THE RECOGNITION AND TREATMENT OF GAS GANGRENE

I. BACTERIOLOGY

A. General Considerations:

Gas gangrene is caused by anaerobic, gram positive bacilli, which are all spore formers (clostridii). There are 25 varieties of anaerobes in the gas gangrene group, but only 4 species are pathogenic for man. Even these are usually harmless saprophytes under ordinary circumstances. They become pathogenic for man only if special conditions are produced in the wounds in which they appear. They produce a powerful exotoxin which is specifically neutralized by the appropriate antitoxin. They may be divided into the following groups:

1) Proteolytic: These digest proteins to form:
   a) Gases - most commonly carbon dioxide, hydrogen, and hydrogen sulphide.
   b) Other chemicals - such as lactic acid, butyric acid, amino acids, and ammonia.
2) Saccharolytic: These ferment carbohydrates to form gas and acid.

The proteolytic organisms decompose muscle, causing it to blacken and producing a putrefactive odor.

B. The common organisms:-

This in gas gangrene are the Cl. welchii, the Cl. of malignant edema (Vibrione Septique of Pasteur), Cl. edematiens, Cl. Sordelle, sporogenes, and hystolyticum. They rarely appear in a pure culture, but usually in a mixed infection. The main tissue damage is produced by the Cl. welchii (present in 90% of cases) and by the Cl. malignant edema (10% of cases). Cl. welchii (saccharolytic) is mainly a gas former. Cl. of malignant edema, edematiens, and Sordelle produce more edema and less gas. Sporogenes and histolyticum are proteolytic and are the main cause of the foul odor. Edematiens also causes a gelatinous necrosis.

The severe tissue damage is caused by the action of the toxin on the tissues, by the further action of the disintegration products of the tissues, and by the pressure produced by the formation of gas. Sporogenes is not pathogenic but increases the virulence of the other pathogens.

C. Source of the organisms - They occur in the intestines of man and animals, in vegetable matter, and they are abundant in cultivated and manured soil. They have been found on surgical instruments, sutures, and commonly on the normal skin. Hence the opportunity for contamination of wounds is great.

D. Wound bacteriology - When the anaerobes of gas gangrene appear in a wound they are usually mingled with other organisms as follows:

1) Anaerobic pathogens.
2) Various non-pathogens.
3) Saprophytic decomposition.
4) Pyogenic bacteria.

The anaerobic pathogens of gas gangrene are carried into the wound at the time of injury. They either kill the patient quickly or rapidly disappear from the wound during the first 7 days. Then pyogenic bacteria (mainly the streptococcus hemolyticus and staphylococcus aureus) become prominent and pus appears in the wound. The pathogenic streptococci and staphylococci (of which the former are the more dangerous) are usually not present at the time of injury; but they are introduced subsequently by improper aseptic technique and from the throats of those attending the patient. Many cases die after the first week from such secondary infection, usually as a result of streptococcus bacteremia.

The mere presence of the anaerobes of gas gangrene in a wound does not necessarily mean that gas gangrene will develop; these organisms are often harmless.

II. CLINICAL PATHOLOGY

Varying clinical pictures, depending upon the predominance of certain organisms, may appear as follows:

1) Marked edematous, suppurative process with or without decomposition.

2) Edema prominent.
3) Non-putrid necrosis with or without gas formation.
4) Foul decomposition and foul smelling gas formation.

The local tissue changes - The favorite site for the development of gas gangrene is muscle (other tissues such as subcutaneous fat are not immune). It becomes discolored, waxy, cecrotic, and finally changes to a pulp saturated with gas bubbles (mainly CO_2 derived from the muscle glycogen). A varying degree of edema appears in the interstitial tissue, which at first is cloudy and then red, due to hemolysis.

Changes ascribed to the circulating toxins:

Vascular: Thromboses in periphery of wound and in smaller vessels throughout the body.
Spleen: Edema, capillary thrombosis, increased hemosiderin content.
Liver: Cloudy, swelling, fatty degeneration, focal necrosis, gas formation.
Kidneys: Cloudy swelling, necrotizing nephrosis, gas formation.

Mode of dissemination of organisms:

1) Direct extension along muscles.
2) Through the lymphatics, to regional nodes, to thoracic duct.
3) By way of the blood stream.

III. FACTORS IN THE PRODUCTION OF GAS GANGRENE

A. The soil:

The bacilli of gas gangrene are usually harmless saprophytes which become dangerous only if a suitable medium is produced for their development.

Especially favorite sites - are deeply lacerated and contused wounds, hematomas, and compound fractures. Disruptive war wounds with a loss of, tearing, and crushing of muscle are extremely liable if neglected or inadequately treated.

B. Destruction of blood supply - creates ideal conditions, as in the case of:
1) Occlusive vascular pathology - in arteriosclerosis, Buerger's disease, diabetes (diabetic gangrene).
2) Vascular injury - as in trauma, burns, surgical excision or secondary occlusive thrombosis.

C. Wounds which are especially susceptible - are those which are inadequately excised, closed by primary suture under tension, and those with imperfect drainage, containing dead tissue into which there has been an extravasation of blood.

Following operative procedures - May follow any operation. In abdominal surgery mainly after gangrenous appendix, gall bladder, intestinal obstruction. Usual site of infection is in the tissues at the periphery of the incision.

D. General predisposing factors - are shock, fatigue, trauma, hemorrhage, exhaustion, cold, and the lowered resistance caused by conditions in the combat zone.

IV. CLINICAL TYPES OF THE DISEASE

1) Infection
2) Infection of a single muscle along its length.
3) Infection of a group of muscles.
4) Infection of a segment of a limb.
5) Fulminating type.

Pseudo gas gangrene:

Cases of subcutaneous cellulitis (as in 1)(above) are seen which locally resemble gas gangrene. These are the common type and they are caused by non-pathogenic, gram negative bacilli and cocci which are not spore formers. They have a slight or no constitutional reaction. They are often seen in patients with diabetic gangrene. The tissues about the gangrenous area are edematous, swollen, crepitant, and contain gas, which is produced by the colon bacillus in the presence of an increased dextrose content in the body fluids, tissues, and blood. Pathologically this is an anaerobic cellulitis of connective tissue and fat, without infection of muscle. It is frequently mistaken for gas gangrene.

The term gas gangrene should be reserved for the type of case in which there is gas production and muscle necrosis.

V. CLINICAL COURSE OF THE DISEASE

May be rapid - 4 to 5 hours after injury a thin brownish, frothy fluid may appear. Edema, emphysema, constitutional reaction advances rapidly; and the patient dies within 24 hours. Between the mild and severe cases all degrees are seen, depending upon:

1) Nature and extent of injury.
2) Sum total of all bacteria present.
3) General resistance of patient.

VI. SYSTEMIC SYMPTOMS

These vary with the virulence of the infection.

Fever - may rapidly approach 103 to 105. In terminal state may reach 108-110.

Pulse - rapidly approaches 120 or more.

There is restlessness and apprehension and often delerium. The patient becomes semistuporous and then may lapse into coma.

VII. DIAGNOSIS

A. Outstanding symptoms - are an abrupt rise in temperature and pain out of all proportion to the character of the wound (usually due to tension in the wound).

B. Appearance time of local physical signs - Usually appear in 1 to 4 days. In fulminating cases in 3 to 4 hours. Average incubation period for gas is 22 hours.

C. Examination of the wound:

Appearance - swelling, edema, redness, acute tenderness. The skin margin at first becomes red, then cyanotic, dark, necrobiotic. The reddened area away from the margin is bronze tinted. The gangrene of the skin margin advances only slowly as the necrosis spreads extensively in the muscle. As the infection spreads the tension may increase, producing numbness in the limb. It may then become cold, clammy, and cyanotic from interference with the blood supply. Vesicles containing edema fluid may appear over the edematous area.

Gentle pressure - on the wound margins usually produces a sanguino-purulent discharge containing bubbles.

Crepitus - reveals the presence of gas. Can sometimes be elicited better by auscultation.

X-ray examination - shows the presence and extent of gas long (24 hours or more) before it is demonstrable on palpation or auscultation. Reveals gas from gas producing organisms on the average 9 hours after injury.

Must differentiate gas from air introduced at time of injury.

Air	Gas
Remains stationary	Increases
Is soon absorbed	Travels along muscle sheaths
	Producing a typical appearance

X-ray examination is recommedded every 4 to 6 hours in suspected cases.

Bacteriological examination: The isolation of anaerobes in the wound has no significance in making the diagnosis nor in determining the results of treatment.

VIII. Treatment

This is mainly surgical. Other measures are merely supplementary. The type of treatment depends upon the general condition of the patient, the extent of the gangrene, presence of shock, hemorrhage, dehydration.

The constitutional symptoms should receive primary and immediate treatment.

Prophylactic treatment

1) Prevent contamination of wound. Aseptic technique and wearing of a mask in treating wounds.
2) Early and complete wound excision. Remove all abnormal tissue, and all tissue the blood supply of which has been interfered with. Remove foreign bodies.
3) Provide aerobic conditions in the wound. This does not mean that the wound must be left exposed to air. If all tissues left behind have a good blood supply, if the walls and contents of the wound consist only of living, pulsating tissues, with normal-colored muscles that contract and bleed freely, the conditions are aerobic and anaerobic bacteria cannot survive.

It may not be possible to achieve this since the avascularity of the tissues may be the result of remote interference with the blood supply.

GAS GANGRENE IS USUALLY THE RESULT OF FAULTY SURGERY

A. Treatment of an established case - This is usually more difficult and uncertain. Complete wound excision irrespective of the time interval since infliction of the wound, if the general condition of the patient permits - even in late cases if the infection remains localized to an area amenable to surgery.

In some late cases because of the extent of the infection and general condition of the patient, may have to limit surgery to debridement (wide exposure and removal of obviously dead tissue, removal of foreign bodies).

In such cases of inadequate excision - zinc peroxide as recommended by Meleny may be of value (see article on tetanus).

Amputation

1) Indicated if a segment of a limb is involved.
2) The site is dictated by the extent of the infection, through normal tissue if possible.
3) May have to operate through tissues already infected.
4) If gas infection develops after amputation, mortality is very high (over 75%). Prevent this by:
   a) Careful preparation of a large area of skin about the amputation site with soap and water.
   b) Careful aseptic technique.
   c) Exclude the gangrenous area from the amputation site by adequate bandaging.
   d) Avoid primary closure (leave the flaps open).
   e) Use guillotine type of amputation.

Note: Whatever surgical procedure is performed in the treatment of gas gangrene wounds, the following principles are obligatory:

1) Rest 2) Adequate and proper compression dressing.
3) Immobilization, and 4) Elevation.

B. Serum Therapy - Not as beneficial as tetanus antitoxin. Its value is questioned by some authorities who have dispensed with its use in favor of adequate wound excision.

1) Prophylactic dose - 5500 units given in a combined serum as follows:
   2000 units Cl, welchii antitoxin
   2000 units Vibrione Septique antitoxin
   1500 units tetanus antitoxin
2) In an established case
   a) Immediate intravenous administration of 30-50 thousand units in 250-500 cc. of glucose solution or physiological saline, slowly by drip method, to combat the toxemia.

b) Observe carefully for anaphylactic reactions. If they appear, discontinue and give adrenalin. Resume injection slowly and with care several hours later.
c) Subsequently - 10,000 units every 4-8 hours if necessary.

C. Sulfonamides - There is considerable disagreement as to their value. They have been used orally and locally into the wound, before and after excision, and also whether the excision has been complete or not. Consequently no, as yet, available conclusions as to their efficacy are valid.

D. X-ray therapy - Kelley and Dowd claim amazing results, and that, combined with adequate wound treatment, the use of serum and amputation are obviated. This view is not shared by most other authorities. Further experience with this method is necessary to determine its true status.

TH EFFICACY OF ADEQUATE SURGICAL TREATMENT OF GAS GANGRENE HAS BEEN AMPLY DEMONSTRATED. THE VALUE OF SERUM, SULFONAMIDES, AND X-RAY ARE, HOWEVER, QUESTIONABLE, AND HAVE BEEN DISCARDED BY MANY AUTHORITIES.

E. General measures -
1) Adequate fluid administration and nutrition.
2) Blood transfusions - to control secondary anaemia, (due to hemolysis of red blood cells by toxins), and to combat toxemic shock.
3) Intravenous glucose (5-10%) - may be of value in protecting the liver which is severely affected by the toxins (jaundice).