LECTURE NOTES ON
General Surgery

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Introduction

The ideal medical student at the end of his clinical course will have written his own textbook—a digest of the lectures and tutorials he has assiduously attended and of the textbooks he has meticulously read. Unfortunately few men are perfect, and most approach the qualifying examinations depressed by the thought of the thousands of pages of excellent and exhaustive textbooks wherein lie the wisdom required of them by the examiners.

We believe that there is a serious need in these days of expanding knowledge and expanding syllabus for a book which will set out briefly the important facts in general surgery classified, analysed and as far as possible rationalized for the revision student. These lecture notes represent our own final year teaching; they are in no way a substitute for the standard textbooks but are our attempt to draw together in some sort of logical way the fundamentals of general surgery.

Because this book is written at student level, principles of treatment only are presented, not details of surgical technique.

These notes cover general surgery; ophthalmology and E.N.T. are already dealt with by lecture notes published by our colleagues, Mr Trevor Roper and Mr Miles Foxen, at Westminster Hospital. Orthopaedics will be the subject of a further publication.

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H.E.
R.Y.C.

Contents

CHAPTER 1

Acute Infections

CELLULITIS

Cellulitis is a spreading inflammation of connective tissues. It is generally subcutaneous, but the term may also be applied to pelvic, perinephric, pharyngeal and other connective tissue infections. The common causative agent is the β haemolytic streptococcus.

The invasiveness of this organism is due to the production of hyaluronidase and streptokinase, which respectively dissolve the intercellular matrix and the fibrin inflammatory barrier.

Characteristically the skin is dark red with local oedema and heat. There may be vesicles and, in severe cases, cutaneous gangrene. Cellulitis is often accompanied by lymphangitis and lymphadenitis, and there may be an associated septicaemia.

Treatment
Immobilization, elevation and antibiotics.

ERYSIPELAS

Erysipelas is a diffuse streptococcal infection of the skin and its underlying lymphatics. It is a notifiable disease. The streptococci enter through a minute breach in the skin, though occasionally a wound or burn may be so affected. It particularly occurs on the face and neck. There is intense local pain, the skin is red and raised and there is profound toxaemia.

Treatment
Antibiotic therapy.

ABSCESS

An abscess is a localized collection of pus, usually, but not invariably, produced by pyogenic organisms; occasionally a sterile abscess results from the injection of irritants into soft tissues (for example, thiopentone).

An abscess commences as a hard, red, painful swelling which then softens and becomes fluctuant. If not drained, it may discharge spontaneously onto the surface or into an adjacent viscus or serous cavity. There

are the associated features of bacterial infection; a swinging fever, malaise, anorexia and sweating with a polymorph leucocytosis.

Treatment

An established abscess in any situation requires drainage. Chemotherapeutic agents cannot diffuse in sufficient quantity to sterilize an abscess completely. Pus left undrained continues to act as a source of toxaemia and becomes surrounded by dense fibrous tissue.

BOILS

A boil (furuncle) is an abscess, usually due to the pyogenic staphylococcus, which involves a hair follicle and its associated glands. It is therefore not found on the hairless palm or sole, but is particularly encountered where the skin is hairy, injured by friction, or dirty and macerated by sweat; thus it occurs particularly on the neck, axilla and the perianal region. Occasionally a furuncle may be the primary source of a staphylococcal septicaemia with osteomyelitis, perinephric abscess or empyema, particularly in debilitated patients. On the face it may be complicated by a cavernous sinus thrombosis, via the facial veins.

Treatment

When pus is visible the boil should be incised. Crops of boils should be treated in addition by improving the general hygiene of the patient, ultra-violet light and hexachlorophane baths, but systemic chemotherapy is not indicated.

CARBUNCLES

A carbuncle is an area of subcutaneous necrosis which discharges onto the surface through multiple sinuses. It is usually staphylococcal in origin. The subcutaneous tissues become honeycombed by small abscesses separated by fibrous strands. The condition is often associated with general debility and particularly with diabetes. The urine should always be tested for sugar in this or any other septic condition.

Treatment

Surgery is rarely indicated initially. Chemotherapy is given and the carbuncle merely protected with sterile dressings. Occasionally a large sloughing area eventually requires a skin graft. Diabetes, if present, is controlled.

CHAPTER 2

Specific Infections

TETANUS

Pathology

Tetanus is caused by the clostridium tetani; an anaerobic, flagellated, spore forming, exotoxin secreting and gram-positive bacillus, which is a normal inhabitant of soil and faeces. The organism remains at the site of inoculation and produces a powerful exotoxin which acts upon the motor cells in the C.N.S. and which is probably conveyed along the peripheral nerves directly from the affected part.

Tetanus follows the implantation of spores into a deep, devitalized wound where anaerobic conditions occur. Infection is related less to the severity of the wound than to its nature; thus an extensive injury which has received early and adequate wound toilet is far less at risk than a contaminated puncture wound which has been neglected. Occasionally dressings or catgut which have been contaminated with tetanus spores are the source of infection of surgical wounds. In primitive communities, where dung is used to dress the umbilical cord in the new-born, tetanus neonatorum may occur.

Clinical features

The incubation time is 24 hours to 24 days. Muscle spasm first develops at the site of inoculation and then involves the facial muscles and the muscles of the neck and spine. As a rule it is the trismus of the facial spasm (producing the typical 'risus sardonicus') which is the first reliable indication of developing tetanus. This may be so severe that it becomes impossible for the patient to open his mouth. The period of spasm is followed, except in mild cases, by violent and extremely painful convulsions which occur within 24 to 72 hours of the onset of symptoms and may be precipitated by some trivial stimulus, such as a sudden noise. The convulsions, like the muscle spasm, affect the muscles of the neck, face and trunk. Characteristically, the muscles remain in spasm between the convulsions. The temperature is a little elevated but the pulse is rapid and weak.

In favourable cases the convulsions, if present at all, become less frequent and then cease and the tonic spasm gradually lessens. It may, however, be some weeks before muscle tone returns to normal and the risus sardonicus disappears. In fatal cases paroxysms become more severe and frequent; death occurs from asphyxia due to involvement of the respiratory muscles or from exhaustion, inhalation of vomit, or pneumonia.

The prognosis is serious when the incubation period from the time of injury to the onset of spasm is under 5 days and when convulsions occur within 48 hours of the onset of muscle spasm.

Differential diagnosis

1. Tetany—which characteristically affects the limbs, producing carpopedal spasm.
2. Strychnine poisoning—flaccidity occurs between convulsions whereas in tetanus the spasm persists.
3. Meningitis—because of the neck stiffness.
4. Epilepsy.
5. Hysteria.

Treatment

Prophylaxis

Active immunization comprises 2 injections of tetanus toxoid (formalin treated exotoxin) at an interval of 6 weeks. Booster doses are given annually and at the time of any injury. Toxoid should be given to any population at risk of injury, for example, service personnel.

The efficacy of passive immunization (1,500 units of anti-tetanus serum given intramuscularly) has recently come under severe criticism. There has never been a controlled trial of the value of A.T.S., severe reactions may occur, particularly if the serum therapy has been given in the past, and skin sensitivity tests to a small subcutaneous dose give no reliable guide to subsequent severe reactions. Tetanus may occur even after A.T.S. has been given and at present it seems that the risk of mortality from serum is of the same order as that of an unimmunized subject acquiring tetanus after injury.

Curative Treatment

1. *Control convulsions.* The patient is nursed in isolation, quiet and darkness and is heavily sedated with phenobarbitone or chlorpromazine. In severe cases curarization with tracheostomy and intermittent positive pressure artificial respiration is required and this may have to be continued

for up to 4 weeks. It is terminated when the spasms and rigidity are absent during a trial without relaxants. These serious cases are best transferred to a special respiratory unit.

2. *Control the local infection.* Excision and drainage of any wound is carried out under a general anaesthetic. Penicillin or tetracycline are administered and these will also act as a prophylactic against pulmonary infection.

3. *Maintain the general condition and electrolyte balance* of the patient by naso-gastric tube feeding.

The value of large doses of A.T.S. (100,000 units intramuscularly and 100,000 units intravenously) is not established and its use carries with it the danger of a severe serum reaction. However, toxoid is given if previous active immunization has been carried out.

GAS GANGRENE

Pathology

Results from infection by clostridia welchii, septicum and sporogenes; anaerobic, encapsulated, spore forming, gas-producing, gram-positive organisms which produce an exotoxin. This group includes both proteolytic and saccharolytic organisms. The characteristic gas formation in the tissues is produced by the liberation of CO_2, H_2S and NH_3 by protein destruction. The organisms are found in soil and in faeces.

Typically gas gangrene is an infection of deep penetrating wounds, particularly of war, but sometimes involvement of the abdominal wall or cavity may follow operations upon the alimentary system. Occasionally gas gangrene complicates abortion or puerperal infection.

Clinical features

The incubation period is about 24 hours. Toxaemia is severe with tachycardia, shock and vomiting. The temperature is first somewhat elevated and then becomes sub-normal. The affected tissues are swollen and crepitate due to gas. The skin becomes gangrenous and the infection spreads along the muscle planes, producing at first dark red swollen muscle and then frank gangrene.

Treatment

Prophylaxis

Consists of adequate excision of wounds which removes both the organisms and the dead tissues which are essential for their anaerobic growth. Antibiotics are given in all heavily contaminated wounds.

Curative

In the established case, all involved tissue must be excised. Implication of all muscle groups in a limb is an indication for amputation. Penicillin and blood transfusion are given.

The value of anti-gas gangrene serum both as a prophylactic and curative measure is not established.

ANTHRAX

Caused by the bacillus anthracis which is gram-positive, encapsulated and spore forming. It is a disease of cattle and sheep, which affects men coming into contact with these animals, e.g. leather workers, wool workers and veterinary surgeons. Anthrax is usually a skin infection, but occasionally it is transmitted by inhalation.

The incubation period is 1–2 days with the formation of a 'malignant pustule' which is a central black slough surrounded by vesicles and oedema. Inhalation of the organisms produces a highly fatal pneumonia ('wool-sorters' disease').

Diagnosis is confirmed by examination of a swab from the pustule or the sputum.

Treatment

Chloromycetin is now the treatment of choice although the older remedies of neoarsphenamine and Sclavo's serum may be given in addition in very severe cases.

ACTINOMYCOSIS

Pathology

Actinomycosis is an infection produced by the actinomyces israelii or ray fungus, so called because the mycelial threads may be seen radiating from the main fungal mass in culture. The fungus is micro-aerophilic and exists as a saprophyte in the mouth (especially where there is dental caries) and in the alimentary canal. Infection may occur via a breach in the mucous membrane, for example following dental extraction, and produces a dense fibrous tissue reaction within which pockets of pus develop. The pus contains typical 'sulphur granules' which are yellow specks of mycelium. The infection spreads along the fascial planes and occasionally by the blood-stream but not via the lymphatics.

Clinical features

Actinomycosis can be classified into three main groups: cervico-facial, abdominal and pulmonary.

Cervico-facial

This form occurs typically after dental extraction or tonsillitis. Although actinomyces do grow on grasses and decayed vegetable matter, these varieties are not pathogenic in man and the infection does not occur by chewing contaminated straw. Nor is the disease transmitted from cattle or horses to man. Swelling occurs over the angle of the jaw and the adjacent tissues become greatly indurated. The skin develops a typical bluish discoloration then sinuses appear, which discharge thin pus. Pain may or may not be a feature, but there is usually marked trismus. Spread may occur by direct infiltration to the orbit, base of skull, jaw, or mediastinum.

Abdominal Actinomycosis

Usually located in the ileo-cæcal region, supervening upon an attack of perforative appendicitis, a perforated peptic ulcer or an abrasion of the alimentary mucosa by some foreign body. A hard fibrous mass honeycombed with abscess cavities develops in the right iliac fossa and multiple sinuses may appear on the abdominal wall. Spread may occur via the portal vein producing a portal pyaemia, the liver being riddled with abscess cavities.

Pulmonary Actinomycosis

May follow inhalation of fungus from the infected mouth. Spread occurs through the lung to the pleura and eventually the chest wall. Pulmonary disease may also occur secondary to spread from the neck via the mediastinum, or from the abdominal cavity through the diaphragm.

Treatment

Comprises a 12 week course of daily injections of penicillin, together with Lugol's iodine. Obvious collections of pus should be drained. The actinomyces should be tested for sensitivity and occasionally other antibiotics e.g. tetracycline may be required.

CHAPTER 3

Shock

Shock is the term used to describe a clinical state comprising pallor, sweating, coldness and peripheral cyanosis. The pulse is usually rapid and the blood pressure low. In severe cases there may be dyspnœa, thirst, nausea or vomiting. There may be confusion and restlessness or the patient may be semiconscious.

Aetiology

Shock is produced by a wide variety of circumstances, the common factor being a reduction in the effective circulating blood volume. This clinical picture may be seen in:

1. *Severe haemorrhage*—an actual reduction of blood volume.
2. *Extensive fluid loss* as a result of exudation of plasma from burns, or loss of extra-cellular fluid in severe vomiting or diarrhoea.
3. *The vasovagal syndrome*, produced by severe pain or emotional disturbance. The mechanism of this is reflex vasodilatation in muscle together with vagal cardiac slowing. This syndrome can be recognized because the shock picture is accompanied by slowing of the heart and responds to the simple measure of lying the patient flat with elevation of the legs.
4. *Severe toxaemia*, as in peritonitis or pancreatitis. Here there is a combination of fluid loss, pain and the effect of chemical or bacterial toxins on the heart; once again the mechanics of the shock picture are produced by circulatory failure.
5. *Heart failure* from myocardial infarction or pulmonary embolus.
6. *Sympathetic interruption* which reduces the effective blood volume by widespread vasodilatation; for example, the spinal shock following transection of the spinal cord, or after a high spinal anaesthetic.

The physiological basis of haemorrhagic shock

Severe haemorrhage produces the following chain of events: reduction in blood volume—diminution in the venous return to the heart—fall in cardiac output (Starling's law, the output depends on the degree of stretch of the heart muscle in diastole)—fall in blood pressure—this is counteracted by the carotid sinus and aortic arch reflexes, which increase the heart rate, and by sympathetic vasoconstriction of the splanchnic bed and of the peripheral cutaneous vessels.

This mechanism maintains essential coronary, cerebral and lung blood flow. The blood pressure is thus at first maintained, but continued haemorrhage eventually cannot be compensated and the blood pressure falls.

A continued low blood pressure produces a series of irreversible changes so that the patient may die in spite of later blood replacement. The oxygen lack affects all the vital organs; there may be tubular necrosis of the kidney resulting in renal failure, the adrenals may lose their normal reaction to stress, the heart may fail due to inadequate coronary perfusion and there may be damage to the cardiac and vasomotor centres in the medulla. In the tissues themselves anoxia produces capillary paralysis and dilatation so that a copious fluid loss occurs into the interstitial spaces.

The clinical features of shock which have already been described are thus easily explicable on this physiological basis. The intense peripheral vasoconstriction produces the cold, pale skin. The rapid pulse and low blood pressure are typical features of the impaired cardiac output. The sweating results from sympathetic overactivity. The cerebral disturbances follow inadequate perfusion of the cerebral centres.

Treatment

This depends on diagnosis. A vasovagal attack, or faint, rapidly responds to lowering the head and elevating the legs; if the shock picture does not improve after this, it is suggestive that some complicating factor such as internal haemorrhage coexists with the vasovagal syndrome. It is important to know that considerable loss of blood into the tissues occurs with major fractures of the limbs even if these are not compound.

Where haemorrhage is the cause of the shock the following steps are taken:

1. Further haemorrhage is arrested; this may require direct pressure to a wound or surgical exploration where continued bleeding is the result of a peptic ulcer haemorrhage, ruptured spleen or ruptured ectopic pregnancy.
2. Blood transfusion to replace the blood loss.
3. Relief of pain by means of an injection of morphia where pain is a marked feature.
4. Excessive warmth should be avoided; this produces vasodilatation of the skin vessels, thereby diverting available blood from the vital tissues.
5. Elevation of the foot of the bed is a quick and effective means of raising the blood pressure and is a useful emergency measure.
6. Oxygen is seldom required since the blood is usually fully oxygenated unless there is an associated chest injury or respiratory depression due to a head injury.

Shock from other causes may require appropriate fluid replacement; plasma or plasma substitute in the case of burns, or saline in severe vomiting or diarrhoea. Nor-adrenaline is occasionally valuable in raising the blood pressure but should not be used as an attempted substitute for blood replacement. It should be given through a fine catheter threaded into a major vein since if given into small peripheral veins leakage may produce extensive local vascular spasm and gangrene.

In long continued stress there may be adrenocortical depletion and intravenous hydrocortisone should be administered. In toxaemic patients, for example those with peritonitis, antibiotics are given.

CHAPTER 4

Burns

Pathology

A burn may be partial or full thickness, depending on whether or not the germinal layer of the skin is intact or destroyed.

A partial thickness burn may be quite superficial, with erythema due to capillary dilatation and with or without areas of blistering produced by exudation of plasma beneath coagulated epidermis. The underlying germinal layer is intact and complete healing takes place within a few days. Deeper partial thickness burns extend down to the germinal layer and may partially destroy it. There is intense blistering followed by the formation of a slough. This separates after about ten days leaving healthy newly formed pink epithelium beneath.

Full thickness burns completely destroy the skin. There may be initial blistering but this is soon replaced by a coagulum or slough; more often this is present from the onset in an intense deep burn. Unlike the more superficial burns this slough separates only slowly over three or four weeks, leaving an underlying surface of granulation tissue. Very small deep burns may heal from an ingrowth of epithelium from adjacent healthy skin; more extensive burns, unless grafted, heal by dense scar tissue with consequent contracture and deformity.