic floor.
- This can treat stress incontinence mainly but also to be known as a conservative treatment for uterine prolapse (1st degree).
- A vaginal cone might be used. It’s a weighted device that is inserted into the vagina. Pelvic floor muscles are contracted to hold the device in place.
- If unable biofeedback and electrical stimulation can be used.

**Medication / Hormonal Treatment**
- Medications used to treat stress incontinence aims to increase the contraction of the urethral sphincter muscle.
- These are Alpha-adrenergic agonist drugs, such as phenylpropanolamine and pseudoephedrine. Additionally, the tricyclic antidepressant imipramine has similar properties.
- Estrogen therapy can be used to improve symptoms of urinary frequency, urgency and burning in postmenopausal women.
- Estrogen is also used in conservative treatment in uterine prolapse.

**Surgery**
There are many types of surgery associated with uterine prolapse, cystocele and stress incontinence with some of them are performed at the same time.

1. Hysterectomy (removal of uterus)
   - Vaginal Hysterectomy
   - Abdominal Hysterectomy
   - Laparoscopic Assisted Vaginal Hysterectomy

2. Uterine Suspension (not removing the uterus)
   - Sacrospinous ligament suspension.
   - Abdominal Sacral Colpopexy
   - Uterosacral ligament suspension.

3. Anterior Repair/Colporrhaphy (cystocele)

4. Surgery for stress incontinence :-
   - Tension Free Vaginal Tape (TVT) sling procedure.
   - Retropubic Suspension
   - Artificial Urinary Sphincter (for males normally)
   - Collagen Injection
   - Needle Bladder Neck Suspension

Case 20

Let’s look at the overview of the gallbladder, the 3 parts of the gallbladder, the position, the relation to other viscera, nerve innervations that will explain the referred pain and variations of cystic ducts and accessory hepatic ducts that are important to take notice during surgery.
The gallbladder is a flask-shaped, blind-ending diverticulum attached to the common bile duct by the cystic duct. In life, it is grey-blue in colour and usually lies attached to the inferior surface of the right lobe of the liver by connective tissue. It usually lies in a shallow fossa in the liver parenchyma covered by peritoneum continued from the liver surface. This attachment can vary widely. At one extreme the gallbladder may be almost completely buried within the liver surface, having no peritoneal covering (intraparenchymal pattern); at the other extreme it may hang from a short mesentery formed by the two layers of peritoneum separated only by connective tissue and a few small vessels (mesenteric pattern).

The gallbladder is described as having a fundus, body and neck. The neck lies at the medial end close to the porta hepatis, and almost always has a short peritoneal covered attachment to the liver; this mesentery usually contains the cystic artery. The mucosa at the medial end of the neck is obliquely ridged, forming a spiral groove continuous with the spiral valve of the cystic duct. At its lateral end the neck widens out to form the body of the gallbladder and this widening is often referred to in clinical practice as 'Hartmann's pouch'. The neck lies anterior to the second part of the duodenum.

The body of the gallbladder normally lies in contact with the liver surface. It lies anterior to the second part of the duodenum and the right end of the transverse colon. The fundus lies at the lateral end of the body and usually projects past the inferior border of the liver to a variable length. It often lies in contact with the anterior abdominal wall behind the ninth costal cartilage where the lateral edge of the right rectus abdominis crosses the costal margin. This is the location where enlargement of the gallbladder is best sought on clinical examination. The fundus commonly lies adjacent to the transverse colon. The gallbladder varies in size and shape.
The cystohepatic triangle is also known as Calot's triangle. The near triangular space formed between the cystic duct, the common hepatic duct and the inferior surface of the liver, it is enclosed by the double layer of peritoneum which forms the short mesentery of the cystic duct. Since the two layers are not closely opposed, there is an appreciable amount of loose connective tissue within the triangle. This space usually contains the cystic artery as it approaches the gallbladder, the cystic lymph node and lymphatics from the gallbladder, one or two small cystic veins, the autonomic nerves running to the gallbladder and some loose adipose tissue. It may contain any accessory ducts which drain into the gallbladder from the liver. Appreciation of the variations in ductal and arterial anatomy as they relate to the triangle are of considerable importance during excision of the gallbladder in order to avoid mistakenly ligating the common hepatic or common bile duct.

The primary nerves that innervate the gallbladder originate from the celiac plexus (which provides sympathetic innervation), and the vagus nerve (provides parasympathetic). Sensory innervation is provided by the right phrenic nerve (C3, 4, 5). The stimulation of the right phrenic nerve is responsible for the referred pain to the right shoulder.

Variations of cystic ducts are very important to us during surgery. There are 3 common variations of cystic ducts and that is low union that result from short bile duct and lies posterior to the superior part of the duodenum or even inferior to it. The second type is high union. The cystic duct join the common hepatic duct near the porta hepatis and the third is swerving course where by the cystic duct spiral anterior over the common hepatic duct before joining it on the left side.

Accessory hepatic ducts also will cause complication to us if we do not take it in to account. Accessory hepatic ducts are common and are in positions of surgical danger. An accessory duct is a normal segmental duct that joins the biliary system outside the liver instead of within it. Leaking of the bile will occur if during surgery it is inadvertently cut.
Acute Cholecystitis

In 90% of cases, acute cholecystitis is caused by gallstones in the gallbladder, which obstruct the duct leading from the gallbladder to the common bile duct (which drains into the intestine). Severe illness, alcohol abuse and, rarely, tumors of the gallbladder may also cause cholecystitis.

The trapped bile becomes concentrated and causes irritation and pressure build-up in the gallbladder. This can lead to bacterial infection and perforation. Attacks may follow a large or fatty meal.

Gallstones occur more frequently in women than men, and it becomes more common with age in both sexes. Native Americans have a higher incidence of gallstones.

History

- 40 year old female
- spasmodic colicky pain in right upper quadrant (RUQ)
- radiating or referred pain to right shoulder
- nausea and vomiting
- fever over the past 24 hours

Physical Examination

- Look for vital signs
  1. heart rate – 110 bpm (normal 72)
  2. blood pressure - 110/70
  3. respiratory rate – 20 per minute (normal 14)
  4. temperature – 102 °F
These prove there is some sort of infection or inflammation

- Obese
- Shows positive Murphy’s sign (uncomfortable on deep inspiration, increase in pain while palpating the RUQ)

**Investigations**

- Ultrasound - reveals lodged gall stone within the cystic duct
  - thickened gall bladder wall, pericholecystic fluid and stones
- Endoscopic Retrograde Cholangiopancreatography (ERCP)
- Others:
  - WCC (should increase if there is infection)
  - Hepatic Immunodiacetic Acid cholescintigraphy (HIDA) to reveal a blocked cystic duct

**Differential Diagnosis**

1. **Gall stones** (ultrasound results, positive Murphy’s sign)
2. **High Retrocecal Acute Appendicitis** – ruled out through location of pain, rectal examination done to confirm, blood test for WBC count
3. **Infection of kidney / Pylonephritis** (common in females of this age, but ruled out because of ultrasound results, typical pain would be at the back and it radiates towards the loins / groins, urine tests also usually done to confirm diagnosis)
4. **Acute Pancreatitis** - could also present with similar signs and symptoms but the pain radiates to the left
   - usually confirmed with blood tests, serum amylase levels should be high
5. **Perforated Bowel / Peritonitis** - ruled out because BP is normal
   - BP should be low as the patient would be in shock

**Other Differential Diagnosis**

1. Intestinal obstruction
2. Perforated peptic ulcers
Diagnosis

- 4 F’s of gall stones – FAT, FERTILE, FORTY, FAIR
- Acute and chronic (2 types of presentation)
  - Acute – nausea, fever, vomiting, RUQ pain
  - Chronic – flatulent dyspepsia, intolerance of fatty food
Surgical treatment and therapy of cholecystitis

Initial treatment:
- nothing is given orally.
- stomach is emptied using a nasogastric tube.
- analgesia is administered, usually pethidine, a drug that reduces sensibility to pain without inducing unconsciousness.
- patient allowed to settle for 2-3 days – conservative management.
- emergency or elective surgery (3 months later).
- bladder is catheterised.
- general anesthetic applied.
Laparoscopic cholecystectomy:

- first performed in 1987, nearly 20 years ago.
- surgery to remove a diseased gallbladder through a fiberoptic scope that is inserted into a small incision near the navel.
- reduces scarring – allows doctor to see into the patients abdomen without making a large incision.
- reduces recovery time and pain associated with traditional methods – return home next day.
- use of endoscopic video equipment – transmits images to a monitor.
- peritoneal cavity must be distended to make room for lighting and the manipulation of the surgical instruments – Carbon dioxide used.
- In event of complications, patient is prepared for an open cholecystectomy.
- 4 small incisions to make room for laparoscope and other instruments such as forceps and electrocautery devices – destruction of diseased tissue by needle or snare that is electrically heated.
- Trocars also inserted – device combined with a cannula to drain fluids from cavity – has a removable shaft.
- Blunt forceps put on fundus of gallbladder – pushes right lobe of liver towards diaphragm – another is used to expose and dissect the gall bladder and porta hepatis.
- Dissections begins along the sides and proceeds to the union of the cystic and common hepatic ducts.
- Cystic artery is located in the cystohepatic triangle of Calot - dissected freely before the cystic duct is divided.
- Arteries and duct ligated.
- Gall bladder opened and gallstones removed.
- Collapsed gall bladder removed.
Open cholecystectomy:
- Bigger scar.
- Subcostal incision – tip of xiphoid process paralleling the costal margin.
- Split rectus sheath and related abdominal muscles – rectus abdominis, external oblique, internal oblique, transversalis fascia, fat and the peritoneum.
- Preserve intercostal nerves by retracting them.
- Gall bladder is identified – thick walled, contracted by scarring – walled off with gauze pads.
- The peritoneal attachment of the gall bladder to the liver and is incised – gall bladder is bluntly and sharply dissected.
- Drain inserted for gastric suction.

Post-operatively:
- given glucose and intravenous saline solution.
- gastric drain inspected for bile leakage.
- removed after three days.

Different types and complications of cholecystitis

Chronic cholecystitis:
- may be open or laparoscopic.
- control symptoms – 1) low-fat diet and 2) shock wave lithotripsy, ultrasound waves or x-rays to destroy stones.

Biliary colic:
- same applies – cholecystectomy.
- impaction of stone on neck of gallbladder.

Mucocele:
- cholecystectomy.
- follows biliary colic and stone impacts on neck, mucus secretion continues.

Empyema:
- infection after impact on neck.
- gall bladder fills with pus.
- same initial treatment as laparoscopy – too much infection to carry out safe surgery – gall bladder must be drained.

**Perforation of the gall bladder:**
- generalised biliary peritonitis.
- leakage of pus.
- high mortality rate.
- laparotomy.
- antibiotics given after surgery for following weeks – extensive course.

**Cholangitis:**
- complete obstruction – ascending infection in the biliary tree.
- severe jaundice.
- antibiotics – cannulise bile ducts to decompress them.

**Complications**

(1) significant post-op haemorrhage from abnormal blood vessels – cystic artery in triangle of Calot – requires immediate reoperation.

(2) adhesions – the result of any intrusion of the peritoneal cavity.


**Perineum**

The perineum is the area between the vagina and the anus. The Osseo fibrous structures making the boundaries of the perineum are:
1. Pubic symphysis anteriorly
2. Inferior pubic rami and the ischial rami anterolaterally
3. Ischial tuberosities laterally
4. Sacrotuberous ligament posterolaterally
5. Inferiormost sacrum and coccyx

A transverse line joining the anterior ends of the ischial tuberosities divides the perineum into two triangles:
1. The anal triangle, containing the anus, is posterior to this line.
2. The urogenital triangle, containing the external genitalia, is anterior to this line.

The perineal body, which is the site for attachment for the perineal muscles, is an irregular fibromuscular mass located in the median plane between the anal canal and the perineal membrane. It is variable in size and consistency. The perineal body is the major structure incised during medial episitomy for childbirth.

There are two perineal pouches, a superficial and a deep pouch.

1. The superficial perineal pouch contains:
   - Root of the clitoris and the muscle associated with it
   - Bulb of the vestibule and surrounding muscle
   - Superficial transverse muscles
   - Related vessels and nerves
   - Greater vestibular glands

2. The deep perineal pouch contains:
   - Proximal part of the urethra
   - Eternal urethral sphincter
   - Deep transverse perineal muscles
   - Related vessels and nerves

**Parturition**
Involves 2 processes:
1. Coordinated contractions of the myometrium
   - by positive feedback mechanisms of hormones such as oxytocin and prostaglandins.
2. Relaxation of cervix
   - relaxation of cervix, pelvic ligaments & pubic symphysis, also increased dilation of cervix by prostaglandins and relaxin hormones.

**Phases of Labor**
1. 1st stage: the longest stage (esp. for first pregnancies); onset of labor; regular painful contractions; rupture of amnion; ends with full dilatation (~10cm)
2. 2nd stage: the expulsion stage; delivery of fetus; crowning occurs where the largest dimension of the baby’s head is distending the vulva.
3. 3rd stage: the expulsion of the placenta from the uterus; sometimes aided by administration of oxytocic drugs.

EPISIOTOMY
A surgical incision made in the perineum to enlarge the vaginal orifice to assist in delivery of a baby.

Why is it done?
- To prevent damage to perineal structures such as perineal body, external anal sphincter and rectum due to uncontrolled tearing of perineum.
- To ease delivery in difficult birth such as breech, forceps and ventouse deliveries; if the baby is large; extreme maternal fatigue; the baby’s in distress
- If the perineal tissue looks fragile- it starts to bleed or doesn’t stretch well during crowning; tearing is unavoidable

During birth, especially during crowning the baby (crown and body) will stretch the perineum, levator ani muscles (esp. pubococcygeus) and pelvic fascia. The pubococcygeus will be at risk of tearing and damage to this muscle will lead to urinary stress incontinence post delivery.

DEGREES OF EPISIOTOMY
1st degree: Incision at skin level only
2nd degree: Incision involves skin and muscles (most common)
3rd degree: Incision involves skin, muscles and rectal sphincter
4th degree: Incision involves skin, muscles, rectal sphincter and anal wall (episiorectoprotomy) least common

TYPES OF EPISIOTOMY
1. Midline episiotomy
   - Incision is made from the vaginal orifice straight down towards the anus.(between the vagina and the anus); usually doesn’t extend to the anus. If it does extend to the anus it is called episioirectoprotomy (4th degree episiotomy).
2. Posterolateral/ Mediolateral episiotomy
   - Incision is made from the vaginal orifice down and laterally at an angle; slanting away from the anus.
A mediolateral episiotomy is preferred because in cases where further tearing, a mediolateral incision won’t extend to the perineal body and rectum.

PROCEDURE
1. The doctor should ask for the mother’s permission before performing an episiotomy.
2. A local anesthetic (Lignocaine 1% 5-10mls) will be administered by injection. If the mother has had an epidural anesthetic this is not necessary. Sometimes a pressure episiotomy is done where no pain relief is given. This happens when the perineum is already quite numb and thinned out by the pressure of the baby’s head.
3. The doctor will use a pair of sterile surgical scissors and cut the perineum. The incision will be about 1 to 3 inches. The layers of tissue incised are
   - Skin and subcutaneous tissues
   - Vaginal mucosa
   - The urogenital septum (mostly fascia but also transverse perineal muscles)
   - Intercolumnar fascia or superior fascia of pelvic diaphragm
   - The lowermost fibres of puborectalis muscle (if episiotomy is mediolateral and deep)
4. Baby is delivered, and the placenta after the baby.
5. Repair of the episiotomy; the doctor will check the area for tears and will proceed to close the incision. Sometimes, another anesthetic will be given before closure. The incision will be closed with absorbable stitches. The stitches will dissolve in about 10 days.

Other methods of delivering babies:
1. Induction method: where birth is induced by hormonal input or physically rupturing the amnion.
2. Caesarean section: removal of baby by operation. An incision is made in the abdomen and the baby is removed.
3. Forceps method: Metal tongs with 2 large spoon shaped edges inserted into the vagina to grip the baby’s head and pull the baby out. Usually requires an episiotomy.
4. Ventouse/Vacuum extraction: A metal plate or a cone-shaped cup of synthetic material. Cup placed over baby’s head and the vacuum created pull baby out.

**Complications of Episiotomy.**

There are very few serious complications associated with this procedure as it is a surgical procedure carried out in hospital and was almost a routine occurrence until recently. Modern ethics dictates that the mothers’ permission must be sought before such a procedure can be preformed.

1. **Infection and poor wound healing.**
   This occurs in 1-8% of childbirths anyway and can be very serious if not taken care of. It can lead to sepsis, septic shock, infertility, scarring and even death. Septicemia can be passed from mother to child in severe cases of infection. This complication is usually treated with antibiotics.

2. **Incontinence for stool and flatus.**
   This occurs with 3rd or 4th degree lacerations- i.e. those that extend as far as the external anal sphincter. This can lead to complete incontinence or stress incontinence. Stress incontinence is brought on when the mothers intra-abdominal pressure is raised such as when sneezing or bearing down. Incontinence can be ameliorated by surgically overlapping the torn ends of the external anal sphincter or by sphincteroplasty of muscle transposition. Muscle transposition is using the gracilis or glutaeus maximus muscles to create a new sphincter while sphincteroplasty is the creation of a new sphincter de novo which is usually controlled by an external low voltage source.

3. **Rectovaginal fistulas.**
   This is a VERY rare complaint-.1-.5% of episiotomies and consists of the formation of a connecting passageway between the vagina and the rectum. It is corrected surgically and is usually the result of perineal layers not healing in the correct fashion.

4. **Discomfort and excessive blood loss.**
   As an episiotomy is a surgical procedure there will be some swelling in the area for 2-3 weeks and this can lead to severe discomfort, not to mention the discomfort/ embarrassment of
incontinence. There is also blood lost from the area after an episiotomy due to the good arterial supply to the area from the Internal, Deep and Superficial pudendal arteries. Dyspareunia can also be a problem but this usually abates after a few weeks. Treatment is Sitz baths (sitting in a bath of disinfectant to maintain hygiene and ease pain) and pain medication.

Possibly the most important factor in recovery after an episiotomy is to maintain hygiene as much as possible to prevent infection and impaired healing.

It must also be borne in mind that the effects of an episiotomy are usually not immediate as incontinence may set in years later after ageing and further childbearing have also taken their toll on the body, this is especially true after damage to the perineal body as this is the centre of support in the female perineum and damage to it leaves the perineum weak and susceptible to further injury.

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CASE 22:

TERRI MCVEIGH
SITI HASMAH MOHD JADIT
MAHMOUD ALBLOUSHI

ANATOMY:

The Femoral Triangle

trigonum femorale; Scarp’s triangle

The femoral triangle is, as the name suggests, a triangular shaped region. It is located in the superior third of the thigh, on its medial aspect. It appears as a depression inferior to the inguinal ligament when the thigh is in an abducted, flexed and laterally rotated position. The boundaries of the Femoral Triangle are:

Superiorly: The inguinal ligament
Medially: medial border of the Adductor Longus muscle
Laterally: Sartorius muscle

Its floor is muscular and gutter shaped, formed from medial to lateral by adductor longus, pectineus and ilioptoas muscles. The skin, subcutaneous tissue and fasciae of the thigh (fascia lata, cribiform fascia), form the roof of the triangle.

The contents of the femoral triangle are, from medial to lateral:

1. **Femoral vein and its tributaries** (e.g. long saphenous vein, deep veins)
2. **Femoral artery and some of its branches**
   *The femoral artery and vein bisect the femoral triangle, entering (artery) and leaving (vein) the adductor canal at its apex, where sartorius crosses over adductor longus.*
3. **Femoral sheath and contents**
   *The femoral sheath is a funnel shaped fascial tube extending inferior to the inguinal ligament. It encloses the proximal parts of the femoral vessels and femoral canal (the main pathway for lymphatic vessels from the lower limb to the abdomen) but it does not enclose the femoral nerve.*
4. **Femoral nerve and its branches**
   - Also palpable in the femoral triangle is the horizontal group of superficial inguinal lymph nodes lying in superficial fascia, just inferior and parallel to the inguinal ligament.

**The Femoral Vein**

The femoral vein passes through the adductor canal, at first posterolateral, and then posterior to the femoral artery. On entering the femoral sheath it is lateral to the femoral canal, terminating posterior to the inguinal ligament to become the external iliac vein.

Its course, simply put, is through the femoral triangle, through the adductor canal, to end in the adductor hiatus. The femoral vein is a continuation of the popliteal vein proximal to the adductor hiatus, formed by the union of anterior and posterior tibial veins and peroneal vein, and it is joined by the small saphenous vein higher up in the leg. As it ascends through the thigh, it receives its tributaries: the medial and lateral circumflex veins. It also receives the deep femoral vein, inferior to the termination of the long saphenous vein and the inguinal ligament. It receives the long saphenous vein in the inferior femoral triangle.

The external iliac vein is one of two tributaries of the common iliac vein, which along with the corresponding common iliac vein on the other leg, empties into the inferior vena cava, and then into the heart. In our case, the catheter follows this course: through the femoral vein, up through the external iliac vein, through the common iliac vein, up the inferior vena cava, and into the right atrium of the heart.
The Inguinal Region

The inguinal ligament is attached between the anterior superior iliac spine and to the pubic tubercle. It lies deep to the skin fold in the groin region and is palpable along its length. The pubic tubercle is also palpable on the upper border of the pubis bone. On the male, it is palpable by invagination of the scrotum, and in the female, it can be felt through the lateral margin of the labium major. The pubic crest is the ridge of bone medial to the pubic tubercle. Also palpable in the inguinal region is the upper margin of the symphysis pubis (this is a cartilaginous joint lying in the midline between the bodies of the pubic bones) and the bodies of the pubic bones, in the lower part of the anterior abdominal wall.

Why use the femoral vein for catheterization?

The femoral veins has several advantages over other possible locations from many procedures involving catheterization:

1. They are large vessels and permit relatively large catheters.
2. They do not present the same risks as catheterization of the neck. e.g. a misplaced needle in the neck could enter the pleural space and result in pneumothorax, and in the carotid arteries, there are the extra risks of thrombosis or artheroembolism caused by puncture of the arteries, which could lead to a stroke.

Thus, femoral catheterization is typically used for procedures requiring access to the circulation around the heart and for procedures in the abdomen.

Risks associated with catheterization in the groin region?

There are also, as with any procedure, risks involved in femoral catheterization, including:

1. Damage to the femoral artery or vein depending on which one is used for the catheterization. This could lead to an internal hemorrhage.
2. Injury to the femoral nerve (because of its close relation to the femoral vessels)
3. Needle introduction to the peritoneal cavity
4. Formation of an arteriovenous fistula (where the puncture made by the needle does not close)

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DIAGNOSIS, TESTS AND DIFFERENTIAL DIAGNOSIS

HISTORY:

- 65 year old female
- Cardiac catheterization/ femoral vein cannulation
- Painful throbbing in right groin
- Numbness, tingling in right anteromedial thigh and leg
- Mass in the right groin
- Right leg felt cool
- Distal pulse in the leg absent

TESTS

- CBC, ESR→haematoma: low Hb & reduced platelets
- Colour Duplex Ultrasonography
- Physical examination
- Electrodiagnostic testing-EMG and NCS
DIAGNOSIS:
HAEMATOMA AND FEMORAL NERVE NEUROPATHY

SYMPTOMS:
Femoral nerve neuropathy:
- Haematoma
- Pain in inguinal region
- Numbness, tingling in right anteromedial thigh and leg
- Distal pulse absent

DIFFERENTIAL DIAGNOSIS:
- Arteriovenous fistula- Colour Duplex US, warm lower extremity
- Femoral Hernia- physical exam,
- Pseudoaneurysm- Colour Duplex US
- Lymphoedema- lymphoscintigraphy

Femoral Neuropathy is usually caused by diabetes and trauma to inguinal region which can cause entrapment to the nerve as well as being a complication associated with femoral artery injury due to needle puncture in medical procedure such as angiography and cardiac catheterization via femoral vein such as in this case.

The symptoms of a femoral neuropathy may include pain in the inguinal region that is partially relieved by flexion and external rotation of the hip and dysesthesia over the anterior thigh and anteromedial leg. Patients complain of difficulty with walking and knee buckling depending on the severity of the injury. The nerve gives rise to the saphenous nerve in the thigh; therefore, numbness in this distribution can be present. Anterior knee pain may also be present due to the saphenous nerve supply to the patella.

Tests that can be use to diagnose this condition is complete blood count(CBC) and erythrocyte sedimentation rate (ESC)- presence of hematoma will give low haemoglobin count and reduced platelets.

On examination, patients may present with weak hip flexion, knee extension, and impaired quadriceps tendon reflex and sensory deficit in the anteromedial aspect of the thigh. Pain may be increased with hip extension and relieved with external rotation of the hip. If compression occurs at the inguinal region, no hip flexion weakness is present. Sensory loss may occur along the medial aspect of the leg below the knee (saphenous distribution).

Electrodiagnostic testing namely electromyelogram and nerve conduction study is typically performed for diagnosis but is also important to determine the extent of the injury and to determine prognosis of recovery. With electrodiagnostic testing, either surface or needle electrodes lateral to the femoral artery in the inguinal region is used for stimulation. The stimulation can be performed above and below theinguinal ligament. Disk electrodes from the vastus medialis are used to record stimulation. Next is the use of colour duplex ultrasonography which is a form of ultrasound use to evaluate blood flow in the vessels.

A saphenous nerve sensory study may also be performed (continuation of the sensory portion of the femoral nerve over the medial aspect of the leg and ankle). Needle examination should be completed for the paraspinal muscles as well as the iliopsoas (also L2-3) and hip adductors supplied by the obturator nerve to determine the presence of root or plexus injury versus peripheral nerve injury. The needle EMG is usually the most revealing portion of the electrodiagnostic test. The examiner must look not only for denervation potentials, but also for any active motor units.

The symptoms of femoral neuropathy as result of catheterization are presence of hematoma; observed as mass at the right groin which is due to inability for the pucture of femoral vein or more likely the artery to close. The numbness and absence of distal pulse is due to compression of hematoma to the nerve, artery and vein.
Differential diagnosis includes arteriovenous fistula and pseudoaneurysm, both are common complication of cardiac catheterization. The diagnosis through colour duplex ultrasonography is most important in distinguishing between simple hematoma, arteriovenous fistula and pseudoaneurysm. Femoral hernia can be excluded in which it can be diagnose through means of physical examination and lymphoedema through lymphoscintigraphy. Pseudoaneurysm is a leakage of blood from an artery into the surrounding tissue with persistent communication between the originating artery and the terminating blood filled cavity. The leakage can result from an artery being punctured during a diagnostic test such as cardiac catheterization. Lymphoedema is abnormal buildup of fluid casing swelling, most often in the arms or legs. The condition develops when the lymph vessels or nodes are impaired or damaged. Lymphoscintigraphy is an intradermal injection of sulphur colloid and serial gamma imaging. It allows visualizations of selected regional lymphatic drainage.

HEMATOMA

Definition:
A localized collection of blood, usually clotted, in an organ, space, or tissue, due to a break in the wall of a blood vessel.

What Causes Hematoma?
- Injuries resulting from accidents or falls.
- Trauma to the blood vessels that might lead to an internal bleeding.
- Chronic use of aspirin, anti-inflammatory drugs (Ibuprofen) or blood thinning (anti-coagulant) medication, chronic heavy alcohol use.
- Diseases associated with blood clotting problems.

Types of Hematomas:
A. Simple (Familiar) types:
   - Bruises
   - Black Eyes
B. Serious types:
   - Epidural Hematoma (bleeding in space between dura & skull)
   - Subdural Hematoma (bleeding in space between dura & brain)
C. Less-serious types:
   - Subungual Hematoma (under fingernail or toenail)
   - Hematoma Auris (in the tissue of the outer ear)
   - Perianal Hematoma (under the skin around the anus)

Treatment:
A. For Simple types of Hematoma (Bruises):
   - Initially applying ice or cold packs a few times a day.
   - If possible, elevate or rest the bruised limb.
   - If the area is still painful after about 48 hours, apply gentle heat with warm towels, a hot water bottle, or a heating pad.
   - Pressure in the form of an elastic adhesive bandage may be helpful to reduce hemorrhage and swelling.

B. For serious types of Hematomas:
Hematomas that occur intracranially require immediate specialized medical attention. Surgery is often required.
The surgery will include:
   - Drilling small holes in the site of Hematoma.
   - Allowing blood to be drained and relieving the pressure it causes.
   - Cleaning the area from any solid blood clots that may have occurred.
   - Suturing the damaged blood vessels. (Stitches, using electrical current to generate heat which seals off blood vessels, or using a topical agent).
   - Rehabilitation services may be required.

C. For less-serious types of Hematomas:
In cases like subungual hematomas, or any other smaller Hematomas, surgery may not be required to remove the blood.
Case 23

Fracture of the neck of femur

Sheila Bingkasan

Bushra Megat Johari

Colin o Rourke

Anatomical relevance

The hip joint is a ball and socket joint. The upper end of the femur is formed into a round ball. A cavity in the pelvic bone forms the socket (acetabulum). The ball is held in the socket by very powerful ligaments such as the iliofemoral, pubofemoral and the ischiofemoral ligaments. The capsule is lined by the synovium. The head of the femur and the socket is covered by cartilage. The cartilage cushions the joint, and allows the bones to move on each other with very little friction.

The joint capsule of the hip extends from the acetabulum to the intertrochanteric line anteriorly and to the junction of the middle and distal thirds of the femoral neck posteriorly. Femoral neck fractures are therefore intracapsular injuries.

The muscles of the hip joint are the iliopsoas, adductor longus, brevis and magnus, gluteus medius and minimus, sartorius, gluteus maximus, obturator externus and internus, superior and inferior gemelli and the piriformis.

The femur has a head, a neck, a shaft, and two condyles. It articulates with the acetabulum, the articular cup on the pelvis superiorly, and with the tibia and patella inferiorly. It forms part of the hip and part of
the knee. The greater trochanter and the lesser trochanter have two bony projections for muscle attachment. Posteriorly, gluteus maximus attaches to the gluteal tuberosity. The biceps femoris attaches to the linea aspera.

The femur is supplied by the extracapsular arterial ring, the ascending cervical branches of the arterial ring and the arteries of the ligamentum teres.

The extracapsular arterial ring is formed by the medial femoral circumflex artery and by a branch from the lateral femoral circumflex artery. The ascending cervical branches ascend on the surface on the femoral neck anteriorly. The lateral vessels are the most vulnerable to injury in femoral neck fractures.

A second ring of vessels is formed as the ascending cervical vessels approach the articular margin of the femoral head. From this, the epiphyseal arteries are formed. The lateral epiphyseal arterial group supplies the lateral weight-bearing portion of the femoral head. The epiphyseal vessels are joined by the inferior metaphyseal vessels and vessels from the ligamentum teres.

**FRACTURES OF THE FEMORAL NECK**

There are two broad groups of fractures in the neck of femur which are intracapsular fractures and extracapsular fractures. As the name implies, intracapsular fractures occur within the fibrous capsule while extracapsular fractures occur outside the fibrous capsule.

There are three kinds of intracapsular fractures which are named in accordance to the level of the fracture line in the femoral neck;

- **Subcapital**
  The fracture line passes across the head of femur.

- **Transcervical**
  The fracture line passes across the neck of femur just distal to the head of femur.

- **Basal (Basicervical)**
  The fracture line passes across the neck of femur just proximal to the trochanters.

Avascular necrosis is a common complication of the femoral neck fractures. When the hip fracture occurs through the neck of the femur and the ball is completely displaced, there is a very high chance that the blood supply to the femoral head has been damaged. This makes it very likely that avascular necrosis (AVN) will occur as a complication of this type of hip fracture. Avascular necrosis of the femoral head causes the bone of the femoral head to die. The bone of the femoral head begins to collapse weeks later--causing more problems in the months to come. This will most likely result in a second operation several months later to replace the hip due to the avascular necrosis.
There are different types of degree of severity of the fracture which can be classified into four types which are;

Type 1 - incomplete fracture without displacement
Type 2 - complete fracture without displacement
Type 3 - complete fracture with partial displacement
Type 4 - complete fracture with full displacement

There are 2 kinds of extracapsular fractures of the femoral neck;

- **Subtrochanteric**
  The fracture line is across the proximal shaft of femur just inferior to the trochanters.

- **Intertrochanteric**
  The fracture passes along, or close to a line between the greater and lesser trochanters and can often be comminuted.

In our case, physical examination reveals that her right leg is externally rotated and she is unable to lift her right leg. The right leg seemed shortened, which is confirmed by measuring the distance between the anterior superior iliac spine and the distal tip of the medial malleolus of the tibia. On palpation, there is tenderness in the femoral triangle in front of the hip joint.

The shortening of the leg is caused by the force of the injury that may drive the distal fragment superiorly. But in general, it is the muscle pull that leads to this result. If the neck of the femur is broken, the strong muscles of the thigh, including the rectus femoris, the adductor muscles, and the hamstring muscle, pull the distal fragment upward, so that the leg is shortened. The gluteus maximus, piriformis, obturator internus, the gemelli and the quadratus femoris rotate the distal fragment laterally, which results in external rotation of the leg. Muscle pull leads to a change in the angle of femoral head and neck with the shaft. The x-rays in our case reveal that the angle is reduced, resulting in a condition called **coxa vara**. Coxa vara causes a mild shortening of the lower limb and limits passive abduction of the hip.

As with any suspected femoral neck fracture, anteroposterior and lateral radiographs are taken. This procedure confirms the diagnosis and demonstrates the fracture just below the head of the femur, which is **intracapsular transcervical type 4 fracture**.

There are many risk factors that can be associated with fractures of the femoral neck. These include:
Age: The rate of fractures increases with age. As you age, your bone density decreases, your vision and sense of balance also decline, and your reaction time slows down. If you're inactive, your muscles tend to weaken as you age. The combination of all these factors increases your risk of having a fracture.

Chronic medical conditions: Osteoporosis is one of the biggest risk factors for femoral neck fracture. In osteoporosis, the structure of your bones becomes weaker because your bones don't contain as much calcium and other minerals. A weaker structure makes your bones more prone to a fracture, even with minor trauma.

Gender: Women lose bone density at a faster rate than men do. The drop in estrogen levels that occurs with menopause accelerates bone loss, increasing the risk of femoral neck fractures.

Heredity - Genetic factors influence bone size, bone mass and bone density. A family history of osteoporosis or fractures later in life is a strong predictor of low bone mass, although not necessarily of fractures themselves.

Nutrition - Lack of calcium and vitamin D in your diet when you're young lowers your peak bone mass and increases your risk of fracture later in life. Serious eating disorders such as anorexia nervosa and bulimia can damage your skeleton by depriving your body of essential nutrients needed for bone building.

Tobacco and alcohol use - Smoking and excessive consumption of alcohol can interfere with the normal processes of bone building and remodeling, resulting in bone loss. These habits also interfere with the production of estrogen and testosterone, two hormones that contribute to bone mass. In addition, smokers tend to enter menopause earlier than non smokers do.

INTRODUCTION TO TREATMENT

Fracture neck of the femur is a common injury. It commonly requires surgery for good results. The surgery needs to be precise to avoid potential complications. Most if not all fractures should be surgically treated such that the patient can be up and about on their fractured legs. Transcervical fractures are commonly treated by replacement in the elderly and fixation in the young. The replacement can be partial when only the head and the neck of the femur are replaced (hemiarthroplasty) or total when the acetabulum is replaced aswell (Total hip replacement)

HEMIARTHROPLASTY

Credentials of hemiarthroplasty (HA) as a treatment modality for displaced transcervical fractures in the elderly have been under cloud since the advent of newer types and designs of bipolar and total arthroplasties. It has been a common fact that the more “active” patients often complain of pain early in their postoperative period. Hemiarthroplasty has been advocated as the best treatment for transcervical
fractures in the elderly, for early rehabilitation, by permitting early mobilization and preventing the
dreaded complications of non-union and avascular necrosis. Currently two types of endoprothesis are in
use - the Thompson type and the Moore type. The main differences between the different
hemiarthroplasties are the design of the stem, the use of cement, and whether a second articulating joint
is included within the prosthesis (bipolar).

AUSTIN-MOORE PROSTHESIS

The Austin-Moore prosthesis should be reserved for patients more than 65 years of age and those who
are less active or debilitated because of other factors, this is because of increased acetabular wear with
time in the younger individual.

The Austin-Moore prosthesis replaces the head of the femur but also has a long stem that is inserted into
the bone marrow cavity almost halfway down the femoral shaft to anchor the head. The stem of the
prosthesis is fenestrated, and bits of cancellous bone, removed from the upper end of the femur, are
placed in these windows as bone grafts. It is expected that these bone chips will become converted into
dense new osseous tissue, locking the prosthesis in place and lending strength and stability to the
femoral shaft for its weight-bearing function.

It is a good option for the elderly and/or poor risk patients with a limited life expectancy. It has a low
intraoperative mortality, a low percentage of re-operation and results in early and good mobilization.
However early loosening with associated pain and poor function with an uncemented implant may
motivate the surgeon to choose a cemented implant.

THOMPSON PROSTHESIS

There are a few key differences between the Thompson and Austin-Moore prosthesis, notably the stem
in the Thompson model is not fenestrated and cement is commonly used to fixate it.

According to studies fixation of the prosthesis with cement might improve the outcome, resulting in
better pain relief and better gait than fixation of prosthesis without cement. Consequently cemented
prosthesis may give a better outcome but because of under-reporting of outcomes and the small number
of patients involved, no definite conclusions can be made.
TOTAL HIP ARTHROPLASTY

Total hip arthroplasty in selective cases of acute femoral neck fractures may provide consistent pain relief and a good functional outcome without much increase in complications. In this type of replacement the acetabulum as well as the femoral head is replaced to provide a totally new articulating surface. The advantages of this operation is that is that the results are longer lasting and provide a virtually pain free hip. The disadvantage is its higher cost and the inability of the patient to squat and sit cross-legged postoperatively. However in a well chosen patient, the advantages outweigh the disadvantages significantly.

TREATMENT

Our patient is 75 years of age and the Austin-Moore prosthesis appears to be the most suitable treatment because its advantages outnumber those of the Thompson prosthesis, bipolar or total hip arthroplasty.

PROCEDURE

(For Austin-Moore prosthesis)

With patient under spinal anesthesia the hip is approached by an inferolateral skin and fascial incision through the lower part of the buttock. The iliotibial tract is identified, and the gluteus maximus is split in the direction of its fibers by blunt dissection. A 5cm-long vertical incision is made to separate the gluteus maximus from its insertion into the iliotibial tract to give better access to the posterior aspect of the hip joint.

The upper and lower portions of this muscle are separated and retracted, thus exposing the sciatic nerve and the lateral rotators of the hip. The lateral rotators are divided close to their insertion into the greater trochanter. After removal of overlying fat, the capsule is incised and partly reflected. The head of the femur is dislodged out of the acetabulum and removed, and the ligament of the head is ligated and excised.

The neck of the femur is sawed across, and the stem of a properly fitted vitallium prosthesis is inserted into the narrow cavity (care is taken to preserve the normal forward angle of head and neck). A tight fit of the prosthesis is obtained after moving the artificial head into place.

Operation lasts 45-50mins and patient is able to sit up in bed and eat the afternoon of the day of the operation.

RECOVERY

The patient will undergo physical therapy in the week following the operation. Generally the patient is not in the hospital for greater than 10 days and is able to walk with assistance the day after the operation.
COMPLICATIONS

The main complications are the dislocation of the joint and the inflammatory response. The dislocation of the joint is because the head of the new prosthesis is smaller than that of the head of the femur and can be easily dislodged if certain movements are carried out. Inflammatory response is due to particles of the new prosthesis breaking away and being absorbed by surrounding tissues, causing inflammation.

Case 24

This case is about neural complications of intramuscular injections. The patient was given antibiotics via intramuscular injections to the buttocks. Following one of these, he walked with a limp that had not been there previously. Based on his symptoms, we decided that the injection had damaged the superior gluteal nerve on the right side.

An inappropriately administered intramuscular injection in the gluteal region could also damage the sciatic nerve, the inferior gluteal nerve, superficial branches of the superior gluteal artery and vein and the inferior gluteal vessels.

The gluteal region or buttock is an area encompassed by the gluteal fold inferiorly, a line joining the greater trochanter and the anterior superior iliac spine laterally, the iliac crest superiorly and the midline medially. It consists of a large bulk of skeletal muscle, covering large and vulnerable neurovascular structures.

The gluteal muscles consist of:

- Three large glutei, (maximus, medius and minimus) which are mainly extensors, abductors and medial rotators of the thigh.
- A deeper group of small muscles, (piriformis, obturator internus, gemelli and quadratus femoris) which are covered by the inferior half of the gluteus maximus and are lateral rotators of the thigh.

Gluteus medius, gluteus minimus and tensor fasciae latae are all innervated by the superior gluteal nerve and so have been weakened by the injection. Gluteus minimus is a fan-shaped muscle that originates from the external surface of the expanded upper part of the ilium, between the inferior and the anterior gluteal lines. The muscle fibres converge inferiorly and laterally to form a tendon, which inserts onto the anterolateral aspect of the greater trochanter. Gluteus medius overlies gluteus minimus and is also fan-shaped. It has a broad origin from the external surface of the ilium between the anterior and posterior gluteal lines and inserts onto the lateral surface of the greater trochanter of the femur.
These two muscles abduct the lower limb at the hip joint and reduce pelvic drop over the opposite swing limb during walking by securing the position of the pelvis on the stance limb. Tensor fasciae latae lies on the lateral side of the hip, enclosed between two layers of fascia lata. It is primarily a flexor of the thigh; however, it does not generally act independently.

There are two important foramina in the gluteal region, the greater and lesser sciatic foramen. The greater sciatic foramen is formed by the conversion of the greater sciatic notch of the hip bone into a foramen by the presence of the sacrotuberous and sacrospinous ligaments.

Several nerves arise from the sacral plexus and enter the gluteal region from the pelvis through the greater sciatic foramen. These are the superior gluteal nerve, sciatic nerve, nerve to the quadratus femoris, nerve to the obturator internus, posterior cutaneous nerve of thigh, pudendal nerve and inferior gluteal nerve.

Of all the nerves that pass through the greater sciatic foramen, only one, superior gluteal, passes superior to the piriformis. This is the nerve damaged in this case. It originates in the sacral plexus and carries contributions from the anterior of L4 to S1. After entering the gluteal region, the nerve loops up over the inferior margin of gluteus minimus and medius muscles. It supplies branches to these muscles and terminates by innervating the tensor fasciae latae.

Two arteries enter the gluteal region from the pelvic cavity, the inferior gluteal artery and the superior gluteal artery. They arise from the internal iliac arteries and enter through the greater sciatic foramen along with their corresponding nerves and pass inferior and superior to the piriformis.

Inferior and superior gluteal veins follow the inferior and superior gluteal arteries into the pelvis where they join the pelvic plexus of veins. Peripherally, the veins anastomose with the superficial gluteal veins, which ultimately drain anteriorly into the femoral vein.

**Causes of a Positive Trendelenburg’s Test**

**Weak Abductors**
- Injury to superior gluteal nerve
- Wasting and pain at hip joint as a result of osteo or rheumatoid arthritis
- Paralytic Poliomyelitis

**Damaged Hip**
- Fractures to head/neck of femur (e.g. Stress fractures)
- Damage to acetabulum

**Absent/Dislocated Hip**
- Congenital Dislocation of the Hip (Developmental Dysplasia of the Hip)
- Tom Smith’s Disease (Absent head/neck of femur due to previous infection in first year of life)
- Excision of the Hip (Girdlestone Procedure)
- Failed Total Hip Replacement (where infection has supervened and prosthesis has been removed)
You may also have a positive TBT in the late stages of Legg-Calve-Perthes Disease (LCPD) and in the case of a Slipped Capital Femoral Epiphysis (SCFE) where you an antalgic gait (painful gait) which may develop into a Trendelenburg's Gait.

There are three major considerations regarding the administration of an injection: route, site and technique.

Three different routes can be taken when administering anion:

Intradermal: this is used when you want a local rather than a systemic effect. Used primarily for diagnostic purposes, eg to check for an allergy.

Subcutaneous: This is used for slow sustained absorption of medication. Ideal for administration of drugs such as insulin.

Intramusculur: This involves the delivery of medication into a well-perfused muscle, providing rapid systemic action and absorption of relatively high doses. This route is taken when the solution to be injected is oily and cannot be injected directly into the bloodstream.

The site chosen for administration should take into account muscle mass, physical status, age, and the amount of drug being administered. There are a number of sites used for IMIs, including:

**Deltoid muscle** is the site for administering of vaccines and Hep B injections etc.

**Vastus Lateralis**, lateral quadriceps muscle, is generally suitable for children under 7 months.

**Dorsogluteal** site was the traditional site for injections in the past but there are associated complications. There is a major possibility of damaging major vessels and nerves such as sciatic nerve and gluteal nerves and vessels (as in our case).

**Ventreogluteal** site is now considered the safer option. This is accessed via the gluteus medius. It is the recommended site which avoids all major vessels and nerves with little complication.

Technique: This is the third important consideration. For IMIs, the needle entry angle should be 90 degrees to ensure needle reaches muscle and in order to minimize pain. To ensure correct entry angle, the injection should be performed with the heel of the palm resting on the thumb of the non-dominant hand. The syringe should be held between the thumb and forefinger and then a firm and accurate thrust of the needle at the correct angle can be achieved.

**Management of Nerve injuries.**

Injuries of this type often results in permanent damage of the nerve which is irreversible. This often results in permanent paralysis of the gluteal muscles. This is most likely what has occurred in our patient case. His prognosis is not very good even after treatment. However for nerve injuries, generally they are treated by two different method: **Conservative treatment**, **Surgical treatment**.

In the conservative treatment, periodic examinations must be done to detect any recovery of paralysis. The paralysed muscles should be splinted in a position that prevents their overstretching. The activity of the opposing muscles should be permitted without stretching the paralysed muscles by the use of dynamic splints. The hip joint should be kept supple by passive
movement, to prevent contracture of the capsule. The patient can be given deep heat followed by electrical stimulation of the involved muscles as the nerve begins to show signs of recovery.

A sign of recovery would be indicated by a gradual increase in motor from a flicker upwards. If the patient can feel tingling along the course of the nerve, this is a sign of recovery. If the injury was relatively minor, a gradual recovery of motor power and sensation occurs in a few weeks and when the power is satisfactory, splinting could be discarded. After a few months, the gluteal muscles should have regained full motor function.

Surgical treatment may be indicated if there is no sign of motor or sensory recovery in the expected time frame. Under general anaesthesia, the nerve is explored by a long incision and the site of injury exposed. The following surgical procedures may be required:

Neuropathy: this is nerve repair in which the cut ends of the nerve are sutured together. Recently microsurgical techniques are used in suturing nerve ends.

Nerve Grafting: this a technique which is used when there is a large gap between nerve ends and the continuity can be restored by use of grafting procedures.

Post operatively, the limb is splinted in such a way that the sutured nerve is in a relaxed position. Efficient physiotherapy is required for muscle re-education during the recovery period.

Case 25: Peripheral Vascular Disease

By: Ailbhe Mc Dermott, Afeedah & Nadiya Hamzah.

Case History

The patient in our case was 65 years old and female. She has a long medical history including diabetes. She was admitted to hospital as a case of peripheral vascular disease with neuropathy. She had a weak dorsalis pedis and popliteal pulsations. The patient also had an area of skin parathesia over the lateral aspect of the right leg. Arteriography was recommended.

A brief introduction on Peripheral Vascular Disease & its anatomical relevance.

Peripheral vascular disease, or PVD, is the name for a group of diseases or problems that cause poor circulation to the peripheral regions of the body. It is mainly the upper and lower limbs, the kidneys and the stomach that are affected. The poor blood supply to these regions is due to a narrowing of the lumen of the arteries which can be as a result of several different conditions. The primary factor for developing PVD is atherosclerosis. Atherosclerosis is a condition in which there is a gradual thickening, hardening and loss of elasticity in the walls of the arteries, creating an occlusion which restricts proper blood flow to the target organs. A normal blood vessel is composed of 3 layers; tunica intima - a single layer of flattened endothelium, supported by a subendothelial connective tissue, tunica media - a thick layer of smooth muscle with varying degrees of elastin & tunica adventitia - an outer layer of supporting
connective tissue. There are several histological changes that can occur as a result of atherosclerosis. In Atherosclerosis, the first step is damage or loss of the endothelium. This may be as a result of diabetes or cigarette smoke for example. Fatty substances and platelets accumulate and attach to the subendothelial connective tissue. Next, monocytes from the blood enter the space, where they differentiate into macrophages and digest the fatty substances resulting in their conversion into foam cells. The platelets secrete growth factor which stimulates smooth muscle cells to multiply and move into the damaged area from the tunica media. The smooth muscle cells secrete a connective tissue matrix to form a fibrous plaque. The foam cells die, leading to the formation of a core of extra-cellular lipid. With time, this plaque becomes calcified. Below is a picture showing a cut section of an artery with atherosclerosis:

The danger of atherosclerosis lies in the spontaneous fissure of the plaque, causing particles to be carried away by the blood. These particles may become lodged in capillaries causing thrombosis, which has very serious medical implications.

The region that must be taken into account in studying our case is the popliteal fossa. Popliteal fossa is a diamond shaped area behind the knee joint that contains the important vessels passing from the thigh to the leg. The Popliteal Fossa contains; popliteal artery, popliteal vein, tibial nerve, common peroneal nerve (which gives off the lateral sural nerve to supply lateral cutaneous innervation of the leg.), small saphenous vein, posterior femoral cutaneous nerve, obturator branch, lymph nodes and vessels & lastly, fat. The popliteal artery lies deep in the fossa, it descends on the floor. To take the popliteal artery pulsation, the patient lies in prone position with the knee flexed to relax the strong popliteal fascia and the hamstring muscles. Pulsations are best felt in the inferior part of the fossa.

More about PVD….
Peripheral vascular disease (PVD) refers to diseases of blood vessels outside the brain and heart. PVD is a build-up of plaque in the arteries that reduces the flow of blood. As a result, some parts of your body don't get the oxygen they need. PVD is a nearly pandemic condition that has the potential to cause loss of limb, even loss of life. It is most likely to occur in the iliac arteries (lower abdomen leading to legs), the femoral and popliteal arteries (legs) as in our case & renal arteries (kidney). PVD can affect both legs but is often more severe on one side. In PVD, arteries can be blocked. There are 2 types of this
circulation disorder. The first one is the functional PVD. It doesn't have an organic cause. They don't involve defects in blood vessels' structure. They are usually short term effects related to 'spasm' that may come and go. For example, Raynaud's disease. The second one is the organic PVD which are caused by structural changes in the blood vessels, such as inflammation and tissue damage. Peripheral artery disease is an example. It is caused by fatty build ups in the arteries that block normal flow. Diabetic neuropathy is a complication of diabetes that affects the nerves. The most common type of diabetic neuropathy is called peripheral neuropathy and affects the peripheral nerves. Peripheral nerves are the nerves that go out from the brain and spinal cord to the muscles, skin, internal organs and glands. Peripheral neuropathy impairs proper functioning of the sensory and nerves. The most common symptoms of neuropathy include numbness and loss of feeling, usually in feet and hands. These explained the presence of an abnormal sensation of the lateral aspect of the right leg as in our case because a patient with poorly managed diabetes may have peripheral neuropathy, characterized by abnormal sensation in cutaneous distribution of the affected nerve. The cutaneous nerve affected in this case is the lateral sural cutaneous branch of the common fibular.

**Risk factors of PVD**

Clinical studies have identified factors that increase the risk of PVD. Some of these factors cannot be changed while others can be managed by greatly reduce the risk of the disease. The first risk factor is diabetes. PVD is common among those individual with diabetes. This correlation is due to complications of the disease that may cause damage to the large and small blood vessels of the legs and feet. Blood must be carefully monitored as damaged to the nerves or foot injury may go unnoticed until an infection or sore develops. The risk of PVD is dramatically increased in smokers. When a person stops smoking, regardless how much he or she may have smoked in the past, their risk of PVD is rapidly declines.

**Differential Diagnosis**

There are many differential diagnoses that can be related to PVD. The first one is atherosclerosis. The second one is diabetes. The third one is Raynaud’s phenomenon which the case where the blood vessels constriction attacks affecting fingers or toes. It is a disorder of small vessels of the extremities, causing coldness and reduced blood flow. In response to cold or anxiety, these vessels go into spasms, causing pain, the sensation of burning and tingling and colour changes. Next is the Buerger’s disease which is the inflammation of the small blood vessels in the leg. And lastly, Leriche syndrome which is a clinical syndrome described by intermittent claudication, impotence and significantly decreased or absent of femoral pulse. The syndrome indicates choronic peripheral arterial insufficiency due to narrowing of the distal aorta.
**DIAGNOSIS**

When the person is suspected to suffer from PVD or if they show the symptoms of the disease, there are several tests that can be done to diagnose it. The first one is **ankle brachial index (ABI)**. The ABI is a simple non-invasive test that measures the ratio of the blood pressure in your ankle to that in your arm. This ratio may indicate a potential vascular problem. The ankle-brachial index (ABI) is a useful test to compare pressures in the lower extremity to the upper extremity. Blood pressure normally is slightly higher in the lower extremities than in the upper extremities. Comparison to the contralateral side may suggest the degree of ischemia. The ABI is obtained by applying blood pressure cuffs to the calf and the upper arm. The blood pressure is measured, and the systolic ankle pressure is divided by the systolic brachial pressure. Normal ABI is more than 1; a value less than 0.95 is considered abnormal. The second test that can be performed is **ultrasound Doppler test**. It is a non-invasive test which provides an image of the inside of the arteries using sound waves to determine if there is plaque buildup, and if so, to what extent. This test is simple, painless, and can be performed as an outpatient procedure in the Cardiologist's office. If the test shows an abnormal result (indicating that there is a blockage), arteriography is performed as recommended by the doctor to this lady to know the exact location of the blockage & how severe it is. **Arteriography** is a procedure in which a contrast material that can be seen using **x-ray** equipment is injected into one of the arteries. An arteriogram can be used to examine almost any artery, including those of the head, kidneys, heart, or lungs. It is sometimes used as part of a procedure to repair the blood vessels called **balloon angioplasty**. First, an intravenous (IV) line is inserted into one of the blood vessels in the groin. A catheter is then inserted through the IV and into the blood vessels using an X-ray machine that produces "live" pictures. Once the catheter is placed into the blood vessel of interest, contrast material is injected and pictures are taken.

**TREATMENT**

There are several treatments available for our patients. Basically, all the treatments focus mainly on controlling the disease. The first one is by taking **medications**.

Medications can be used alone or in combination with one of the treatments. While medications do not eliminate the narrowing of arteries, they can help improve the efficiency of the heart and reduce symptoms such as leg pain, claudication, and hypertension. In some cases, **bypass surgery** may be required in the peripheral arteries if the atherosclerosis is severe enough. Bypass surgery is a way of
creating new channels to carry blood around the blocked areas in your peripheral arteries. With the patient under general anaesthesia, surgeons take a portion of a small blood vessel from the leg or chest to use as the new "bypass artery." They sew or "graft" one end of the bypass to the affected artery and the other end to the artery beyond the narrowed area. Blood then flows through the new grafted vessel, "bypassing" or avoiding the blockage in the peripheral artery. Another alternative to bypass surgery is **angioplasty/balloon angioplasty** which is a minimal invasive interventional procedure. Balloon angioplasty is a similar technique used to open the coronary arteries but in this case, it is performed on blood vessels of the affected lower extremities. This test is performed by threading an instrument down the artery to widen the blocked area. Sometimes a stent is also used along with balloon angioplasty. A stent is actually a wire meshwork that can expand & keep the blood vessel open (patent). The use of **stent** is very important when balloon angioplasty alone is not successful. It also prevents the narrowing of the blood vessel from recurring.

Reference:

www.virginiaradiology.com/vvc/pvd.htm
www.hgcardio.com/pvd.htm

Case 26

Testicular Torsion; Anatomical Aspects.

Testicular torsion is twisting of the spermatic cord upon itself due to rotation of the testes. Intravaginal torsion occurs within the tunica vaginalis.

The testes are the pair of male sex organs that produce spermatozoa and secrete testosterone. They are contains within the scrotum which is an out pouching of the anterior abdominal wall. Each is ovoid shaped and anteriorly related to the epididymus. This is a firm structure consisting of a superior head, a body and a tail. The internal anatomical structure of the testes and epididymus is not relevant to this case. The vas deferens emerges at the tail of the epididymus which points inferiorly. The vas deferens then turns superolaterally and enters the spermatic cord. Also contained within the spermatic cord are the testicular artery, pampiniform plexus, testicular lymph vessels, blood supply to the vas deferens and cremaster muscle and genital branch or the genitofemoral nerve. All of the aforementioned structures are contained within a musculofascial pouch; the spermatic fascia. This is formed from the layers of the anterior abdominal wall as the vas deferens passes through the inguinal canal. Also contained within the spermatic fascia is the tunica vaginalis which is very important in relation to this case.

The tunica vaginalis is a closed sac of peritoneum. The testes invaginate this sac so they are covered by it on their sides and anterior surface. The posterior surface is not covered by the tunica vaginalis and here it can attach firmly to scrotal wall. The tight attachment allows for little mobility and intravaginal testicular torsion does not occur in males presenting normal anatomy as described above.

When testicular torsion does occur it is due to anatomical anomalies of tunica vaginalis or epididymus that allow for excessive mobility. The most common anomaly is the Bell Clapper Deformity. This is
the mane given to the deformity in which the tunica vaginalis completely encircles the epididymus, distal spermatic cord and testes. This leaves the testes free to swing and rotate within the tunica vaginalis like a clapper in a bell. An intermediate form also occurs with a short posterior attachment of the tunica vaginalis to the epididymus. The clinical implications are the same.

Almost any movement of the testes in a person with this deformity can cause rotation of the testes and so twisting of the spermatic cord, i.e. exercise, an active cremasteric reflex, even cold weather can cause this movement. If the spermatic cord does twist to 720 degrees the blood supply to the testes becomes occluded. The result of this is arterial ischemia leading to infarction of the testes. Unless it is treated within 6-8 hours the testes is no longer viable. If the spermatic cord is not twisted as severely it is possible the testes may still be viable after this time. Unfortunately, however, it is unlikely.

Other forms of Testicular Torsion:
Extravaginal Testicular Torsion occurs in the neonatal / prenatal period, as the testes may freely rotate prior to the development of testicular fixation in the scrotum.
In mesorchial torsion an abnormally narrow mesenteric attachment (mesorchium) allows the testis to rotate freely and predisposes to torsion the so-called bell-clapper deformity.

Picture from www.emedicine.com

By Rachel Brodie.
How to diagnose testicular torsion?
By clinical evaluation by urologist, consisting of medical history and physical examinations. Time very important, if urologist cannot exclude torsion or suspects it, surgery without delay. Ultrasound and nuclear medicine techniques can be used to assess blood flow to testicle and therefore can also exclude or confirm torsion.

Classic presentation of testicular torsion

- Scrotal swelling
- Nausea and vomiting (20-30%)
- Abdominal pain (20-30%)
- Fever (16%)
- Urinary frequency (4%)

In older boys, classic presentation of testicular torsion is sudden onset of severe testicular pain, followed by inguinal and/or scrotal swelling. Pain may lessen as necrosis becomes more complete. Problems with urination may occur such as burning and also increase in frequency. Early in the process there may be no swelling. Shortly after, swelling and redness of scrotal skin. After many hours of torsion, testicles die. Then the scrotum would be very tender, reddened and swollen.

Physical:
- Involved testicle painful to palpation; frequently elevated in position when compared to the other side
- Horizontal lie of the testicle
- Enlargement and edema of the testicle; edema involving the entire scrotum
- Scrotal erythema
- Ipsilateral loss of the cremasteric reflex
- Usually, no relief of pain upon elevation of scrotum (elevation may improve the pain in epididymitis [Prehn sign])
- Fever (uncommon)

Ultrasound
Ultrasonography is the use of ultrasound to produce images of structures in human body. Ultrasound probe sends out a short pulse of high frequency sound and detects the reflected waves (echoes) occurring at interfaces within the organ. Direction of pulse moved to area of interest thus building the image. Ultrasound demonstrates an enlarged, heterogeneous right testicle. No loops of peristalsing bowel are present. On sonography, the torsed testicle is usually enlarged and hypoechoic compared with the contralateral normal testicle, and may contain echogenic areas representing hemorrhage.

Doppler
- Doppler examination demonstrates diminished flow in the right testicle and increased flow in the scrotal wall. The left testicle and epididymis are normal. These findings are consistent with a right testicular torsion. Color Doppler ultrasonography increasingly is being used to demonstrate arterial blood flow to the testicle while providing information about scrotal anatomy and other testicular disorders. Color Doppler has a sensitivity of 86%, specificity of 100%, and accuracy of 97% in the diagnosis of testicular torsion when the presence of identifiable intratesticular flow is the sole criterion for diagnosis.
Differential Diagnosis of Testicular Torsion.

- Epididymitis
- Inguinal Hernias
- Orchitis
- Epididymo-orchitis
- Hydrocele
- Torsion of appendix testicle
- Testicular trauma

When a man complains of scrotal pain, acute or chronic epididymitis is far and away the most common diagnosis. Epididymitis is an inflammation of the epididymis. This condition most commonly occurs from the reflux of infected urine or from sexually acquired disease caused by Gonococci and Chlamydia. A patient may occasionally develop these conditions following excessive lifting or straining and the reflux of urine.

The symptoms of epididymitis include painful scrotal swelling, a testicular lump, a tender swollen testicle on affected side, a fever, pain with urination and ejaculation and there may be blood in the semen. Physical examination shows that tenderness is localized to a small area of the testicle where the epididymis is located. A Doppler ultrasound will rule out testicular torsion.

Inguinal hernias in men can cause both scrotal pain and swelling. A hernia in the protrusion of an organ through the wall that normally contain it. An inguinal hernia caused by weakness in the abdominal wall occurs when a part of the intestine protrudes through the abdominal wall into the inguinal canal. The inguinal canal contains the spermatic cord in men thus an inguinal hernia may protrude into the scrotum leading to the symptoms of tenderness and scrotal swelling.

When a hernia cannot be pushed back into place it means a piece of the intestine has become incarcerated or trapped in the inguinal canal or scrotum. This is an incarcerated hernia. Symptoms for this include pain, nausea, vomiting, inability to have a bowel movement, and a bulge that remains when lying down. When a piece of intestine is incarcerated, its blood supply can be cut off, which means the intestinal tissue will die. The hernia is now called a strangulated hernia and is much more serious.

Orchitis is an acute inflammatory reaction of the testis to secondary to infection. Most cases are associated with a viral mumps infection. The characteristics signs of orchitis are testicular pain and swelling. The associated system symptoms are fatigue, nausea, headache, fever and chills. As well as pain and swelling of the testicles erythematous scrotal skin will be seen as scrotal examination. If the epididymis is seen to be enlarged this is a sign of epididymo-orchitis.

Hydroceles are fluid collections within the tunica vaginalis of the scrotum or along the spermatic cord. Thus, a hydrocele is another condition that leads to swelling of the scrotum. The presence of fluid within the hemiscrotum imparts little clinical impact on the testis. However, determining the cause for the increased fluid, specifically any associated clinically significant pathology, remains the primary concern with regard to hydroceles. Once pathology that is more ominous has been excluded, persistence of the hydrocele or the association of discomfort indicates the need for surgical intervention.

Testicular trauma is uncommon because of its anatomic location in the body and the mobility of testes within the scrotum. Most testicular trauma occurrences are related to sports injuries. Other causes include direct trauma, motor vehicle accidents, and straddle injuries. Patients present with acute scrotum and a history of the trauma. Physical examination reveals scrotal oedema, the scrotum is tender to touch and discreet testis may be difficult to palpate due to the oedema of the scrotum. The various types of testicular trauma are testicular fracture, testicular rupture, testicular contusion, testicular haematoma.

The appendix testicle is present in 92% of men and is usually located at the superior testicular pole in the groove between the testicle and the epididymis. The vestigial tissues forming the appendices are structurally predisposed to torsion. In fact, it may undergo spontaneous torsion, this condition is known as torsion of testicular appendage. This condition leads to ischaemia and infarction. It presents as acute testicular pain. Diagnosis can be made when after physical examination reveals a non-tender (or tenderness localised to the upper pole of the testicle ). A small tender lump may be felt at the upper pole of the testis and a blue-dot may be present at the site of the lump. This blue –dot indicates the infarcted testicular appendage.
Treatment of Testicular torsion.

Once testicular torsion has been diagnosed prompt urologic referral is essential since time is critical in salvage of the testicle. Mild analgesic pain relief can be administered once testicular torsion has been diagnosed or while awaiting further studies. Some consultants, however, prefer no analgesics be administered so that their examination is not biased.

Manual detorsion is attempted first. As most tension twist inward and toward the midline, manual detorsion of the testicle inudie twisting outward and laterally.

In a case of suspected torsion of the right testicle, the physician is in part of the standing or lying patient and holds the patient’s right testicle with the left thumb and forefinger. The physician the rotates the right testicle outward 180 in a medial to lateral direction. Rotation of the testicle may need to be repeated2-3 times for complete detorsion and to provide pain relief to the patient. This type of manual detorsion is successful in 30-70% of patients.

It is unlikely that this procedure would work in the case of our patient as the testicle has been in a state of torsion for two days. This patient would be prepared for immediate emergency surgery for detorsion of the testicle if possible and orchiopexy.. An orchidectomy (removal of a testicle) will need to be performed if too much of the testiula tissue has died.

In surgery the man will receive either general or local anaesthesia. The surgeon will make an invasion incision into the scrotum revealing the twisted spermatic cord of the testicle. The doctor will examine the testicle to see how extensive the tissue death is. In the case of our patient, who presented after two days of symptoms, an orchidectomy is carried out. The surgeon isolates, clips and divides the testicular vessels and the vas deferens. The testes is then removed from the scrotum, once it has been ligated from the vessels.

A simple procedure known as orchiopexy is carried out on the remaining testicle. This involves suturing the testicle to the scrotal skin to prevent future torsion. The stitches will be absorbed by the body. The scrotum is then closed with stitches.

It is possible to get a prosthetis to replace the testicle that has been removed. This can help men get over the trauma of this surgical procedure.

CASE 27: Tomás Mc Guinness, Siobhán O’Meara , Juaidy Aizat Zakaria

ANATOMY OF THE RIGHT POSTEROLATERAL ABDOMINAL WALL

The diagram and accompanied cross section (hard copy handed up) gives us an excellent overview of the affected area in our case. Obviously the muscles and other structures affected will be dependent on the angle, precise location of knife entry and also the dimensions of the blade itself. The first layer to encounter the blade is clearly dermis which can be quite thick and contain large amounts of adipose tissue especially in the male

The most superficial muscles are latissium dorsi- a large fan shaped muscle spanning from the shoulder to the lower back and the external oblique may be involved in the injury sustained-fibres
running in an infer medial fashion from the external surface of the 5th-12th rib to the pubic tubercle and anterior half of the iliac crest. Following perforation of these muscles the apex of the blade will encounter the quadratus lumborum muscle and/or the internal oblique and transverse abdominal muscle (as highlighted on the diagram).

The posterolateral abdominal wall is covered with a continuous layer of endoabdominal fascia that lies between the parietal peritoneum and muscles. This fascia lining the posterior abdominal wall is continuous with transversalis fascia that lines the transverse abdominal muscle. The thoracolumbar fascia is an extensive fascial sheet that engulfs the posterolateral muscle and splits into anterior, posterior and middle layers enclosing the quadratus lumborum and deep muscles of the back. As with any injury of this type nerves and vessels are of extreme importance and this region lies close to the lumborum plexus. The ilioinguinal and iliohypogastric nerves arise from the anterior ramus of L1 and course inferolaterally and on the anterior of quadratus lumborum muscle to supply the suprapubic inguinal region and they also send branches to the abdominal wall. The subcostal nerve arises in the thorax and passes through this region to supply the external oblique and skin of the anterolateral abdominal wall. Blood vessels arise from the abdominal aorta and also subcostal arteries arise from the thoracic aorta and distribute inferior to the 12th rib. Damaging of these vessels may be the cause of haemorrhaging in our case. Also accompanying veins are located in this region and drain into the inferior vena cava. The left testicular or ovarian veins enter the renal vein before joining the inferior vena cava (this is not relevant to our patient as he sustained his injury on the right side).

The right kidney also gains protection from the 11th rib-crossing its upper pole and the 12th rib-crossing at the level just above the hilum. Even though we cannot see it in the cross section the diaphragm lies in the superior posterior position in relation to the kidney but does not seem to have been affected in this incident. The hepatorenal recess provides further distance between the right kidney and the liver.

RENA FASCIA AND FAT

The kidneys are situated in the posterior part of the abdomen, one on either side of the vertebral column, behind the peritoneum, and surrounded by a mass of fat and loose areolar tissue. Their upper extremities are on a level with the upper border of the twelfth thoracic vertebra, their lower extremities on a level with the third lumbar. The right kidney is usually slightly lower than the left, probably on account of the vicinity of the liver. The long axis of each kidney is directed downward and lateralward; the transverse axis backward and lateralward.

The kidney and its vessels are imbedded in a mass of fatty tissue, termed the adipose capsule, which is thickest at the margins of the kidney and is prolonged through the hilum into the renal sinus. The kidney and the adipose capsule are enclosed in a sheath of fibrous tissue continuous with the subperitoneal fascia, and named the renal fascia. At the lateral border of the kidney the renal fascia splits into an anterior and a posterior layer. The anterior layer is carried medialward in front of the kidney and its vessels, and is continuous over the aorta with the corresponding layer of the opposite side. The posterior layer extends medialward behind the kidney and blends with the fascia on the Quadratus
lumborum and Psoas major, and through this fascia is attached to the vertebral column. Above the suprarenal gland the two layers of the renal fascia fuse, and unite with the fascia of the diaphragm; below they remain separate, and are gradually lost in the subperitoneal fascia of the iliac fossa. The renal fascia is connected to the fibrous tunic of the kidney by numerous trabeculae, which traverse the adipose capsule, and are strongest near the lower end of the organ. Behind the fascia renalis is a considerable quantity of fat, which constitutes the **paranephric body**. The kidney is held in position partly through the attachment of the renal fascia and partly by the apposition of the neighbouring viscera.

Renal injuries are the most common injuries to the urinary system. Renal injuries can be classified as blunt/non-penetrating injuries or as penetrating injuries and these can further be divided into major and minor injuries. Renal contusions are the most common type of blunt renal injury. Penetrating injuries usually result from gunshot wounds or stab wounds.

Classifications of renal injuries are necessary to determine the extent of injury and possible management according to type of injury. There are five grades of injury: grades I and II classified as minor trauma and grades III, IV and V classified as major trauma. A grade I injury is characterised by a non-expanding subcapsular haematoma, contusions and small infarcts, but no parenchymal laceration has occurred. A grade II injury is characterised by a non-expanding perirenal haematoma and a laceration of less than one centimetre in depth into the parenchyma. No urinary extravasation occurs. A grade III injury is characterised by a parenchymal laceration greater than one centimetre in depth, but it does not extend into the collecting system. No urinary extravasation occurs. A grade IV injury is characterised by a parenchymal laceration extending through the coticomedullary junction into the collecting system, thus leading to urinary extravasation. The main renal artery or vein may be injured with a contained bleed. A grade V injury is characterised by a shattered kidney, or avulsion of the main renal artery or vein or the ureteropelvic junction. Thrombosis of the main renal artery is also classified as a grade V injury. Devascularization of the kidney occurs in a grade V injury.

A patient who has suffered renal trauma will typically present with haematuria that may be either massive or microscopic, but the extent of the injury cannot be measured by the volume of haematuria of the appearance of the wound. Other signs that indicate renal trauma include lumbar and abdominal pain, sometimes rigidity of the anterior abdominal wall and local tenderness. Nausea and vomiting can be present. Extensive blood loss and shock may result from retroperitoneal bleeding.

Investigation of a renal injury begins with an IV urogram. An IV pyelogram is necessary if the possibility of a nephrectomy arises. Ultrasonography may also be used to determine the extent of injury. Further images of the damage done may be obtained by Helical CT which has become the standard CT technology to assess the urinary tract. It is considered to be more accurate than urography or ultrasonography. Arteriography may also be used to determine the extent of the damage. For patients who have suffered major trauma, surgical exploration is usually necessary.

The patient in this case developed peritonitis following surgery. There are two possible explanations for this. Firstly, the peritonitis may be a direct result of the stab wound, in which an
infected knife introduced bacteria into the abdomen around the peritoneum. Secondly, the peritonitis may be due to and infection during or after surgery, although this is less likely to be the case.

**Grade II Renal Injury**

**Grade V Renal Injury**

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**NEPHRECTOMY**

Nephrectomy is a surgical procedure of removing kidney. There are 4 types of nephrectomy, partial, simple, radical and bilateral nephrectomy. Partial nephrectomy is removal of part of one kidney, simple nephrectomy is removal of one whole kidney, radical nephrectomy is removal of one whole kidney together with its neighbouring lymph nodes and bilateral nephrectomy is removal of both kidneys.

Nephrectomy can be done by 2 procedure, laparoscopic procedure and open conventional procedure. For laparoscopic procedure, four small incisions are made at the abdominal wall. Then laparoscope is introduced into the abdominal wall. Laparoscope is used to guide the surgical instrument and to free the kidney. Laparoscopic causes less pain during recovery, allows a quicker recovery and less obvious scarring compared to conventional open procedure. However it takes longer and requires a skilled surgeon.

According to our case, one kidney is removed, therefore it is simple nephrectomy which is done by conventional open procedure. Conventional open nephrectomy is more suitable because the patient’s kidney is seriously injured and haemorrhage occurred. Thus the kidney needs to be removed instantly.

**Procedure of conventional open nephrectomy**

Firstly, general anesthesia is introduced to the patient. The patient is placed on an angled operating table, with his body bent sharply at the waist as shown in the picture below:-

Normally, an angled incision is made through the skin and muscle, along the lower border of the ribs. But in this case, the incision is done at the area of stabbed wound. So the abdominal opening will be widened to about 8 to 12 inches. Nearby organs are moved aside gently. Renal artery, renal vein and
ureter are tied off and cut. Kidney is taken out. Finally, the internal and external layer of incision is closed with suture.

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Case 28

The anatomy and arches of the foot
By Nurain Kasuan

The bone of the foot comprise the tarsus, metatarsus, and phalanges. The tarsus consists of 7 bones: calcaneus, talus, cuboid, navicular and 3 cuneiforms. The calcaneus is the largest and strongest bone in the foot. It articulates with the talus superiorly and the cuboid anterioly. The calcaneus transmits most of the body weight from the talus to the ground.

The talus rests on the anterior two thirds of the calcaneus. The head of the talus articulates anteriorly with the navicular. The navicular a flattened, boat-shaped bone, is located between the talar head and the cuneiforms. The cuboid is the most lateral bone in the distal row of the tarsus. Anterior to the tuberosity of cuboid on the lateral and plantar surfaces of the bone is a groove for tendon of the fibularis longus muscle. There are 3 cuneiform bones: medial (1st), intermediate(2nd) and lateral (3rd). Each cuneiform articulates with the navicular posteriorly and the base of the appropriate metatarsal anteriorly.

There are 5 bones of metatarsus (metatarsals). The 1st metatarsal is shorter than the others. The 2nd metatarsal is the longest. Each bone consists of a base, a shaft and a head. The bases of the metatarsals articulate with the cuneiform and cuboid bones and the heads articulate with proximal phalanges. There are 14 phalanges. The 1st digit has 2 phalanges and the other four digits have 3 each. Each phalanx consists of a base, a shaft, and a head.

The major plantar tarsal ligaments are long plantar ligament, short plantar ligament and plantar calcaneonavicular ligament. The long plantar ligament passes from the plantar surface of the calcaneus to the groove for fibularis longus on the cuboid. The short plantar ligament extends from the anterior aspect of the inferior surface of the calcaneus to the inferior surface of the cuboid. The plantar calcaneonavicular ligament extends from the talar shelf to the posterior surface of the navicular.

The tarsal and metatarsal bones are arranged in longitudinal and transverse arches. The longitudinal arch is composed of medial and lateral parts. Functionally both parts act as a unit with the transverse arch, spreading the weight in all directions.

The medial longitudinal arch is higher and more important. This arch is composed of the calcaneus, talus, navicular, 3 cuneiforms and 3 metatarsals. The tibialis anterior attaching to the 1st metatarsal and medial cuneiform helps strengthen the medial longitudinal arch. The fibularis longus tendon passing from lateral to medial also helps support this arch.
The lateral longitudinal arch is much more flatter than the medial part of the arch and rest on the ground during standing. It is composed of the calcaneus, cuboid, and lateral 2 metatarsals.

The transverse arch runs from side to side. It is formed by the cuboid, cuneiforms, and bases of the metatarsals. The tendon of the fibularis longus helps to maintain the curvature of the transverse arch.

The integrity of the bony arches of the foot is maintained by the shape of interlocking bones, strength of the plantar ligaments especially the plantar calcaneonavicular ligament, and the long and short plantar ligaments, plantar aponeurosis and action of muscles through their tonus and the bracing action of their tendon. The plantar ligaments and plantar aponeurosis bear the greatest stress and are most important in maintaining the arches.

The arches of the foot add the weight-bearing capability and resiliency of the foot. The arches act as shock absorbers for supporting the body weight and for propelling the body during movement. The resilient arches make it adaptable to surface and weight changes. The elastic arches of the foot become slightly flattened by the body weight during standing but they normally resume their curvature when the body weight is removed.

**Differential Diagnosis of Flatfoot**

*By David Tiernan*

Rigid flatfoot is characterized by a lowered arch on both weightbearing and nonweightbearing and by a decrease or absence of motion of the rearfoot and midfoot. Rigid flatfoot can be symptomatic or asymptomatic. Most cases are associated with underlying primary pathology that can be diagnosed by clinical and imaging examinations.

*Congenital Vertical Talus*

CVT deformity, also known as congenital convex pes valgus, is characterized by severe equinus of the rearfoot and by a rigid rocker-bottom appearance. There are 2 classes of this deformity: teratologic and idiopathic. Teratologic CVT indicates the presence of underlying comorbid conditions. These include genetic syndromes, spinal dysraphisms, prune belly syndrome, de Barsy syndrome, distal arthrogryposis, arthrogryposis multiplex congenita, congenital metacarpotalar syndrome, Rasmussen syndrome, and a host of chromosomal abnormalities. Idiopathic CVT lacks specific etiologic factors. CVT has been associated with a tarsal coalition. Genetic issues in idiopathic CVT have not been resolved because of inconclusive data. Results of some studies suggest a hereditary component, whereas others fail to show patterns of inheritance. CVT deformity should be diagnosed at birth but it is sometimes confused with calcaneovalgus deformity or physiologic flatfoot. Symptoms begin at walking age, with difficulty bearing weight and wearing shoes. There may be a history of previous unsuccessful treatment. CVT is characterized by a rigid rocker-bottom appearance to the foot. Pathology findings include dorsal dislocation of the talonavicular joint, ankle equinus, contracture of the tendo-Achilles, long-toe flexors, posterior ankle capsule, peroneal tendons, and the anterior compartment tendons. The tibionavicular ligament is contracted; the calcaneonavicular ligament is elongated. The calcaneocuboid articulation often remodels so that the entire plantar aspect of the foot is convex. Tibialis posterior and the peroneals may be displaced, the talar head becomes misshapen, and the deformity is extremely rigid. Diagnosis of vertical talus is made by the appearance of a rigid and irreducible foot, with support from imaging studies. Initial management of CVT consists of manipulation and serial casting for approximately 6 weeks. The patient must be carefully observed because of an extremely high recurrence rate. If closed reduction is not successful, open surgical reduction is necessary. The goal of surgery is to correct hindfoot equinus, to restore talonavicular congruity, and to restore functional anatomy. Recurrence is a common problem and bracing is recommended.

*Tarsal Coalition*
Tarsal coalition is a congenital union between 2 or more tarsal bones that may be an osseous, cartilaginous, or a fibrous connection. The incidence of tarsal coalition is 1% to 2%. Tarsal coalitions may be asymptomatic. The child and parents may become aware of stiffness in the foot and ankle, altered foot shape, muscle spasm, and protective gait abnormalities. Symptoms of tarsal coalitions most commonly present in preadolescents or adolescents who suddenly gain weight and who take on physical activities. Diagnosis of tarsal coalition is based on pain and loss of motion and supported by appropriate imaging studies. Coalitions are classified by site, type of interposing tissue, extent of involvement, and secondary degenerative changes. The initial treatment for any coalition should be nonsurgical. Patients with mild symptoms may respond well to footwear modifications, arch supports, or custom orthoses. Activity modifications, weight reduction, antiinflammatory medication, and local anesthetic blocks may also be indicated. Surgical consideration should be given to those who fail to respond to nonsurgical treatment. Surgical treatment depends on the type of coalition. Resection of the coalition may be indicated for individuals without significant deformity or arthrosis. Observation and supportive orthoses should follow surgery. If symptoms recur, the patient may need to return to nonsurgical options. These measures are not likely to provide adequate relief of symptoms.

**Peroneal Spastic Flatfoot Without Coalition**

Peroneal spastic flatfoot without coalition is a painful foot deformity made rigid by spasm of the extrinsic muscles. Although tarsal coalition is the most common cause of peroneal spastic flatfoot, its presence cannot be confirmed in a number of cases. Other possible causes include juvenile chronic arthritis, osteochondral fractures in the rearfoot, osteoid osteoma. The patient develops pain in the foot, followed by protective limitation of motion by the extrinsic muscles. Pain is experienced with activity, and symptoms may be precipitated by trauma. Peroneal muscle spasm, restricted subtalar and ankle motion, valgus appearance of the foot, and constant or intermittent pain in response to activity are the hallmarks of the condition. Peroneal spastic flatfoot without coalition is a diagnosis of exclusion and may be ultimately considered idiopathic.

**Skewfoot**

Skewfoot is characterized by forefoot adduction (metatarsus adductus) and heel valgus. The more severe cases have midfoot abduction. There are no universally accepted clinical or radiographic criteria for skewfoot and the natural history of idiopathic skewfoot is poorly understood. There are 4 types of skewfoot: congenital idiopathic, congenital associated with syndromes, neurogenic, and iatrogenic. Skewfoot may be asymptomatic or associated with activity-related pain and difficulty in fitting shoes. It is often misdiagnosed as metatarsus adductus and flexible flatfoot. The deformity is characterized as an S- or Z-shaped foot with forefoot adductovarus and rearfoot valgus. Asymptomatic skewfoot in older children needs no treatment. Management of skewfoot is based on age, degree of severity, and presence of symptoms. Manipulation and serial casting may be indicated for infants. Stretching exercises and activity modification may relieve mild symptoms but they will not change the deformity. Orthoses may be used for symptomatic relief but may exacerbate the symptoms in the presence of ankle equinus. Persistence of severe symptoms may require surgical intervention. Surgical treatment must address both the forefoot and the rearfoot components. Useful procedures include metatarsal osteotomies and midfoot osteotomy to correct the forefoot. Lateral column lengthening, calcaneal displacement osteotomy, and tendo-Achilles lengthening are used to correct the rearfoot.

**Other Causes of Pediatric Flatfoot**

Some forms of pediatric flatfoot deformity do not fit into the previous schemes. They are unique because their clinical findings are dictated by the underlying pathology.
Additionally, the clinical approach to diagnosis and treatment is dependent on the cause. Some have natural histories that are totally unpredictable, and early intervention is undesirable until the problem has fully expressed itself. These forms of pediatric flatfoot are associated with generalized ligamentous laxity; Marfan disease; Ehlers-Danlos; and Down syndrome, cerebral palsy, myelomeningocele, developmental delay, genetic diseases, and other syndromes. A variable pattern of foot deformities may be seen. The deformities range from hypermobile to rigid. Physical examination of these children must include observational gait analysis, assessment of generalized joint mobility for hyperlaxity and hypolaxity, and thorough neurologic examination. In planning the treatment of flatfoot in children with underlying diseases, it is important to consider the patient’s baseline function, the demands placed on the feet, and the natural history of the underlying disease. Asymptomatic hypermobile flatfeet in syndromatic children are usually best left alone. If bracing is not tolerated or does not provide a solid base of support, surgical intervention may be considered. Surgical options are aimed at the specific pathoanatomy and include osteotomies, arthrodesis, arthroereisis, and tendon transfers. Long-term orthosis management after surgical intervention is usually recommended to maximize function.

**Treatment : Flat Feet**

*By Aishling Egan*

Painless flat foot that does not hinder the ability to walk requires no special treatment or orthotic device.

Flexible flat foot in children usually improves with age as the ligaments in the foot and change. Conservative treatments are first used to alleviate the pain caused by flat feet. These include making shoe modifications such as wider fitting shoes, and using orthotic devices such as arch supports and custom-made orthoses. Also non-steroidal anti-inflammatory drugs such as ibuprofen can be prescribed to relief and a corticosteroid can be injected into the joint. Rest and physical treatment such as exercises to strengthen the muscles and ligaments are helpful during the early stages of flat feet. Orthotics are orthopaedic devices used to treat biomechanical foot disorders such as a fallen arch. They can be simple commercially made devices such as heel grips or insoles, or custom-made devices designed to meet the needs of each specific individual.

The orthotic is placed in the patients shoe and helps to keep the foot in proper alignment.

There are 4 categories of orthotic. The first are functional orthotics, and these incorporate special wedges to adjust the heel or forefoot to correct over-pronation. The second type of orthotics are weight-dispersive or accommodative orthotics. These have padding to relieve pain caused by excessive pressure on the metatarsal heads. The third type is supportive orthotics which are arch supports used to treat problems of the plantar arch. The fourth and final type of orthotic is early childhood orthotics. These correct biomechanical walking problems in young children and include splints and night bars.

An orthotic works by bringing the ground into even contact with the foot. This allows the entire foot to support the weight of the body. Some orthotics have extra cushioning sp that some of the force experienced when walking does not affect the foot at all. Most people respond well to a non-operative treatment program.

If conservative treatments do not relieve pain, surgery may be needed to correct the problem. Many types of surgery are available to treat a painful flat foot.
Arthrodesis is a procedure where one or more bones in the foot or ankle are fused or welded together. Osteotomy involves the cutting or reshaping of a bone to improve alignment. Excision is when a bone or bone spur, which is a sharp projection of a bone, is removed. Synovectomy is the cleaning of the sheath covering a tendon. Tendon transfer is a procedure where a piece of tendon is used to lengthen or replace another.

Case 29

THE CAROTID ARTERIES AND RELATED STRUCTURES

The particular region that we are interested in is the carotid triangle, which is a subdivision of the anterior triangle of the neck. The carotid triangle is bounded by the superior belly of the omohyoid, the posterior belly of the digastic, and the anterior border of the SCM.

The Carotid systems of arteries are located in this triangle of the neck. The left CCA arises from the aortic arch while the right CCA arises from the bifurcation of the brachiocephalic artery. Each CCA ascends within the carotid sheath. This fascial sheath blends anteriorly with pretrachial layers of fascia and posteriorly with the prevertebral layer of deep cervical fascia. Within the carotid sheath is also the descending internal jugular vein and vagus nerve. At the level of the superior border of the thyroid cartilage, about the level of C4 the CCA terminates by dividing into the internal and external carotid arteries. It is at this bifurcation that atherosclerotic lesions are most likely to occur.
The External Carotid arteries supply the jaw, face, neck, - that is, structures external to the cranium. They give rise to such arteries as the maxillary, occipital and superficial temporal arteries.

The Internal Carotid Arteries have no branches in the neck. They pass up the neck to the base of the skull, where they enter the carotid canals of the petrous bone. They become the main arteries supplying the brain and the orbits.

The brain, though only representing 2% of the total body weight, receives 1/5 of the resting cardiac output. This blood is carried by the two internal carotid arteries and the two vertebral arteries that anastomose at the base of the brain to form the circle of Willis. Carotid arteries and their branches (referred to as the anterior circulation) supply the anterior portion of the brain while the vertebrobasilar system (referred to as the posterior circulation) supplies the posterior portion of the brain.

There are many important structures in this area. Firstly, at the bifurcation of the internal and external carotid arteries is a slight dilatation of the proximal part of the internal carotid artery - the carotid sinus. This is a baroreceptor that is stimulated by changes in arterial blood pressure. It is innervated by the glosopharyngeal nerve, through the carotid sinus nerve, as well as the vagus nerve. The carotid body is an ovoid mass of tissue, which lies, on the medial (deep) side of the bifurcation of the common carotid artery in close relation to the carotid sinus. It is a chemoreceptor that monitors the level of oxygen and carbon dioxide in the blood. It is supplied by the carotid sinus nerve and also the vagus nerve.

Most veins in the anterior triangle are tributaries of the internal jugular vein, which is usually the largest vein in the neck. The IJV drains blood from the brain, anterior face, cervical viscera, and deep muscles of the neck.

The most important nerve in this area is the vagus nerve. It passes down through the neck within the posterior part of the carotid sheath, in the angle between and posterior to the internal jugular vein and the common carotid artery. It gives off many branches in the neck and elsewhere in the body, such as right and left recurrent laryngeal nerves (which supplies the intrinsic muscles of the larynx except cricothyroid), superior laryngeal nerve, pharyngeal nerve etc. (It also gives off cardiac branches, pulmonary, oesophageal branches etc.) It is sensory from the inferior pharynx, larynx, (and thoracic and abdominal organs.) It is motor to the soft palate, intrinsic laryngeal muscles -necessary for phonation. It is also proprioceptive to these muscles (and is parasympathetic to thoracic and abdominal viscera.)
Another important nerve in this area includes the hypoglossal nerve (CN XII), which is motor to the muscles of the tongue and the thyrohyoid and geniohyoid muscles.

Branches of the sympathetic nerves are also associated with the carotid arteries as well as deep cervical lymph nodes.

**PATIENT’S HISTORY**

The patient is a 65-year-old man, and is suffering from frequent headaches, primarily on the right side over the last two months. There is also four episodes of transient dysfunction over the last two months. Transient dysfunction is a temporary loss of brain function, which causes several neurological disturbances. Those episodes involved the left side of his body and showed progressive symptoms:

1st -- numbness in his left hand
2nd & 3rd --muscle weakness in his left arm and left leg
4th episode -- speech difficulties

**EXAMINATIONS**

**Neck auscultation with stethoscope reveals** bruit (BROO-ee) present on the right side of the neck. A bruit is an abnormal rushing sound caused by turbulent and noisy blood flow over a roughened internal surface of carotid artery. Bruits may be heard at times even with minor blockages. **Arteriogram/Angiogram** is also performed on the patient. Arteriogram is a radiograph obtained after an injection of a special contrast dye into the arteries via a catheter to make those vessels appeared opaque on the x-ray image. It is designed to diagnose various vascular abnormalities or malfunctions such as an aneurysm (enlargement of a blood vessel), stenosis (narrowing of a blood vessel) or blockage.

**DIAGNOSIS**

This patient’s carotid arteriogram revealed ulcerated stenosis of the right carotid bifurcation and mild one on the left-sided carotid stenosis.
Figure: An angiogram of the right carotid artery showing a severe stenosis of the internal carotid artery just past the carotid fork. There is enlargement and ulceration in the area after the stenosis

CAROTID ARTERY STENOSIS.
Like the coronary arteries, the carotid arteries also can develop atherosclerosis. As people age, plaque can build up on the innermost layer of the walls of their carotid arteries. This atherosclerotic plaque (atheroma) which is a deposit of cholesterol, fats followed by further calcification. Alternatively, pieces of plaque from elsewhere in the body could break free, travel to brain and block the blood vessel supplying the brain. As the plaques enlarge, the arteries become narrow and stiff. This process is called atherosclerosis or 'hardening of the arteries' with the loss of elasticity. The presence of plaque could later trigger the immune response and causes ulceration at the spot. More commonly, as the narrowing worsens, pieces of plaque or loose blood clot in the internal carotid artery can break free, travel to the brain and block blood vessels that supply blood to the brain. Stenosis creates an ischemia; condition lacking of oxygen due to insufficient blood flow to the brain. Brain cells require a constant supply of oxygen to stay healthy and function properly. If oxygen is lost, the brain cells will die.

SYMPTOMS
Sometimes the artery may be partially narrowed and there may be asymptomatic. Significant narrowing of the carotid vessels may produce symptoms:
Headache (back of the head), dizziness or confusion, Loss of coordination, Blurred or loss of vision in one or both eyes, Weakness/ numbness of your face, arm or leg; sometimes causing a sudden fall, inability to speak clearly or understanding what others are saying, trouble swallowing, and seizures/ epilepsy.

The type of functions affected and their severities depend on where the blockage has occurred in the brain and the extent of the damage.
The brain has two sides, or hemispheres, with functions assigned to different areas on each side. The nerve fibers from the brain cross as they enter the spinal cord.
Thus, in this case, a worse blockage with the right carotid artery resulted in the patient’s left body weaknesses.

Why were the patient's symptoms generally transient and not permanent?
The effects of the ischemia of the brain do not last long because there is an alternative circulation provided by an important anastomosis in the brain, Circle of Willis, which managed to keep the ischemic region at least partially perfused. This condition is called transient ischemic attack (TIA).
TIA's are temporary/brief episodes of neurological disturbance. Signs of TIA (Transient Ischemic Attack) are the same as signs of stroke except that they may last a few minutes and completely resolve in 24 hours and patient recovered without permanent neurological deficits. People who have suffered a TIA are at significant risk of having a stroke.

Stroke, or Ischemic Cerebrovascular Accident, happens if condition severe where the brain lack of blood flow lasts for more than 3 to 6 hours. A stroke is rather similar to a heart attack. Strokes are longer lasting neurological disturbance, associated with permanent neurologic deficit. A stroke is usually defined as two types:

a) Ischemic (caused by a blockage in an artery). 80% of the all strokes

b) Hemorrhagic internal bleeding of the brain due to tear in the ulcerated artery's wall

If you have coronary artery disease, you are more likely to get carotid artery disease because there is a close relationship between atherosclerosis, coronary artery disease and carotid artery disease. The risk factors for carotid artery disease are similar to those for coronary artery disease; Family history of atherosclerosis (either coronary artery disease or carotid artery disease) due to inborn genetic and inborn factors, and the age and gender factor, particularly those with high blood pressure, who are sedentary, overweight, smoke. Diabetic is also of risk, perhaps because of accompanying factors such as obesity and high blood pressure. Statistics showed greater occurrence in men than women of age less than 75, but higher in women after age 75, but young people are not immune. Strokes in younger people affect men and women equally. Other factors includes smoking, hypertension with abnormal systolic and diastolic blood pressure, diabetes, bad cholesterol level, due to unhealthy diet though it is more of a major role for the heart disease.

The patient was advised that he should undergo a carotid endarterectomy immediately in order to prevent stroke.

CAROTID ARTERY ENDARTERECTOMY
A carotid endarterectomy is a surgical procedure in which a doctor removes fatty deposits from one of the carotid arteries, two main arteries in the neck supplying blood to the brain. Carotid artery problems become more common as people age. The disease process that causes the buildup of fat and other material on the artery walls is called atherosclerosis, popularly known as "hardening of the arteries."

Procedure for doing the carotid artery endarterectomy:

Usually a general anaesthetic is given, and this is for the interest of patient itself so they don't really have to endure the procedure consciously, but some physicians do use local anaesthetic on the basis that it enables the physician to continuously monitor the patients neurologically. Then, an incision is made in the neck from close to the ear lobe and courses down towards the collar bone in the midline, although there are variations in ways which surgeons made the incision to access the posterior triangle of the neck (incision across the neck etc). The carotid arteries are then located and prepared. During the operation to remove the diseased lining, the arteries must be clamped, which means the internal carotid artery that supplies blood to the brain is blocked, which is why usually surgeon insert a plastic tube to carry blood to the brain (shunt) during this operation. The diseased lining of the artery is then carefully removed to leave as smooth a surface as possible. A drain is usually left in place to remove any fluid that may accumulate in the tissues around the artery.

Following operation post operative care, such as neurologic assessment may be taken. The whole operation itself usually last for two hours. Patients are also asked to refrain from some physical activities, such as driving, that could cause some pain at the neck, for a few months.

There are some risk that is associated with endarterectomy. One of them could be injuries to the surrounding cranial and peripheral nerve. For examples, injuries to the hypoglossal nerve could cause distension of tongue to one side of the face. Injuries to the branches of cervical plexus, for example greater auricular and transverse cervical nerve usually only results in the tingling sensation and numbness on one side of the face only. An injury to the recurrent laryngeal nerve could cause hoarseness in patients voice since its a very important nerve that supplies the vocal cord muscle. Most of the time these symptoms healed by itself within a few months since nerve are only bruised most of the time, not severed off. There could be other complications such as stroke, although the recurring rate is much lower than if patients opted to do other treatment options, such as carotid artery angioplasty and stenting. Restenosis of the artery may also occur although this is very uncommon. Basically most of this
complications heavily depends on the patients lifestyle itself, for example, if the patients continue to smoke, the possibility of restenosis is significantly higher

GRAND CLINICAL ROUND 14: CASE 30

By

Ciaran O’Fearraigh, Zawani Karim and Dilan Jogendra

Anatomy of the Subclavian Vein:
The subclavian vein is found in the lower part of the subclavian triangle, a division of the posterior triangle of the neck. It begins at the lateral border of the 1st rib as the continuation of the axillary vein. It crosses behind the clavicle just medial to the mid-clavicular point, as it arches over the superior part of the first rib in a groove on the superior surface of that rib.

When it reaches the medial border of the scalenus anterior muscle, which lies posteriorly and attaches to the first rib, it is joined by the internal jugular vein, which drains blood from the skull, brain, superficial parts of the face and much of the neck.

It then turns posteriorly and descends into the thorax as the brachiocephalic vein. The right and left brachiocephalic veins unite to form the superior vena cava which enters the right atrium, thus providing direct access to monitor central venous pressure.

The subclavian vein is the most anterior neurovascular structure in the subclavian triangle. The clavicle and subclavius muscle lie anteriorly. Posteriorly lies the scalenus anterior muscle, which separates the subclavian vein from the posterosuperior subclavian artery, as well as the inferior trunk of the brachial plexus. Posteromedially it is separated from the dome of the pleura by the suprapleural membrane.

The phrenic nerve courses downwards on the anterior surface of the scalenus anterior muscle behind the subclavian vein to enter the thorax. This nerve is important because it supplies complete motor innervation to the diaphragm and so should be carefully avoided. In 45% of cases an accessory branch of the phrenic nerve passes anterior to the subclavian vein and thus may be at risk of damage during attempted venipuncture. Lying inferiorly is the first rib. The vein usually has a pair of valves located about 2-2.5cm from its termination.

The external jugular vein joins the subclavian vein lateral or anterior to the scalenus anterior muscle and the junction usually contains a pair of valves. Sometimes the subclavian vein receives the anterior
jugular vein instead of it joining the external jugular vein, and occasionally a small branch ascends in front of the clavicle from the cephalic vein.

The left subclavian vein receives the thoracic duct near its junction with the internal jugular vein. This duct constitutes the entire lymphatic drainage of the body, except that of the right arm and the right half of the head neck and thorax. Most importantly, the cisterna chyli, which collects the lymphatic drainage of the gut, drains into the thoracic duct. The right subclavian vein receives the right lymphatic duct which is smaller than the right thoracic duct. It is for this reason that right subclavian vein catheterization is preferred, since puncture of a lymphatic duct is less likely on the right side.

Technique for Subclavian Catheterization:

The patient is placed in Trendelenburg’s position [head down at least 15 degrees to distend the neck veins and prevent air embolism], and a small rolled towel is placed between the shoulder blades [i.e. to bring the subclavian vein in close relation to the clavicle and to minimize interference with the humeral head]. After identification of the landmarks (see below), sterile preparation, and administration of local anaesthesia, the skin is punctured 2 to 3 cm caudal to the midpoint of the clavicle with a large calibre introducer needle. The needle is advanced in the direction of the suprasternal notch where the finger of the other hand is placed, until the tip of the needle abuts the clavicle at the junction of its medial and
middle thirds – this is done to align the needle with central axis of subclavian vein and to minimize puncture of subclavian artery or piercing pleura or apex of lung. The needle is then passed beneath the clavicle, with the needle hugging the inferior surface of the clavicle. If no blood returns with passage of the needle, the needle is withdrawn past the clavicle while gentle suction is applied. Blood return may be achieved during withdrawal of the needle. If the first pass is unsuccessful, the needle should be angled in a slightly more cephalad direction on the next insertion attempt. Obtain a Chest X-ray STAT to identify the position of the tip of the catheter and a possible pneumothorax.

Position of the Catheter: should be on the level of the Right Atrium, but no more than 3-4 cm above this level in the Superior Vena Cava. Tip is malpositioned if it is in the Right Atrium or Right Ventricle, Ipsilateral Internal Jugular Vein, Peripheral Vein.

**Surface anatomical landmarks for guiding Infraclavicular Subclavian Catheterization:**

- The **midclavicular point** [i.e. 1 cm below and 1 cm lateral to the junction of the middle and medial thirds of the clavicle] and the **lower border of the clavicle** are essential for locating the Subclavian Vein for venipuncture.
- Further, the **suprasternal notch** is used to aim the needle once it is inserted under the clavicle.

**RIGHT-SIDED versus LEFT-SIDED approach to Subclavian Catheterization:**

A **right-sided approach** is preferred because laceration of the **THORACIC DUCT**, which empties into the **Left Subclavian Vein** [near the union of the Left Internal Jugular and Subclavian Veins or *Left-Venous Angle*], can be avoided by catheterization on the right side.

**Potential Complications to Subclavian Catheterization**

Complications to catheterizations are not rare. However, subclavian catheterization may cause the least number of complications compared to other types of catheterizations such as femoral or internal jugular venous catheterization. So, it is the most favourable location compared to the other two types of catheterization.

The most common complication that may arise from subclavian venous catheterization is **pneumothorax**. Pneumothorax is a condition of accumulation of air or gas in the pleural cavity (sometimes induced to collapse the lung in the treatment of tuberculosis and other lung diseases). In the case of subclavian venous catheterization, it is due to the location of cervical pleura and apex of the lung that arises up to about 2.5 cm superior to the first rib and may be punctured while performing subclavian
venous catheterization. So an X-ray is always performed immediately after the procedure to check for pneumothorax. This complication usually arises especially when several attempts to locate the subclavian vein are made, and usually occurs when one is not adhering to the proper anatomically based performance method.

Another complication that could potentially develop is **hemothorax**. The concept is the same as with pneumothorax but in this case, blood is accumulated in the pleural cavity instead the air and may happen if the subclavian artery or vein or any blood vessels in the area or the apex of the lung is punctured during the procedure.

Subclavian venous catheterization is preferably done at the right-side instead of left-side. This is due to larger thoracic duct at the left-side compared to the smaller right lymphatic duct on the right-side. If the catheterization is made on the left-side, puncture of the thoracic duct may cause **chylothorax**. Chylothorax is a condition where lymph is collected in the pleural space. This condition is very difficult to treat and has a relatively high rate of morbidity.

The other complication that could potentially arise after the catheterization is cardiac tamponade. Cardiac tamponade is a compression of the heart caused by blood or fluid accumulation in the space between the myocardium and the pericardium, preventing the ventricle from expanding maximally, thus they cannot adequately fill or pump blood. Generally, it often happens after the placement of central venous catheter by direct perforation of the right ventricle, superior vena cava, or right atrium due to unsuccessful puncture attempts of the right subclavian vein. Bleeding from the perforation of the intra-pericardial ascending aorta results in rapid filling at the pericardium, which later on induces cardiac tamponade.

The next complication is **bacterial infection or sepsis**. This may occur when maximal sterile barrier precautions are not used during the procedure. These include the use of sterile-catheter, mask, cap, sterile-gown, gloves, and a large sterile drape. Application of antibiotic ointments to catheter-insertion sites increases the rate of catheter colonization by fungi, thus promoting the emergence of antibiotic-resistant bacteria and further has not been shown to lower the rate of catheter-related bloodstream infection.

Last but not the least, another potential complication of the subclavian venous catheterization is **brachial plexus injury**. Puncture or damage to the brachial plexus may lead to neurological injury to the arm, depending on which part of the brachial plexus is injured.

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CASE 31
RECTUM AND ANAL CANAL

ANATOMY OF RECTUM
The rectum is the part of the alimentary tract that is continuous proximally with the sigmoid colon and distally with the anal canal. The rectosigmoid junction lies at the level of S3 vertebrae. The rectum follows the curve of the sacrum and coccyx and ends anteroinferior to the tip of the coccyx where it turns posteroinferiorly to become the anal canal. The rectum is ‘S’ shaped and has three flexures as it follows the sacrococcygeal curve.

BLOOD SUPPLY OF RECTUM AND ANAL CANAL
The continuation of the inferior mesenteric artery, the superior rectal artery, supplies the proximal part of the rectum. The two middle rectal arteries – arising from the inferior vesical in the male and the uterine artery in the female, supply the middle and inferior parts of the rectum, and the inferior rectal arteries arising from the internal pudendal arteries supply the anorectal junction and anal canal. Blood from the rectum drains via superior, middle and inferior rectal veins. Because the superior rectal veins drain into the portal venous system and the middle and inferior rectal veins drain into the systemic system, this communication is an important area of portacaval anastomosis. The submucosal rectal venous plexus in females the rectal venous plexus consists of two parts, the internal rectal venous plexus just deep to the epithelium of the rectum and the external rectal venous plexus external to the muscular wall of the epithelium. Internal haemorrhoids are associated with dilation of the superior rectal plexus of veins in the canal columns. The superior rectal veins communicate with the middle and inferior rectal veins; they have no valves and back pressure in the portal venous systems will therefore fill the haemorrhoidal plexus. The internal and external haemorrhoidal plexuses are connected by small veins crossing beneath the skin. Increased pressure in the internal plexus may therefore be transmitted to the external plexus to produce external skin covered haemorrhoids.

ANATOMY OF ANAL CANAL
The muscular anal canal forms a sphincter at the distal end of the G.I. tract. Posteriorly the canal is separated from the top of the coccyx by the anococcygeal ligament. The ischiorectal fossa lie on either side of the anal canal and are continuous via the retrosphincteric space between the coccygeal attachment of the external sphincter below and the levator ani above the anal canal ends at the anus. It is surrounded by the internal, involuntary sphincter surrounding the superior two thirds of the anal canal which is a thickening of the circular muscle of the intestine
and also by the external and sphincter, a large voluntary sphincter that forms a broad band on each side of the anal canal. The anal canal descends posteriorinferiorly between the anooccygeal body and the perineal body.

**INTERNAL ANAL CANAL**

Internally, the superior half of the mucous membrane of the anal canal is characterised by a series of longitudinal ridges called anal columns, which contain terminal branches of the superior rectal artery and vein. The anorectal junction is indicated by the superior ends of the anal columns. The inferior ends of the anal columns are joined by anal valves. The inferior comb-shaped limit of the anal valves forms an irregular line called the pectinate line. The lining of the anal canal above the pectinate line is columnar epithelium continuous with that of the rectum and below the pectinate line is skin. In the upper anal canal longitudinal folds of the columnar epithelium run down to the pectinate line and cover vascular submucosal connective tissue to form ‘anal cushions’ which when abnormally enlarged form haemorrhoids. As regards innervation, the mucosa above the pectinate line level has a visceral innervation from the inferior hypogastric plexus and is sensitive only to stretching. The innervation inferior to the pectinate line is schematic innovation from the inferior rectal nerves, branches from the pudendal nerve and is sensitive to stimuli.

**STRUCTURES WHICH MAY BE PALPATED IN DIGITAL RECTAL EXAM**

- Lower edge of internal and external sphincters with anal intermuscular groove between them.
- Anorectal ring, formed by the upper end of the anal sphincters lies about 3 cm from the anal verge and is felt posteriorly.
- Normal rectal mucosa is smooth.
- Male → Prostate and seminal vesicle felt anteriorly.
- Female→ Cervix and uterus felt anteriorly.
- Posteriorly sacrum and coccyx may be felt.

**DIFFERENTIAL DIAGNOSIS**

Rectal bleeding or hematochezia refers to passage of blood from the anus, often mixed with stool and/or blood clots. Most rectal bleeding comes from the colon, rectum, or anus. The colour of the blood during rectal bleeding often depends on the location of the bleeding in the GIT. Generally, the closer the bleeding site to the anus, the blood will be a brighter red. Thus, bleeding from the anus rectum and sigmoid colon tends to be bright red whereas bleeding from transverse colon and the right colon tend to be dark red or maroon.

Many diseases and conditions can cause rectal bleeding and these include, haemorrhoids, anal fissures, diverticulosis (colon that contains outpouchings in people usually older than 40 years old), inflammatory bowel disease, angiodysplasia (a vascular problem that involves enlarged veins and capillaries in the wall of colon), polyps (lumps of tissue that bulge out from the lining of the colon) and tumour. In case of our patient, based on the symptoms she had and her obstetric history it is most likely that she is suffering from haemorrhoids (internal) or an anal fissure.

**Haemorrhoids** are swelling of the veins that line the wall of anus and lower rectum. It is thought that these veins become wider and engorged with blood if the pressure in and around them is increased. Basically, there are three types of haemorrhoids; internal haemorrhoids which develop above the dentate line, pr
obtruding haemorrhoids, a grade 4 internal haemorrhoids that permanently hangs down from within the anus and external haemorrhoids, a small lump that develops on the outside edge of the anus. The symptoms of internal haemorrhoids include painless rectal bleeding, uncomfortable feeling of fullness after passing stools, mild skin irritation, and itching. It can also cause pain. The symptoms of external haemorrhoids include slight painful swelling of veins near the anus, inflammation, skin irritation, itching and burning. Associated risk factor of haemorrhoids include constant sitting, straining with bowel movement, diarrhoea, severe coughing, pregnancy and childbirth and also heavy lifting.

**Anal fissure** is a tear or split in the lining of the anus. Anal fissure that lasts less than six weeks is called acute anal fissure and most heal by itself. A fissure that lasts more than six weeks is called chronic anal fissure. Fissures can extend upward into the lower rectal mucosa or extend downward causing a swollen skin tab to develop at the anal verge called sentinel pile. The symptoms include anal pain, pain when passing a bowel motion and for sometime afterwards and also bright red blood from the anus, this problem is common in children younger than one year old and affects 8 out of 10 infants. Common causes in adults include chronic constipation, and trauma to the anus such as a difficult childbirth. Other causes are diarrhoea, inflammation, Crohn’s disease and anal injury.

**Treatment of Haemorrhoids:**

Initial treatment is aimed at easing symptoms. First of all, haemorrhoids and anal fissures are often irritated by **constipation** therefore reducing pressure in the affected area is important. Suggested measures include:

- Addition of extra fibre (cereals, whole-grain breads, fresh fruit and vegetable) and fluids (plenty of water & fruit juices e.g. prune juice) to diet
- Listen to your body. Never avoid going to the bathroom when you feel the urge
- Try not to strain when you’re moving your bowels, and don’t linger on the toilet
- Exercise regularly
- Do Kegel exercises daily (pelvic floor exercises recommended during and post pregnancy that involve tensing of muscles around vagina and anus, hold for 8-10 seconds before releasing and relaxing. Repeat 25 times.)
- Lose weight if overweight
- Stool softeners – laxatives are drugs used to stimulate and increase frequency of bowel movements. They also encourage the passage of a softer, bulkier stool.

**Anal itching** may be reduced by the following suggested measures

- Wear cotton undergarments
- Avoid toilet paper with perfumes or colours. Moistening the tissue may help.
- Try not to scratch the area

**Anal ache or pain** can be relieved by

- Application of an ice pack or cold compresses. Some advise alternating hot and cold treatments as in a Sitz bath, which allow rapid alternation of hot and cold water.
- Sit in a bath with 8-10 inches of warm water several times a day
- Pain medication e.g. the local anaesthetic lidocaine is often prescribed in the treatment of anal fissures.
Procedures
No anorectal procedure should be undertaken without digital and sigmoidoscope examination. An abdominal examination is generally performed to rule out any other causes.

External Haemorrhoids
*Conservative treatment* may be followed but if not successful and recovery is prolonged, surgical removal, termed a *haemorrhoidectomy* would be considered, particularly if the haemorrhoid is thrombosed. Local anaesthetic is given and haemorrhoid is exposed. An ellipse of skin is cut out and it reveals the typical black blood clot inside a thrombosed haemorrhoid and it is removed. Wound is checked so as to make sure that there is only one haematoma and if more were present, they would be removed. Many now recommend excising the ellipse of skin above the haemorrhoid as this has a reduced recurrence rate than simple excision and removal (expression) of thrombus. Suture closure is generally required.

Internal Haemorrhoids
It is important to note that generally the conservative treatment (advise and medication) is successful and the need for any of the following procedures is low.

*Sclerotherapy*
This involves the submucosal injection of a sclerosing fluid (e.g. 5% oily phenol) into the upper end of each haemorrhoid, usually located at 3 o’clock, 7 o’clock and 11 o’clock positions. Injection must be given above pectinate line so as to avoid pain. The sclerosing fluid causes thrombophlebitis, which encourages obliteration of varicose veins by thrombosis and subsequent scarring. Mucosal collapse is prevented by fixing it to a bowel using the sclerosant.

All haemorrhoids can be treated at the same time. If one is larger (commonly the right anterior) it may given two injections. This treatment is not suitable for patients with bleeding disorders or those taking anticoagulation medication.

*Rubber Band Ligation*
This is a non-surgical technique that has good success rate and is often used to treat Grade II, III AND IV haemorrhoids. It is essentially tying off the haemorrhoid using a rubber band to strangulate it. This procedure is only suitable if base of haemorrhoid is 2cm above the anorectal junction. Below this level the patient would be in sever pain and it would be very difficult to remove the constricting band. Secondary haemorrhage occurs in one percent of cases.

*Infrared Coagulation*
This is a new non-surgical technique, which has a high success rate. Surgery can be avoided in approximately eighty per cent of cases. It is the simplest procedure to apply. Much like a laser, this treatment involves the focusing of infrared light on haemorrhoid tissues coagulating them. It causes coagulation of blood vessels supplying the haemorrhoid and by cutting off it’s’ blood supply, the haemorrhoid shrinks and symptoms will subsequently subside. This procedure is suitable for Grade I, II and some grade III lesions. Usually three to four treatments are required. Advantages include that the procedure takes about five minutes, is painless and does not involve lengthy recovery periods.

*Cryosurgery*
This is defined as the “the use of extreme cold in a localised part of the body to freeze and destroy unwanted tissues.” Cryosurgery involves the use of an instrument called a cryoprobe, which has a fine tip cooled by
allowing carbon dioxide or nitrous oxide as to expand within it. The nitrous oxide cry probe has the advantage that it adheres to the tissue, so that gentle traction may be applied. This prevents freezing of deeper tissues. The probe is rewarmed before it is separated from the haemorrhoid. Treatment is well tolerated although some patient feels a dull ache during and immediately after freezing. Watery, blood stained discharge can be expected in the weeks following treatment.

The above procedures are used in the treatment of Grade I & II haemorrhoids.

**Haemorrhoidectomy**

Enemas are given prior to surgery, which is generally performed under spinal or general anaesthetic. Sigmoidoscopy is performed, if it did not form part of earlier investigations. Anal Dilation is performed and a gauze sponge is inserted into lower rectum so as to prevent downward faecal leakage. Haemorrhoids are identified and using a clamp haemorrhoids are grasped at usual positions (3, 7 and 11 o’clock). A triangular shaped incision is made, including the skin of the anal verge up to the pectinate line or to the base of the haemorrhoid. (The anal verge is the line that separates the pigmented perianal skin from the pink pigmented skin.) Clamped haemorrhoid is excised including the mucosa but not the skin of the anal verge. Enough mucosa must be left if anal stenosis to be prevented. Stenosis is defined as “abnormal narrowing of a passage or opening”. Possible complications of a haemorrhoidectomy include infection, stricture of anus (stenosis), and haemorrhage.

**Complications if Haemorrhoids**

Iron deficiency anaemia may result if blood loss is significant. Infection or ulceration of haemorrhoid may result. Prolapsed irreducible haemorrhoids can become gangrenous

**Treatment of Fissures**

Anal fissures can be treated quite simply if identified within three months of onset. Many respond to the giving of anal suppositories, which contain a corticosteroid and antiseptic cream. As the rectal suppositories melt, they relieve pain and promote healing. Some advocate the use of a cream instead of a suppository because suppositories cause pain on insertion. Measures taken to reduce constipation would also be advised in this case. Once a fissure is chronic, surgery is generally required and a procedure called a lateral sphincterectomy is performed. This involves incision of the lowest fibres of the internal sphincter allowing the anal musculature to relax, giving the anal fissure time to heal.

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Case 32

Claire Reid
Ariffin Shah
Kok Jin Gan
**Anatomy of Breast Region with particular emphasis on Lymphatic Drainage**

**Position and Shape**
Breasts vary greatly in size and shape. This is due to genetic, racial, and dietary factors. The breast is a rudimentary structure in males.

The roughly circular base of the female breast extends vertically from ribs 2-6, and traversely from the lateral border of the sternum to the midaxillary line.

2/3 of the breast rests on the deep pectoral fascia covering pectoralis major. Approximately 1/3 rests on the fascia covering serratus anterior.

**Structure**
Between the breast and the deep pectoral fascia is the retromammary space. There are 15-20 lobules of secretory glandular tissue embedded in fat. Our patient is 66 so we presume that she is post-menopausal. As a result, glandular elements will have regressed and breasts will have lost some of their fullness. A well-developed connective tissue stroma surrounds the ducts and lobules of the mammary gland. Suspensory ligaments are formed from fibrous septa that separate and support the lobules. They run from the subcutaneous tissue to the fascia of the chest wall and are continuous with the dermis of the skin.

Each lobule drains by its lactiferous duct onto the nipple, which is surrounded by the pigmented areola. This area is lubricated by modified sebaceous glands called “Areolar Glands of Montgomery”.

The breast may be divided into 4 quadrants, and “clock positions” may be used to describe the location of tumours and cysts.

**Blood Supply**
Axillary artery: lateral thoracic and thoracoacromial branches.
Internal thoracic artery: perforating branches, the 1st and 2nd perforators are the largest.
Posterior intercostal arteries.
A superficial venous plexus surrounds the areola. Venous drainage is mainly to the axillary vein. There is some drainage medially to the internal thoracic vein and possibly the external jugular vein.

**Innervation**
Nerve supply derives from branches of 4th-6th intercostal nerves. Sensory fibers are sent to the skin of the breast, and sympathetic fibers are sent to blood vessels and smooth muscle in the overlying skin.

**Lymphatic Drainage**
Carcinoma of the breast can arise in the epithelium of the lymphatic ducts, and secondaries may spread early through the lymphatic channels draining the breast. This was likely to be the mode of cancer spread for our patient.

Lymph passes from the nipple, lobules, and areola to the subareolar lymphatic plexus. From here:
- 75% (mainly from the lateral quadrants) drains superolaterally into axillary nodes, mainly to the pectoral (anterior) node.
- 20% drains medially into parasternal nodes which are associated with the internal thoracic artery, passing from there into bronchomediastinal trunks. Lymph, and a cancerous growth may pass from here to parasternal nodes of the contralateral breast.

Most of the remaining 5% drains through lymph vessels that follow the lateral branches of posterior intercostal arteries and connect with posterior intercostal nodes situated near the heads and necks of ribs. Most lymph drains from here to bronchomediastinal trunks.

Lymph from the lower quadrants may pass to the inferior phrenic (abdominal) nodes.
The presence of clumps of cancerous cells in lymphatics and lymph nodes often results in unusual patterns of drainage opening up in compensation. Secondaries may then be found in a variety of places, eg:

**Groin nodes** - via lymph vessels in the trunk wall.

**Cervical nodes** - as a result of retrograde extension from blocked thoracic or jugular trunk.

**Peritoneal Lymphatics** - spreading there in a retrograde manner from the parasternal nodes.

**Cancer alters the anatomy and appearance of the breast.**

Permeation of the lymphatic channels of the breast by cancer produces a leathery thickened appearance of the overlying skin. Tension of the carcinoma on the suspensory ligaments creates dimpling of the skin. Prominent pores may develop due to edema resulting from blocked lymphatic drainage. These features give rise to the classic “peau d’orange” sign which is characteristic of well developed cancer—this is the stage our patient was at when she presented with her symptoms.

Our patient suffered pleural effusion. This is a build up of fluid in the space between the visceral and parietal pleurae of the lungs. It is possible that the lymphatic drainage of the pleural cavity is blocked by a lymphoma. Lymph from the lungs and pleura passes to bronchomedistinal trunks, as does some lymph from the breast and this may be how the cancer spread. As a result, interstitial fluid is not being removed and so pools in the pleural cavity.

Clearly, the lymphatic drainage of the breast is quite extensive and involves many lymph nodes which drain other tissues too. In this way, cancer may spread to tissues and organs such as lungs, bone and liver.

Ultimately, all lymph and its contents passes to the right lymphatic duct or the thoracic duct and is returned to the venous circulation at the junction of the internal jugular vein and the subclavian vein.

**Sources of Information/ Bibliography**

- Essential Clinical Anatomy , Keith L. Moore and Anne M.R. Agur, 2nd Ed
- Clinical Anatomy, Harold Ellis, 9th Ed
- Textbook Of Anatomy, AW Rogers

Main image: Anatomy Of Female Breast

A = Lactiferous Duct
B = Lobules
C = Lactiferous Sinus
D = Nipple
E = Fat Tissue
F = Pectoralis Major
G= Ribcage/ Chest Wall

**Enlarged Duct Lumen: Normal Duct**
A= Normal Duct Cells
B= Basement Membrane
C= Lumen Of Duct.
If the patient has an Invasive Ductal Carcinoma, much of the lumen would be obliterated by the rapidly dividing cancerous cells. Ductal cancer cells would break through the basement membrane and spread to other tissue.

![Diagram](image)

**Lymphatic Drainage Of The Breast**
Subareolar lymphatic plexus lies deep to the areola
A= Pectoralis Major Muscle
B= Axillary Lymph Nodes Level I
C= Axillary Lymph Nodes Level II
D= Axillary Lymph Nodes Level III
E= Supraclavicular Nodes
F= Internal Mammary (parasternal nodes)

**Patient history.**
We do know that the patient is a 66 year old female having mass of increasing size in the left breast. Most of the lymph nodes are removed. A modified radical mastectomy was performed and the biopsy revealed metastasis. Apart from that, pleural effusion recurred despite radiation therapy and she died 3 months after surgery.

**Signs and symptoms:** The following are the most common symptoms of breast cancer. However, individuals may experience symptoms differently. Early breast cancer usually does not cause pain and may cause no symptoms at all. And, approximately 10 percent of breast cancer patients have no pain or lumps, or other indications of a problem with their breasts.

- a lump or thickening (a mass, swelling, skin irritation or distortion) in or near the breast or in the underarm area
- a change in the size or shape of the breast
- a change in the color or feel of the skin of the breast, areola or nipple (dimpled, puckered or scaly)
- nipple discharge, erosion, inversion or tenderness
Classification

- 2 types

a) In situ carcinoma: Is characterized by growth within the ducts without penetration of the basement membrane. In situ carcinoma is subdivided into ductal carcinoma in situ (DCIS) and lobular carcinoma in situ (LCIS).

b) Invasive carcinoma: Denotes neoplastic penetration of the basement membrane of a duct containing DCIS and extension of neoplastic cell aggregates into the mammary stroma. It is further subdivided into these types: ductal, which accounts for about 75% of all invasive breast cancers; medullary; mucinous, or colloid; papillary; tubular; adenoid cystic carcinoma; and carcinoma with metaplasia.

c) Paget disease of the nipple: Is a type of breast cancer that starts in the breast ducts and spreads to the skin of the nipple and then to the areola.

Related conditions

- Two other entities also need consideration: inflammatory breast cancer and phyllodes tumor.

Risk Factors for Breast Cancer

1. Risk factors that cannot be changed:

- Gender
- Aging
- personal history of breast cancer
- family history and genetic factors
- previous breast biopsy in which the tissue showed atypical hyperplasia
- menstrual periods that began early in life
- menopause began later in life

2. The most frequently cited lifestyle-related risk factors:

- smoking
- not having children or having first child after age 30
- oral contraceptives used for a period of time greater than 10 years
- obesity and a high-fat diet
- alcohol: more than three drinks per week
- long-term, post-menopausal use of combined estrogen and progestin (HRT)
- weight gain and obesity after menopause
Differential diagnosis.

For circumscribed lesions, the differential diagnosis includes benign breast disease (e.g., fibroadenomas, cysts), breast cancer, breast lymphoma, and metastasis to the breast from other primary sites.

For skin thickening, the differential diagnosis includes inflammatory carcinoma and mastitis.

For stellate lesions, the differential diagnosis includes breast cancer, traumatic fat necrosis, a radial scar, and a hyalinized fibroadenoma.

For dilated ducts with or without nipple discharge, the differential diagnosis includes papilloma, ductal carcinoma, duct ectasia, and fibrocystic disease.

Patterns of Metastasis

Breast cancer is considered to be a heterogeneous, highly variable disease. Even among women with the same histological type, clinical stage, and treatment some will be cured, while others have emergence of metastatic disease within six months of therapy. The development of aberrant cell clones, with diverse growth rates and metastatic potential may in part account for the differences seen in clinical behavior. Research concerning the role of angiogenesis in tumor growth and its specific relevance in transformation, progression, and metastasis of breast cancer is ongoing. While the process of metastasis is a complex and poorly understood phenomenon, there is evidence to suggest that angiogenesis (neovascularization) of the tumor plays an important role in the biological aggressiveness of breast cancer. Breast cancer metastasizes widely and to almost all organs of the body, but primarily the bone, lungs, nodes, liver, and brain. The first sites of metastases are usually local or regional involving the chest wall or axillary supraclavicular lymph nodes or bone. Women with estrogen receptor negative disease are more likely to have recurrences in visceral organs whereas women with ER (+) disease more often have recurrences in skin and bone. Patients with metastatic disease generally present with symptoms specific to that organ. For instance, women with metastatic disease to bone often complain of bone pain or in the case of liver metastases, anorexia, weight loss, malaise, and occasionally right upper quadrant pain. CNS metastases may present with specific neurological symptoms such as headache that is more severe in the morning or relate to specific neurological damage such as cranial nerve palsies (double vision), motor dysfunction, or spinal cord symptoms.

Treatment

Surgery:

Lumpectomy: A surgical procedure to remove a tumor (lump) and a small amount of normal tissue around it.

Partial mastectomy: A surgical procedure to remove the part of the breast that contains cancer and some normal tissue around it. This procedure is also called a segmental mastectomy.

Lumpectomy & Partial mastectomy is considered as Breast-conserving surgery, because only cancer cells are removed and not the breast itself.

Total mastectomy: A surgical procedure to remove the whole breast that contains cancer. This procedure is also called a simple mastectomy. Some of the lymph nodes under the arm may be removed for biopsy at the same time as the breast surgery or after. This is done through a separate incision.
Modified radical mastectomy: A surgical procedure to remove the whole breast that contains cancer, many of the lymph nodes under the arm, the lining over the chest muscles, and sometimes, part of the chest wall muscles.

Radical mastectomy: A surgical procedure to remove the breast that contains cancer, chest wall muscles under the breast, and all of the lymph nodes under the arm. This procedure is sometimes called a Halsted radical mastectomy.

Common complications of breast surgery are bleeding and infection. Seromas (which is accumulations of clear fluid under the arm) can develop and are usually treated with percutaneous needle aspiration. Major complications after axillary dissection include lymphoedema of the arm (which is build-up of fluid in the arm if the lymph nodes have been removed) and nerve damage. Injury to the long thoracic nerve denervates the serratus anterior muscle and causes a winged scapula. Intercostobrachial nerve damage causes loss of sensation in the upper inner arm.

**Radiation therapy** is a cancer treatment that uses high-energy x-rays or other types of radiation to kill cancer cells. There are two types of radiation therapy. **External radiation** therapy uses a machine outside the body to send radiation toward the cancer. **Internal radiation** therapy uses a radioactive substance sealed in needles, seeds, wires, or catheters that are placed directly into or near the cancer.

**Chemotherapy** is a cancer treatment that uses drugs to stop the growth of cancer cells, either by killing the cells or by stopping the cells from dividing. When chemotherapy is taken by mouth or injected into a vein or muscle, the drugs enter the bloodstream and can reach cancer cells throughout the body (systemic chemotherapy). When chemotherapy is placed directly into the spinal column, an organ, or a body cavity such as the abdomen, the drugs mainly affect cancer cells in those areas (regional chemotherapy).

**Hormone therapy** is a cancer treatment that removes hormones or blocks their action and stops cancer cells from growing. Hormone therapy with tamoxifen is often given to patients with early stages of breast cancer and those with metastatic breast cancer (cancer that has spread to other parts of the body). Hormone therapy with tamoxifen or estrogens can act on cells all over the body and may increase the chance of developing endometrial cancer.
Started menses age 12.
First sexual intercourse age 13.
Now married for two years.
Smokes 1 pack of cigarettes a day.
Prior pap smear 5 years back.

Family History: Nothing significant

Medical History: History of genital warts during last pregnancy.

Physical Examination: Revealed an ulcerated 2-3 cm mass on the cervix, which bleeds on touch.

Discuss the anatomy and histology of cervix. Describe the staging and spread of cervical carcinoma. Mention briefly the treatment options.

By Miswani Salleh 03112764 87
Niall Kelly 03442438 45

Case 33
Cervical carcinoma

Invasive carcinoma is the sixth most common malignancy in females. It is rare before the age of 20 years. The peak incidence is in the 45 - 55 year age group but occurs slightly earlier among those from lower socioeconomic groups.

Anatomy of the cervix;
- Cervix is the cylindrical, narrow, inferior part of the uterus that protrudes into the uppermost part of the vagina
- The cervix of the uterus lies between the bladder and the rectum in the true pelvis
- It is divided into vaginal and supravaginal parts
- The rounded vaginal part of the cervix communicates with the vagina via external os
- The supravaginal part of the cervix is separated from the bladder anteriorly by loose connective tissue and from the rectum posteriorly by the rectouterine pouch
- The cervix contains cervical canal that links the uterine cavity with the vagina
- The uterus, in the pelvis is supported principally by the pelvic fascia and the urinary bladder on which it normally rests
• The cervix is the least mobile part of the uterus because it is held by ligaments;
  o Cardinal ligament: thickened free inferior part of the broad ligament. Extend from the
cervix and lateral part of the vaginal fornix to the lateral walls of pelvis
  o Uterosacral ligament: from sides of cervix goes posteriorly and attached to the middle of
the sacrum.
• Peritoneum covers the uterus anteriorly and superiorly, except for the vaginal part of the cervix.
• The peritoneum is reflected anteriorly from the uterus onto the posterior margin of the superior
surface of the urinary bladder
• Leaving the cervix lying in direct contact with the bladder without intervening peritoneum. This
allows cervical cancer to invade the urinary bladder easily
• Posteriorly, the peritoneum is reflected over the posterior part of the vaginal fornix onto the
rectum
• The supravaginal part of the cervix is separated posteriorly from the rectum by the rectouterine
pouch
• Laterally, the uterine artery crosses the ureter superiorly, near the cervix, in the root of the broad
ligament. This artery gives a cervical branch that supplies the cervix. Venous blood drains into
uterine venous plexus formed on each side of the uterus and vagina. Veins from this plexus drain
into the internal iliac veins.
• The lymphatic vessels from the cervix pass to the internal iliac and sacral lymph nodes.

Histology of the cervix;
• The uterine cervix differs in histologic structure from the rest of the uterus

![Histology of the cervix image]

- Stroma composed of tough collagenous tissue containing some smooth muscle.
- Ectocervix-stratified nonkeratinized squamous epithelium continuous with vagina
- Endocervix-mucin secreting simple columnar epithelium
- Squamocolumnar junction-between ecto and endocervix(indicated by arrow) is normally located at the
  external os

**transformation zone**

- Cervical stroma influenced by ovarian hormones, particularly estrogens which causes the cervix
to stretch, thin and dilates as in late pregnancy and during labour
To a much lesser extent, similar changes occur during normal menstrual cycle. Changes in stroma volume causing eversion of endocervix, exposing it to the vaginal environment. Induces growth of stratified squamous epithelium over exposed area. Zone may undergo malignant changes causing cervical cancer.

Referring to case 33;
- D.G. has a medical history of getting genital warts during last pregnancy. Genital warts?
  - Wart: a benign growth on the skin caused by infection with human papillomavirus (HPV)
  - Genital warts are frequently associated with other genital infections
  - Affected women have increased risk of developing cervical cancer
  - HPV is linked to about 95% of cervical cancer cases

- Our patient also;
  - had first sexual intercourse at the age of 13
  - smokes a pack of cigarettes a day

- Risk factors for cervical cancer include:
  - human papilloma virus - types 16, 18 and 33; note that neither partner may have visible warts
  - smoking
  - human immunodeficiency virus
  - age of first coitus - adolescent cervix more susceptible to carcinogenic stimuli as squamous metaplasia in the transformation zone is active during this time
  - number of sexual partners - increased likelihood to be exposed to carcinogens; cervical cancer is virtually unknown in celibate women
  - young age at first pregnancy - metaplasia most active during first pregnancy
  - high parity
  - low social status - possibly, due to vitamin A deficiency
  - sexual partner with multiple sexual partners

**Cervical carcinoma**
- cancer of the cervix
- may develop from the squamous epithelium of the vaginal part of the cervix (squamous carcinoma)
- or from the endocervical simple columnar epithelium (adenocarcinoma)
- the squamous carcinoma is more common and frequently develops at the transformation zone
- Cervical cancer follows a progression of dysplasia to in situ carcinoma to invasive carcinoma.
Diagnosis Of Cervical Cancer

There are three tests that can help diagnose cervical cancer. These are:

- Pap Smear
- Colposcopy
- Biopsy

Pap Smear

A Pap Smear (named after it’s inventor Dr. Papanicolaou) involves the doctor taking a scraping of the surface of the cervix with a spatula or a brush. The patient should be in the lithomy position, which is lying on the back with hips and knees flexed and feet supported high and rotated outwards. This should be a painless procedure and may be preceded by a vaginal exam in which the doctor places two fingers inside the vagina and presses the other hand on the abdomen to check that the uterus is in the proper position. A speculum is inserted to help view the cervix. The resulting scraping is then sent to the lab for analysis. It should be noted that an abnormal smear test does not indicate cervical cancer on it’s own, but can be used as a basis for performing the other tests.

Colposcopy

A colposcopy exam is done with the patient in the same lithomy position with speculum inserted. The doctor inserts a high-powered microscope called a colposcope through which he can view the cervix surface. He sprays the surface of the cervix with acetic acid, which will stain any abnormal cells white, due to their elevated levels of glycogen. Small areas of staining may be treated immediately using a number of methods including:

- Loop Excision
- Cryotherapy
- Cold Coagulation

Loop Excision

Involves a wire loop through which a high frequency current runs which can be used to remove the area of tissue
This is a procedure that can be done easily in the outpatient clinic and means that there is a tissue sample for pathological tests.
It does however have some association with cervical incompetence and stenosis

Cryotherapy

This involves freezing the area of the cervix with a nitrogen probe.
It is also an easy outpatient procedure but there is an unknown depth of tissue affected and there is no sample for pathological analysis

Cold Coagulation
This involves the heating of the tissue to over 100°C. It can easily be performed in the outpatient clinic but again there is an unknown depth of tissue affected and there is no sample that can be sent to the lab.

**Biopsy**

This involves the removal of a portion of the cervix tissue, usually bigger than, but including, the stained area from the colposcopy. A cone biopsy is when a cone shape is cut from the cervix. This may be enough to treat a small staining and leaves a good sample that can be sent to the lab to check the cell type.

Complications of a cone biopsy include haemorrhage, and also cervical stenosis, which may be accompanied by dysmenorrhea or haematuria. In more severe situations, it may also cause cervical incompetence and a subsequent mid-trimester abortion. However, a biopsy will be taken at all times, if possible, to ensure that the cells in the stained region are not cancerous.

Excision of the affected area is only really possible in small areas of change, which occur in a condition called **Cervical Intraepithelial Neoplasm (CIN)**, where the cells of the cervix have become irregular, or dyskaryotic, for various reasons.

There are 3 types of CIN:
- CIN I, or mild dyskaryosis
- CIN II, or moderate dyskaryosis
- CIN III, or severe dyskaryosis

Progression through these stages can be very rapid and more than one stage may be seen on the one sample.

CIN III is the last non-cancerous phase in the development of cervical cancer, and is only differentiated from Stage 0 of Cervical Cancer (also called Carcinoma In Situ (CIS)) in that its cells are not cancerous.

**Differential Diagnosis of the Symptoms**

The mass that was observed on the cervix could be a Benign Neoplasm of the Cervix, or a Cervical Polyp. These arise from the endocervix and have a central fibrous core and a covering of endocervical epithelium. These will present as a bright red vascular growth and their presenting symptoms will be intermenstrual or post-coital bleeding. These can be treated surgically with excision of the lump.

Another possibility for the cervical mass could be cervical fibroids, which are similar to uterine fibroids. They are derived from smooth muscle and any malignant change in these is very rare.

The bleeding, post-coital and intermenstrual, could also be caused by a Sexually Transmitted Infection (STI), like chlamydia or gonorrhea or by Vaginitis, though vaginitis would most likely present with other symptoms.

**Staging of Cervical Cancer**

There are 5 stages in the development of cervical cancer, each requiring different modes of treatment

Stage 0 (Carcinoma In Situ)
This is the same as CIN III, except that the cells have now become cancerous.

Stage I

This is an invasive carcinoma that is confined to the cervix, or one that has originated in the cervix and has extended upwards to involve the body of the uterus.

It is subdivided in to 2 categories, which are each further divided in to two more.

<table>
<thead>
<tr>
<th>Stage IA1</th>
<th>Size</th>
<th>5 Year Survival Rate</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depth: &lt;3mm Width: &lt;7mm</td>
<td></td>
<td>84% - 90% when less than 3cm</td>
<td>Local Excision</td>
</tr>
<tr>
<td>Stage IA2</td>
<td>Depth: 3-5mm Width: &lt;7mm</td>
<td></td>
<td>Simple Hysterectomy</td>
</tr>
<tr>
<td>Stage IB1</td>
<td>Depth: &lt;4cm</td>
<td>66% when greater that 3cm</td>
<td>Radical Hysterectomy or Radiotherapy</td>
</tr>
<tr>
<td>Stage IB2</td>
<td>Depth: &gt;4cm</td>
<td></td>
<td>Radical Hysterectomy or Radiotherapy</td>
</tr>
</tbody>
</table>

Simple Hysterectomy

Involves the total removal of the uterus and the cervix.

Radical Hysterectomy

Involves a total hysterectomy as well as excision of the parametrium and the upper 1/3 of the vagina and dissection of the pelvic lymph nodes (Internal Iliac, External Iliac and Obturator nodes). The ovaries may be conserved and coital function is preserved.

Radical Radiotherapy

Either External Beam Therapy (Teletherapy) to the Pelvis, which is applied numerous times over the course of a few weeks, or Brachytherapy, which is local vaginal therapy where a probe is put in the vagina for 12 to 18 hours. Radiotherapy may lead to sexual dysfunction, radiation cystitis or vaginal stenosis, and thus surgery is the recommended for younger patients.

Stage II

The carcinoma has extended beyond the cervix but has not reached the lateral pelvic wall. The vagina is also now involved but only the upper 2/3.

This is also divided in to two stages:

<table>
<thead>
<tr>
<th>Stage IIA</th>
<th>5 Year Survival Rate</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper 1/3 Vagina</td>
<td>62%</td>
<td>Radical Hysterectomy or Radiotherapy</td>
</tr>
<tr>
<td>Stage IIB</td>
<td>Upper 2/3 Vagina and also Parametrial Disease</td>
<td>Radiotherapy ± Chemotherapy</td>
</tr>
</tbody>
</table>

Parametrial Disease

This is diagnosed through a rectovaginal exam.

Stage III

At this stage, the carcinoma has extended to the pelvic wall and the whole vagina, including the lower third, is also involved.
Stage IIIA  Lower 1/3 Vagina  40%
Stage IIIB  Pelvic Side Walls  Radiotherapy ± Chemotherapy

Stage IV

Here, the carcinoma has extended beyond the true pelvis, and may involve the bladder or the rectal mucosa

Stage IVA  Bladder, Rectum  Radiotherapy ± Chemotherapy
Stage IVB  Beyond Pelvis  15%

Recurrent Disease

This is when the disease keeps appearing after initial treatment. There is a one-year survival rate of just 10 – 15%. Radiotherapy may be used as a treatment option, but will only be effective if it has not been used before, and in the majority of cases it will have been. It may be cured by Pelvic Exenteration which involves the excision of the Vagina, Uterus, and the Bladder or Rectum or both. If the patient is carefully selected for this operation, there may be a 5-year survival rate of 60% but will cause major morbidity. Chemotherapy and Radiotherapy may be used in palliative care of the disease-related symptoms.

How this information affects our patient

Our patient, D.G., had a 2cm mass on her cervix, which was found to be cancerous. This would suggest that it is in Stage IA2 and that her treatment options are a Radical Hysterectomy or Radical Radiotherapy. She is still quite young at only 36 and already has 2 children, so if she does not plan to have any more children, a Radical Hysterectomy would be the recommended choice. It would spare the ovaries radiation damage and would preserve coital function. Should she follow this course of treatment, she will have to take a Pap smear every 6 months for the first 2 years and at least once a year from then on.

Sources:

Textbook Of Gynaecology, J.H. Peel and J.M. Brundenell
Essential Obstetrics and Gynaecology, M. Symonds and I. Symonds
Clinical Obstetrics and Gynaecology, Drife and Magowan
Branches of the aorta

The ascending aorta arises from the aortic orifice of the left ventricle. It continues as the arch of aorta at the level of sternal angle, over the left main bronchus. The arch of aorta gives of three major branches, the first branch being the right brachiocephalic artery which further divides to form the right subclavian and right common carotid arteries. The second branch is the left common carotid artery and the final branch is the left subclavian artery. These vessels supply the upper limb, head and neck region.

The thoracic (descending) aorta is the continuation of the arch of aorta. It begins on the left side of the body of T4 vertebra and descends in the posterior mediastinum on the left sides of T5 to T12 vertebrae. It gives off branches such as the bronchial arteries, pericardial arteries, posterior intercostal arteries, superior phrenic arteries, esophageal arteries, mediastinal arteries and subcostal arteries. These arteries supply the thoracic region. The thoracic aorta enters the abdomen via the aortic hiatus of diaphragm becoming the abdominal aorta.

The abdominal aorta is a continuation of the thoracic aorta from the level of T12-L4 vertebrae anterior to the vertebral bodies. It terminates at the level of L4 vertebra as right and left common iliac arteries. The abdominal aorta gives of three major unpaired branches. The first being the coeliac trunk at the lower border of T12 vertebra. As soon as it comes off the abdominal aorta the coeliac trunk divides to give off the left gastric artery, common hepatic artery and splenic artery. The second major branch of the abdominal aorta is the superior mesenteric artery at the levels of L1 vertebra. It gives off inferior pancreaticoduodenal artery, jejunal and ileal branches, ileocolic artery, appendicular artery, right colic artery and middle colic artery. The final major branch of the abdominal aorta is the inferior mesenteric artery at the level of L3 vertebra. It gives off left colic artery, sigmoid artery and superior rectal artery.

The paired branches of the abdominal aorta are the suprarenal arteries, right and left renal arteries, gonadal arteries, lumbar arteries and inferior phrenic arteries. The abdominal aorta terminates as the right and left common iliac arteries and median sacral artery at the level of L4 vertebra.

Histology of aorta

The aorta is a large elastic artery. It is composed of three layers. The innermost layer is the tunica intima. This layer is composed of flattened endothelial cells. The middle layer is the tunica media, which is primarily composed of smooth muscle and elastic fibres. The outermost layer of this vessel is the tunica adventitia, which is composed of collagen.
Layers of the heart wall

The heart wall is composed of three layers. The epicardium is the outermost serous layer composed of visceral pericardium. The middle layer is the myocardium. This is a thick layer and is composed of cardiac muscle. The innermost layer is the endocardium. It covers the internal surface of the heart and covers the valves of the heart.

What is coarctation?

Coarctation of the aorta is a localized narrowing of the lumen of the aorta. The aorta is the main artery of the body. The first branch goes to the upper body, the arms and head, and after that the blood goes to the lower body, the abdomen and legs. Coarctation of the aorta is a narrowing of the aorta between the upper-body artery branches and the branches to the lower body. This blockage or narrowing can increase blood pressure in the head and upper body and reduce pressure in the legs. This puts a serious strain on the heart.

The reason that the blood pressure is so dramatically elevated in the upper extremities and not the lower extremities is for two reasons.

1 The blockage itself
2 The kidneys do not detect the high blood pressure from the blockage as high blood pressure. As they are in the lower half of the body they do not receive sufficient blood and they release substances which eventually convert to angiotensin 2. This angiotensin 2 causes vasoconstriction and increases arterial blood pressure as a whole in systemic circulation.

It is usually a birth defect. It occurs most commonly just past the point where the aorta and the subclavian artery meet. It constitutes 7-8% of congenital cardiac defects.

It is more common in some genetic conditions such as Turner’s syndrome, but it can also be associated with congenital abnormalities of the aortic valve, such as the bicuspid aortic valve.

Symptoms and signs

Symptoms of coarctation of the aorta depend on the severity of blood flow restriction. In severe cases, symptoms are present during infancy; in milder cases, symptoms may not develop until adolescence. Adult type is usually not diagnosed until late childhood or early adulthood when hypertension and a difference in arm and leg pulses are detected on routine physical.

Our case is of adult type as it is only at the age of 23 that it has been detected.

Symptoms include decreases exercise performance, cold feet or legs and shortness of breath all of which are present in our case.

Other symptoms include

- dizziness or fainting which our case does not have although she does feel weak and easily tired
- pounding headache which our patient experiences frequently
- epistaxis or nose bleeds which our patient also experiences
- leg cramps with exercise
- hypertension (high blood pressure) with exercise
- A soft bruit or murmur may be heard over the coarctation site. A systolic murmur is heard in our patient over the 5th left intercostals space which radiates to the interscapular region.
- Femoral pulses although often palpable are diminished and delayed compared with brachial pulses
- Rib notching due to increased blood flow through and dilation of the internal mammary arteries may be detected on x-rays although it is not usually visible before the age of 10. Rib notching is present in our patient.

The adult type often has intercostal rib notching due to collateral flow through dilated pulsatile intercostal arteries and it is seen in 75% of older children with coarctation. It is seen in the 3rd - 9th ribs because the 1st and 2nd ribs intercostal arteries come from the thyocervical trunk which is above the coarctation. Irregular wavy densities seen on the lateral film reflect dilation of the internal mammary arteries.

An ECG indicates left ventricle enlargement as the heart is trying to pump against the obstruction and putting extra strain on itself. X-rays usually show a normal heart-size with coarctation visible in a slight left anterior oblique view. Another sign in the adult type is the "3" sign which is created by a prominent left subclavian artery, the coarctation and post stenotic dilation of the descending aorta. The barium swallow shows a reversed "3" which represents the aortic impression on the esophagus above and below the coarctation.

**Physical Exam**

Coarctation of the aorta is often discovered during a newborn infant’s first examination or during a well baby exam. This is obviously not the case with our patient. The coarctation in our case as I have already mentioned is the adult form.

During a physical exam:

Blood pressure and pulse are measured in both arms and both legs. The cuff for measurement of blood pressure must be under 20% of the diameter of the limb. The blood pressure in the upper limbs will be significantly higher than in the lower limbs. This is the case in our patient with the blood pressure in the upper limb being measured at 190/100 and the blood pressure in the lower limb at 40/10.

To test for hypertension the blood pressure is measured on the supine seated and standing positions.

The radial and femoral pulses are also measured. In coarctation there is a delay between the pulses or the femoral pulse may be absent completely or diminished as the blood to the lower extremities is being somewhat constricted by the blockage or narrowing of the aorta. This is known as
the radial-femoral pulse delay. The femoral pulse in our patient is diminished and the distal pulses are absent.

The heart is listened to through a stethoscope and usually reveals a murmur that is harsh and can be heard in the back. There may be signs of left sided heart failure and signs of aortic regurgitation.

*Aortic regurgitation is also known as aortic insufficiency. This is a heart valve disease in which the aortic valve weakens or balloons preventing the valve from closing tightly. This leads to backflow of blood from the aorta into the left ventricle.*

Other tests include an X-ray of the chest may also show abnormal ribs or ‘notching’ of the ribs caused by enlargement of the rib arteries which is present in our patient.

**Treatment**

Surgery is usually advised. Occasionally balloon angioplasty may be an alternative to surgery. In balloon angioplasty they use a technique similar to that used to open the coronary arteries except in this case it’s used on the aorta.

Usually in surgery the narrowed part of the aorta is removed and then repaired by anastomosis. This is a procedure where the 2 free ends of the aorta are stapled or sewn back together after the narrowed section is removed.

Anastomosis can only be used if the segment with the coarctation is short. If the segment is longer then a Dacron graft is used to fill the gap. A Dacron graft is a synthetic material used to replace normal body tissues. It is usually made into tubular form for replacing or repairing blood tissues as in this case. It causes very few reactions because it is chemically inert and easily tolerated by the body.

After surgery prophylactic antibiotics are needed to prevent endocarditis. This is a microbial infection of the heart valves and endocardium which as is the innermost layer of viscera around the heart.

Anastomosis would be recommended in our case as balloon angioplasty is usually only used in the very young due to reoccurrence tendencies in older people. The walls are weakened by the original coarctation so there is a tendency for it to reoccur. Although these are the most commonly used treatments there are some alternatives. Although they are not used that often due to increased success with the procedures mentioned before.

Patch augmentation is where a patch is added to the aorta to enlarge it. A by-pass like graft can be placed in the area of narrowing known as a jump graft. Very rarely, an ascending to descending aortic bypass graft is used to bypass the narrowing in the aorta. This procedure is quite invasive and can be used where valve damage has also occurred as it allows the surgeon to replace the faulty valve at the same time as remedying the coarctation.

**Expectations (prognosis)**

Coarctation of the aorta is curable with surgery, and rapid improvement of symptoms often occurs after the repair. There is an increased risk for death caused by cardiovascular problems among patients who have undergone aortic repair; however, without treatment, most people with this condition
die before they reach the age of 40. Early surgical intervention (before 10 years old) is usually advised. Today, diagnosis of a coarctation and subsequent surgical repair typically occur during infancy. Blood pressure needs to be checked every 1-2 years. The reason is that the patient is at risk of developing generalized high blood pressure or problems with the aortic valve. More surgical intervention may be needed if resting blood pressure in arms and legs does not return to normal level. Depending on the success of the treatment the patient may be advised to avoid certain forms of strenuous exercise. Other treatment may be needed for other complications arising from the original coarctation. For example aneurysms may be a problem due to the fact that when the coarctation originally occurs the other blood vessels dilate to try to increase blood flow to the lower half of the body. Due to this aneurysms may occur as the blood vessels prolonged dilation will result in weakening and eventually bursting of the blood vessels. This may cause problems in organs such as the kidneys.
SCAPHOID FRACTURE

Functional anatomy

The wrist joint is actually a collection of many joints and many bones. These joints and bones let us use our hands in many ways. The wrist must be extremely mobile to give our hands a full range of motion. At the same time, the wrist must provide the strength for heavy gripping.

The wrist join or carpus comprises the interval between the distal end of the radius and ulna and the proximal end of the metacarpal bones. The 8 carpal bones (also called the navicular bone) are arranged in 2 rows and are cuboid with 6 surfaces. Of these 6 carpal surfaces, 4 are covered with cartilage to articulate with the adjacent bones and 2 are roughened for ligament attachments. The proximal row, which contains the scaphoid, lunate, triquetrum, and pisiform, articulates with the radius, triangular cartilage and fibrocartilaginous end of the ulna to form the carpus. The ulna does not articulate with the carpus. The distal row contains the trapezium, trapezoid, capitate, and hamate n articulates with the proximal surface of the metacarpal bones.

What we call the wrist joint is actually made up of many small joints. Ligaments connect all the small bones to each other, and to the radius, ulna, and metacarpal bones. The wrist has 5 large joint cavities in addition to the intercarpal joint spaces, and they are the radiocarpal joint, distal radioulnar joint, midcarpal joint, large carpometacarpal joint (between the carpus and the second, third, fourth, and fifth metacarpals), and small carpometacarpal joint (between the first metacarpal and trapezium). Wrist joint is synovial ellipsoid joint.

The muscles of the hand originate primarily in the forearm and pass over the wrist. The flexor carpi ulnaris, which inserts into the pisiform bone, is the only muscle that inserts into the wrist. Strong, ligamentous attachments stabilize the wrist.

Wrist joint innervated by anterior interosseous nerve and also deep branch of radial nerve. For the wrist joint, Blood is supplied via the radial and ulnar arteries, which form the dorsal palmar arch. The scaphoid bone receives its blood supply from the distal part of this arch, which is prone to injury.

Movement that can occur at the wrist joint are motion at the wrist joint occurs between the radius and carpal bones Wrist movement is 80° in flexion, 70° in extension, 30° in ulnar deviation, and 20° in radial deviation. We have to remember that Pronation and supination occur at the radioulnar articulation in the forearm, not at the wrist.
The scaphoid bone is a small carpal bone on the thumb side (radial side) of the wrist. It is the most commonly fractured carpal bone. This is probably because it actually crosses two rows of carpal bones, forming a hinge. A fall on the outstretched hand puts heavy stress on the scaphoid bone. This stress can cause either a small crack through the middle of the bone or a complete separation of the bone into two pieces. A separation is called a displaced fracture but it won't give so much prob as fracture do since it wd b easily to spot when xray taken, so it can b treat at an earlier stage b4 in worsen. Anatomic considerations at this joint, blood enters the bone along the dorsal surface near its mid portion. Thus, the scaphoid is prone to avascular necrosis.

TREATMENT OF SCAPHOID FRACTURE
Treatment is determined by the fracture site, the degree of displacement, and any associated injuries. Most scaphoid fractures are treated with immobilization in either a cast that covers the forearm, the wrist and the thumb or one that covers the full arm, wrist and thumb. However, it is not easy to immobilize this particular bone, due to its small size and location.

Fracture
If the fracture is identified immediately and is in good alignment, you will probably wear a cast for 9 to 12 months which will cover your forearms, wrist and thumb. This is necessary to hold the scaphoid bone very still while it heals. X-rays once a month is also necessary to check the progress of the healing. Once the doctor is sure that the fracture is healed, the cast is removed. Casting is most effective for patients who have a fracture that is incomplete. However, even with this type of treatment, there is still a risk that the fracture may not heal well and will become a non-union.

Non-union
A fracture that doesn’t heal within several months is considered as a non-union. There is a smooth fibrocartilage covering the fracture site in non-union. Synovial fluid is present in the interval. About 10-15% of all scaphoid fractures do not unite.

Delayed union
Delayed union is incomplete union after 4 months of cast immobilization. Delayed union is anticipated if fracture treatment is delayed for several weeks. The risk of nonunion increases after a delay of 4 weeks. These delays may be related to the patient’s failure to seek treatment for a presumed sprain, but they more
frequently are related to improper or incomplete immobilization or a failure to
diagnose and treat the acute fracture.

- Treatments of delayed and nonunion vary. If the delayed union is stable and
  less than 6 months old relative to the time of injury, prolonged cast
  immobilization with or without electrical stimulation may be used. Several
  methods of internal fixation and surgical techniques exist; none have
  universally good results. The treatment of choice for a symptomatic nonunion
  is placement of a bone graft and fixation. Occasionally, intercarpal fusion and
  excision of the proximal pole or entire scaphoid are used as treatment
  methods. Rarely, proximal row carpectomy is performed. Many surgeons treat
  all non-unions rather than only the symptomatic ones because chronic non-
  unions are associated with secondary osteoarthritis.

**Electrical Stimulator**

If the injury is fairly recent, the surgeon might recommend more time in the cast. He
or she might also prescribe an electrical stimulator which sends a small electrical
current to the scaphoid bone. It is worn like a bracelet for 10 to 12 hours to help the
bones heal.

![Electrical stimulation device](http://www.handuniversity.com)

**Surgery**

If the non-union is quite old, or if the cast and the electrical stimulator fail to heal the
fracture, the surgeon will recommend surgery. The surgeon will first make an incision
in the wrist directly over the bone; nerves and blood supply are then moved aside.
This lets the surgeon see the joint capsule. Next, the surgeon finds the old fracture line
on the scaphoid bone. All the scar tissue lining the 2 halves of fractured bone must be
removed. This creates a fresh bone surface to allow healing. Anesthesia used: Auxillary Block / local anesthesia.

**Bone Graft**
The surgeon may use a bone graft. This involves taking bone tissue (from patient’s pelvis) and inserting it to the fracture. After bone graft is placed between parts of scaphoid, metal pin or screw is inserted to hold the bone together, allowing them to fuse into 1 bone. Anesthesia used: light general anesthesia which last for less than 20 minutes.

- Sometimes bones still do not heal as planned. Doctors call a fused bone that fails to heal as **pseudoarthritis**. If pain continues, a second operation might be needed.
- After surgery, physical or occupational therapy is needed for 6 to 8 weeks. The 1st few treatments is for controlling pain swelling (swell because blood from fractured bone fills the wrist joint)
- Other exercises are used to improve fine motor control and dexterity of the hand.

**Complications:**

1. **Ligament injury** – since it takes such a violent injury to fracture the scaphoid bone, additional injuries to surrounding ligament (scapholunate ligament) often occur along with scaphoid fracture.
2. **Traumatic arthritis** – the patient initially get better until pieces of broken bone loose inside the wrist joint cause deterioration. In this condition, joint become painful and stiff, grip strength decreases.
3. **Avascular necrosis** – occurs due to a much more serious type of non-union whereby lower half of the fractured bone loses its blood supply and actually dies. Below: Avascular necrosis of the proximal pole is evidenced by the collapse of the proximal pole in this chronic non-union.