TEXTBOOK OF BRITISH SURGERY

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VOLUME FOUR: INFLAMMATION AND PYOGENIC INFECTIONS — BURNS — ACCIDENTAL WOUNDS — INFECTED WOUNDS — ORTHOPÆDICS — DISEASES OF BONE — ARTHROSTEAL TUBERCULOSIS — HÆMATOGENOUS OSTEOMYELITIS AND SEPTIC ARTHRITIS—TUMOURS OF BONE—SURGERY OF THE HIP JOINT—THE KNEE JOINT—SURGERY OF THE HAND—SCOLIOSIS—ANOMALIES OF THE SPINE—PAIN IN THE NECK AND ARM — ANTERIOR POLIOMYELITIS — ORTHOPÆDIC SURGERY IN SPASTIC CONDITIONS — PERIPHERAL NERVE INJURIES — AMPUTATIONS AND ARTIFICIAL LIMBS

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EDITORS' PREFACE

THE advances of Surgery in the last twenty years have been so great that no one individual can master all the fields which they have opened. On the other hand it is important that candidates for the higher examinations should be familiar with the whole subject, for only thus will they be able to select for their future career the branch for which they are best adapted. This new *Textbook of British Surgery* aims to meet their requirements.

It has been compiled by more than fifty authors, each an acknowledged master in his own particular branch, and the aim has been to give a clear and succinct but complete account of the present position in each field. So rapid has progress in surgery been that several of these articles have been completely rewritten while the book was being compiled. At the moment they present an accurate view of surgical practice today on its highest plane. We hope that they will be of material use to the student in acquiring the knowledge which is necessary for his work, and that later on they may inspire him to add to that knowledge and by his own labours to develop still further the great subject to which he is devoting his life.

At the conclusion of the fourth and final volume we should like to take the opportunity of thanking the various contributors whose efforts have made this book possible. In a collaborative work of this kind some delays are unfortunately inevitable and the pace of the entire team is largely determined by that of its slowest member. This must be our excuse to those who rendered their manuscripts promptly and found that it took rather longer to translate them into print than they had anticipated. We should also like to acknowledge our indebtedness of Mr. J. Johnston Abraham and Mr. Owen R. Evans, our publishers, for their wise counsel and helpful co-operation at all times and for the admirable manner in which their firm has produced these volumes.

London, 1959

H. S.
J. C. G.

GENERAL PREFACE

THIS is the fourth and final volume of the *Textbook of British Surgery*. It covers the following subjects: Inflammation and Pyogenic Infections—Accidental Wounds and their Management—Infected Wounds—Acute Fractures and Dislocations—Mal-Union—Burns—General Orthopædics—General Diseases of Bone—Arthrosteal Tuberculosis—Hæmatogenous Osteomyelitis and Septic Arthritis—Tumours of Bone—Surgery of the Hip Joint—The Knee Joint—Surgery of the Hand—Scoliosis—Anomalies of the Spine—Pain in the Neck and Arm—Anterior Poliomyelitis—Orthopædic Surgery in Spastic Conditions—Peripheral Nerve Injuries—Amputations and Artificial Limbs.

Sir Henry Souttar and Professor Goligher as the general editors have in this volume had the assistance of Mr. Norman Capener as editor of the section of growing importance associated with orthopædic conditions, and for his help they are most grateful.

All the authors are recognized authorities on their subjects and each has been allowed a free hand to discuss his line of treatment. Any overlapping has been accepted deliberately. The authors' names are sufficient guarantee that their views are authoritative. They are drawn from leading medical schools and hospitals throughout Great Britain and represent the concensus of opinion in present day British surgery.

The volume is essentially clinical and practical, with such pathology as is necessary for diagnosis and treatment. Surgical procedures are described and the authors discuss the advantages and disadvantages of each procedure in vogue, indicating their reasons for preferring one to another.

The volume is illustrated by original drawings, photographs, X-rays, and diagrams. The audiences it is intended to interest are general surgeons, registrars, postgraduate students and those reading for the Fellowship and other higher examinations.

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CHAPTER I

SECTION I

INFLAMMATION AND PYOGENIC INFECTIONS

IAN GORDON

IN dealing with the lesions grouped under this heading it has been the time-honoured custom to describe as separate conditions, cellulitis, abscess, boils, and carbuncles. That is a matter of convenience for descriptive purposes but it should be clearly understood that each is in fact but a modification in time, place or intensity of the same basic process, namely the reaction of the tissues to invasion by an organism. If the surgeon accepts this truth and if he combines it with a sound understanding of the processes of inflammation and repair, then the intelligent management of patients suffering from pyogenic infections will be firmly in his grasp.

From a study of the salient features of inflammation and repair the surgeon gains an insight into the body's defence against bacteria and can ensure that his treatment will be aimed at helping the defence mechanism and will never impede the formation of or cause damage to the natural barriers set up to resist further invasion. From this study he can evolve certain principles of treatment which are applicable to all bacterial infections, although subject to modification according to the stage of the infection when first seen, the site of infection, the virulence of the invading organism, and the reaction of the host.

INFLAMMATION

This term embraces a series of vascular changes brought about in tissues injured or invaded by an irritant. The process is fundamentally the same whether it is initiated by an injury, a chemical irritant, or bacteria. Here we are concerned with the response to invasion by bacteria. Certain factors modify the response made by the tissues and these will be considered later.

How precisely the series of changes in inflammation are brought about is still controversial, though the changes themselves have been recognized for a hundred years. Of prime importance is the realization that the changes occurring in inflammation are essential to the defence of the tissues and are not in themselves detrimental.

Immediately the bacteria gain a footing in the tissues the small blood vessels of the part dilate and the rate of blood flow through them increases. Capillaries not previously open are brought into action. The number of leucocytes in the blood flowing through the dilated vessels is increased. The plasma is rich in antibodies produced at distant sites (Fig. 1).

To employ a military metaphor, the sole objective of this initial phase is the transportation of the body's troops to the site of invasion. The battle is joined when the defending forces disembark from the blood vessels. The second stage of the inflammatory process is concerned with this disembarkation. The flow in the small vessels slows down and may even become stagnant, the lining endothelium becomes swollen

and the blood cells, both leucocytes and red corpuscles, move from their previous position in the centre of the blood stream towards the vessel wall where they adhere to the endothelium. The leucocytes show amœboid movements and migrate into the tissue spaces, fluid plasma escapes through the walls of the capillaries and even some red cells find their way outside the vessel walls. Although towards the centre of the area of infection the blood may be stagnating, the periphery is still in the first phase or stage of

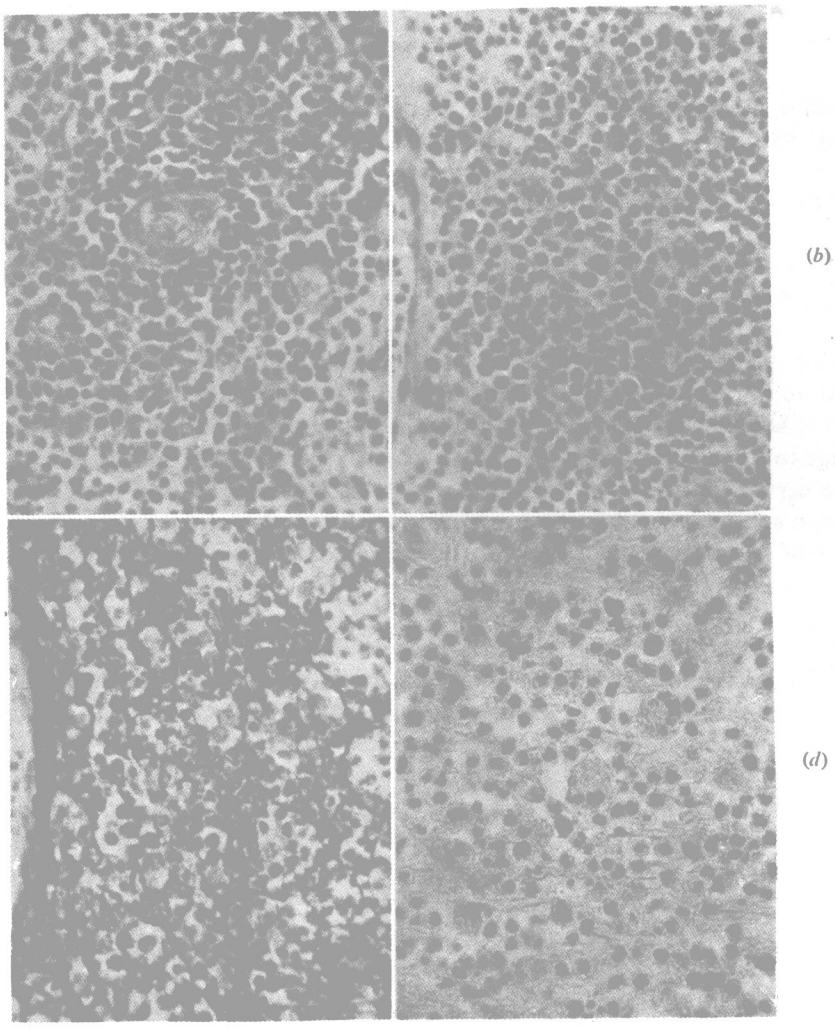


FIG. 1. *Pyogenic Inflammation of the Skin:* the subepidermal zone of a furuncle. Vascularity is increased by opening up of capillaries and the formation of new vessels (*a*). The exudate, which is composed of neutrophil granulocytes and plasma cells, appears to be unsupported and to consist simply of pus (*b*), but silver impregnation reveals a well developed network of young collagen fibres, indicating that the processes of repair are already active (*c*). The earliest obvious evidence of healing is the appearance of phagocytes in the purulent exudate. The darker masses in the macrophages are the nuclei of ingested pus cells, (*d*).

accelerated flow and since many new vessels have opened up the total blood flow through the affected part is much greater than normal. At the site of infection in the tissues the invading organisms are now multiplying rapidly, manufacturing their toxins, and causing the death of the cells in their vicinity. The defence forces have now arrived and their objective is to contain, neutralize, and destroy the invaders. Fibrin contained in the exuded plasma is precipitated in the tissue spaces and thus assists in sealing off the battle zone. The exudate also contains antibodies and opsonins, the former to neutralize the toxins and the latter to render the bacteria easy of phagocytosis. The leucocytes move in and ingest the organisms (Fig. 1 (*c*, *d*)).

In the fully developed process three zones can be recognized, though with the waxing and waning of the battle the zones become intermingled. In the central area the microbes are actively dividing, cellular death of the invaded tissue is occurring and the leucocytes are ingesting bacteria or are themselves being destroyed. This is the zone where pus will form and be recognized by the clinical sign of fluctuation.

Immediately surrounding this zone is an area where the flow in the capillaries has become slow or stagnant, or where thrombosis has already occurred. Here the defending forces are disembarking. Clinically, in a superficial lesion, this is the area of cyanosis, swelling, and induration.

The peripheral zone is the area of dilated vessels with fast flowing stream—a blood stream in which leucocytes are numerous. In this zone the defending reinforcements are being brought to the scene of battle. Clinically this zone is seen as the red blush which surrounds the pyogenic lesion.

Outcome. Such is the start of all pyogenic infections. What follows depends on many variable factors. Should the defences prove overwhelming, the organisms are quickly ingested and all destroyed. The lymph vascular system absorbs the remaining exudate together with any leucocytes and tissue cells which have been destroyed. Such an issue is known as resolution.

Should the invaders at first prove successful the defence is forced to retreat and the involved area constantly extends. The central zone increases in extent with organisms multiplying and spreading in the tissue spaces. The tissue cells are damaged but not necessarily destroyed. This is the state known clinically as cellulitis. The end of this type of infection is resolution over a wide area but with a small central area of suppuration or abscess formation.

If the defenders succeed in localizing the infection but the organisms prove locally destructive then from an early stage suppuration is the outstanding feature and a well defined abscess cavity will form. Sometimes death of tissue en masse occurs and this result is spoken of as bacterial gangrene. It is most often seen in infections of the fingers, though rarely since the use of antibiotics.

REPAIR

Since bacterial invasion frequently results in tissue loss it is appropriate to consider here the process of repair. Once tissue is destroyed the body immediately endeavours to replace it, first by unspecialized tissue and later by specialized tissue approximating as nearly as possible to the original. While the inflammatory process is active in an area where the bacteria are advancing, in an adjacent area, where pus has formed, the abscess wall will show the changes associated with repair. In this latter area granulation

tissue is forming. The newly formed capillaries extend in loops from the tissues in the wall of the cavity, these loops being surrounded by young fibroblasts. The fibrin deposited as part of the inflammatory reaction forms the scaffolding for the support and growth of this new granulation tissue. The objective of the granulation tissue is to fill up the cavity resulting from destruction of body cells and the subsequent evacuation of the pus

As this process advances the older layers of granulation tissue become organized The fibroblasts become re-orientated parallel to the surface of the abscess cavity, i.e. at right angles to the outgrowing tufts of capillaries. Collagen fibres are deposited and scar tissue formed. This tissue serves two purposes, the filling in and eventual healing of the abscess cavity and as a barrier against spread of the invading organisms.

Regional and Systemic Spread of Infection

In addition to the local changes at the point of invasion just described, spread to distant sites may occur.

Spread by Lymphatic Channels. The lymph flow from inflamed tissues is increased. The extent of this increase is dependent in some measure on the muscular activity of the part—the more active the greater the flow. Bacteria and their toxins, which are of relatively high molecular weight, are absorbed by the lymph stream rather than by the blood stream. If the part is immobilized, absorption of toxins is minimized. Clinically this absorption of toxins will produce systemic effects but will give no local sign.

Under certain circumstances, however, organisms will invade the lymph channels and as they are swept in the lymph stream towards the regional lymphatic gland group they may produce an inflammatory reaction in the walls of the channels and even in the surrounding tissues. Once the glands are reached the organisms produce a similar reaction there. These manifestations are known as lymphangitis and lymphadenitis and are marked clinically by the familiar red streak on the skin overlying the affected channel and by the patient experiencing discomfort in the region of the swollen glands.

Blood Stream. In all pyogenic lesions of any size or severity the blood stream is invaded by toxins which produce the general systemic upset. The severity of the resulting disturbance is dependent especially on the type and virulence of the organism, the anatomical site of the lesion and the resistance of the patient.

Other forms of blood spread are of greater significance. In lesions of marked local severity, stagnation of the blood stream in the related capillaries and venules may go on to complete arrest, with formation of clot. If this clot becomes invaded by organisms, then spread of the infection can occur in two ways. First the thrombotic processes may spread rapidly along venous channels and thus infection reach a distant site by direct extension. Cavernous sinus thrombosis and infection may be brought about in this way from a primary focus in the upper lip (Fig. 2). Secondly, infected clot may become detached and be swept into the blood stream as an embolus. This is the condition of pyæmia and it results in metastatic abscesses in sites distant from the original lesion. Metastatic abscesses in the liver following acute appendicitis are brought about as the result of portal pyæmia. Blood culture in pyæmia is usually sterile.

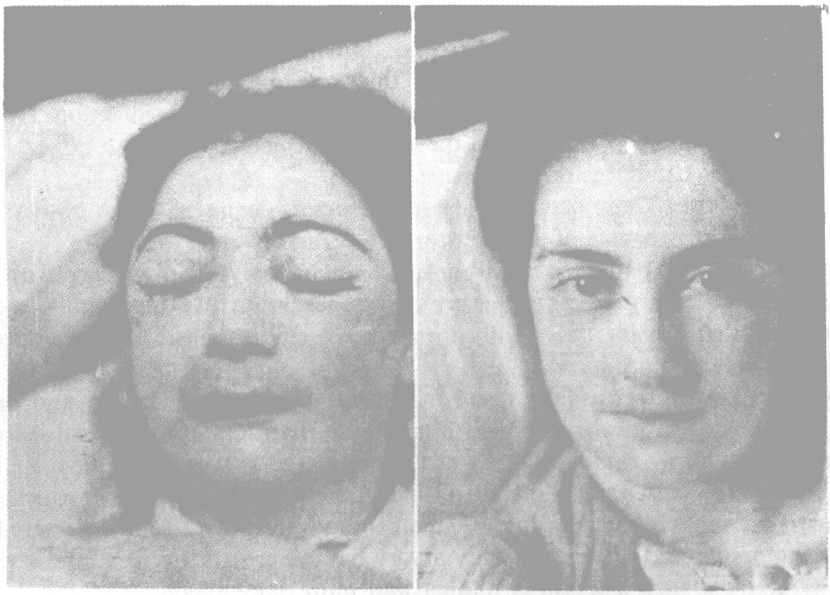
In the related state known as septicæmia, however, blood culture is positive and the invading organisms are found free in the blood stream. It may be that the organisms are actively growing and dividing in the blood stream, or that their numbers are being

constantly augmented by fresh entrants from the primary lesion. The clinical aspects of these conditions will be considered in more detail later.

Factors Modifying the Inflammatory Reaction

Of the many factors which may modify the inflammatory process only the more important will be discussed.

(1) The Resistance of the Host. The patient in excellent general health, surrounded by an environment rich in pyogenic organisms and constantly subjected to minor



(*a*) (*b*)

FIG. 2. *Carbuncle of lip.* (*a*) At time of admission. Patient had a small boil on upper lip. She had squeezed this, rapid extension of infection occurred and cavernous sinus thrombosis resulted from spread along tributaries of the ophthalmic vein. (*b*) Same patient four weeks later. Recovery followed treatment with sulphonamides and penicillin.

traumata, must have a strong resistance to invasion by these organisms. Even if the organisms succeed in invading his tissues he is probably capable of repelling the infection.

On the other hand, if the host's general condition is poor, if he is the subject of a disease such as diabetes or if his defence mechanism is inadequate, for example by lack of antibodies or low polymorphonuclear leucocyte count, then he is at grave risk of being overwhelmed by the invading bacteria.

(2) The Type of Organism. Broadly speaking a certain organism will always tend to produce a specific type of lesion. The final pattern of the lesion will, however, be modified by the general condition of the host, by his degree of immunity to the invading organism and by the virulence of that particular strain. Often the organism involved in a particular infection can be predicted from the situation of the lesion, e.g. coliform bacilli in ischio-rectal abscess, staphylococci in hair follicle infection.

(*a*) STAPHYLOCOCCI. *Staphylococcus aureus* and *Staphylococcus albus* are the commonest of this group and are found on the skin and in the nose. This group of pyogenic organisms is responsible for the majority of soft tissue infections and of cases of osteomyelitis.

Staphylococci all produce one or more exotoxins which stimulate the production of antitoxins. The most consistent association between staphylococci and pathogenicity, however, is the production of coagulase. This causes coagulation of plasma which can act as a barrier against the entry of the agents of tissue defence to the site of growth of the staphylococci.

The staphylococcus, therefore, usually produces a suppurative lesion. The infection tends to localize early but with tissue necrosis. Softening occurs with the discharge of a central core of necrotic tissue, followed by thick creamy pus. The danger of these infections is local tissue damage. Examples of staphylococcus infection are boils, carbuncles, and infections of breast and the palmar surface of hand and fingers. Many forms of staphylococci are resistant to penicillin.

(*b*) STREPTOCOCCI. These occur in infected tissues as chains of Gram positive cocci. They are found in the throats of a proportion of normal people.

The most important pathogen of this group is the hæmolytic *Streptococcus pyogenes*. Like the staphylococci, organisms of this group produce powerful exotoxins and enzymes. Of the enzymes that causing lysis of fibrin may be chiefly responsible for the spreading type of lesion generally produced.

Streptococcal infections usually result in lesions which spread rapidly and produce a diffuse reaction. The local reaction is less intense than in staphylococcal infections. The organisms multiply rapidly and extend along tissue spaces not sealed off by the deposit of fibrin. The lesions included under the terms cellulitis, erysipelas, and lymphangitis are most frequently due to streptococcal infections. Many of these lesions resolve without suppuration or with only a very limited area of necrosis and softening. The discharge from such a lesion is thinner and more sanguineous than the discharge from a staphylococcus lesion. As a rule the danger of streptococcus infections lies in the general systemic upset produced. Fortunately as yet there are no forms of *Str. pyogenes* resistant to penicillin.

(*c*) COLIFORM BACILLI. Among the less common pyogenic organisms are the coliform bacilli which are associated with infection outside the alimentary tract of which they are normal inhabitants. They are commonly found, for example in appendicitis, peritonitis, cholecystitis, and ischio-rectal abscess. Not infrequently also they are found in nail fold infections in nail biters. The urinary tract is another system commonly invaded by the coliform bacilli. The lesions are usually of suppurative type.

(*d*) PSEUDOMONAS PYOCYANEA. This is a pigment producing Gram negative bacillus like the coliforms. It often gives rise to a persistent form of infection in a wound. It seldom occurs alone and is most often superimposed on an existing infection. Due to the production of a pigment the pus has a distinctive greenish blue colour. *Pseudomonas pyocyanea* is not sensitive to penicillin and thus as a causal agent of infection it has been on the increase in recent years. It commonly occurs in burns, urinary infections, and chronic suppuration in the middle ear.

(3) The Time Factor. Time has to be considered not only as judged by the clock but in the sense of the stage reached by a pathological process—what might be called

"pathological time." For example, at one time the view was widely held that in a patient with appendicitis, seen for the first time 48 hours after onset, conservative or expectant treatment should be applied because by that time the inflammatory process would be walled off and any surgical interference would only serve to spread infection. In one case, however, the infective process might be walled off in 24 hours, in another not before 3 days. While in general terms the stage of inflammation is dependent upon the time which has elapsed since the onset of infection, there may be wide variation in the time required to reach a particular stage.

All active infections are at first in a stage similar to what is called clinically, cellulitis. Immediately after onset all the modifying factors come into play, and in one patient a lesion may spread rapidly, localize, and resolve all in a matter of days, whereas in another patient a lesion caused by the same organism, in the same site, may continue for weeks before the infection is overcome.

We think of the common pyogenic organisms as producing acute inflammatory conditions and of an organism such as the tubercle bacillus producing a chronic inflammatory lesion, but the terms acute and chronic simply imply rapid and slow. Under one set of circumstances the pyogenic organisms can produce a slowly progressive and slowly healing lesion, as in chronic osteomyelitis and varicose ulcer. On the other hand, the tubercle bacillus, although usually producing a chronic lesion, may on occasion produce a rapidly spreading one, e.g. tuberculous meningitis.

Broadly speaking, with the passage of time an inflammatory lesion becomes chronic irrespective of the causative organism. Tissue destruction and liquefaction occurs. Granulation tissue forms only to be infected and replaced by a more organized type of fibrous tissue. Thus the lesion becomes more indurated and its walls more rigid. Where pus forms and discharges on the surface, the tract leading from the lesion to the surface has rigid walls lined with infected granulation tissue.

In several respects the exudate in chronic inflammation differs from that in the acute type. Many of the inflammatory cells are of the mononuclear series, lymphocytes, monocytes, and plasma cells. These cells multiply locally and also reach the tissues by migration from the vessels. Macrophages and foreign body giant cells are frequently present in chronic inflammatory lesions, especially where degenerative changes have taken place.

(4) The Site of Infection. Whatever the site of an inflammatory lesion the most important factor in deciding its fate is the power of the natural defence mechanism to contain the infection by setting up an inflammatory barrier around it to destroy and ingest the organisms and to repair and replace the damaged tissue. At the same time the course and ultimate fate of an infection may be influenced by certain anatomical and physiological features of the particular site. An area, clean and with a rich blood supply, though subjected to frequent minor traumata, will seldom be the site of an extensive inflammatory lesion. Such an area is the tongue.

In contrast, avascular fatty tissue near the anus, subject to the repeated trauma of pressure, readily falls a victim to infection. Such conditions exist in the ischio-rectal region.

Areas of skin rich in hair follicles, and in sweat and sebaceous glands, are frequently the site of pyogenic infection by organisms which normally inhabit the area. The normal outlet from such cavities becomes blocked and in the retained secretions the organisms find an opportunity to multiply and invade the tissues.

Once infection is established in a particular site the line of direct spread may be

determined by the anatomical features of the part. As the invaders press home their early advantage they tend to spread along the natural tissue spaces, e.g. where there is areolar tissue and they tend to be turned aside by coming up against a rigid structure such as an aponeurosis.

Here it must be stressed, however, that the inflammatory barrier as it is forced outwards by the advancing organisms, affects all tissues, so that the exudate seals off potential spaces, causes the adhesion of tissues which previously have moved freely in relation to one another and in addition vascularizes, thickens, and softens even rigid structures such as an aponeurosis. Thus, though the pus resulting from the infection may track more readily to the nearest free surface, or spread more rapidly along the surface of an aponeurosis, yet, if given time, it may penetrate even an aponeurosis. For example, pus contained under the periosteum in osteomyelitis may erupt through the periosteum and an abscess in the tracheo-bronchial glands may rupture into the trachea.

Lastly, it is noteworthy that certain structures are more liable to infection when physiologically active. Examples of this are the lactating breast and the growing end of a long bone.

(5) Trauma. The site of infection is frequently determined by a single trauma—an abrasion of the surface, a cut or penetrating injury or a blow, which causes a hæmatoma in the deeper structures without breaking the skin.

Trauma, however, may modify an existing infection. A patient who develops a boil in the eyelids or lip imagines he can shorten the duration of the infection by squeezing the boil when pus has not formed, or if pus is present it is still some distance from the surface. The effect of this trauma is disastrous. The newly formed inflammatory barrier is damaged and there is a rapid spread and intensification of the infection (*see* Fig. 2).

If, however, the trauma is minimal but often repeated, then the effect is less dramatic but none-the-less definite. As the repeated trauma injures the defence mechanism and leads to further spread and continuation of the infection, so more tissue is damaged, more fibrous tissue is laid down and chronicity results.

These effects are well known to every surgeon but few pay heed to the lessons to be learnt from them.

(6) Hormonal. Drugs such as cortisone and its analogues are now in daily use. Cortisone modifies the tissue reaction to irritants and to tissue loss to such an extent that this side effect must be borne in mind when the question arises of administering the drug to a patient who has an acute pyogenic infection or a recently repaired wound.

Clinical Effects of Bacterial Inflammation

Local Effects. The local heat, redness, and swelling are readily related to the vascular and exudative phenomena of inflammation. Pain is a prominent symptom but is not so readily related to the pathology of pyogenic infection. As pain is intensified by any movement or injury to the inflamed tissues, it plays an outstanding part in the defence by compelling rest. The pain arising from an infected area is generally localized accurately to the area involved, it is constant and throbbing in character and is aggravated by movement or injury. It is important to emphasize that pain continues only so long as the infection is active and spreading and once the defence succeeds in localizing the activity of the bacteria the pain subsides. It has been argued that increased tension plays its part in the production of pain—the classical example being infection of the pulp of the

finger. It is often preached and practiced that incision of the inflamed pulp will relieve pain, but if the pulp is incised in the early stages, while the infection is still spreading and before localization with pus formation has occurred, then either no relief is obtained or relief is fleeting.

Tenderness is diffuse in the early phases of the infection, but in late, well localized conditions it is usually confined to the area of fluctuation. Extension of the area of tenderness even beyond the confines of the erythematous area is evidence of spread, while shrinkage of this area is an indication of localization.

Constitutional Effects. In addition to the local effects there are certain constitutional changes which occur simultaneously with the local reaction to invading bacteria. These changes fall into two groups, firstly those subserving the primary function of defence, and secondly, those which are due to poisons absorbed from the local lesion.

PYREXIA. Pyrexia is usual in inflammatory conditions and may even be part of the defence mechanism. The high temperature is due to increased metabolism in the tissues and to reduction in loss of heat by the skin. The loss of heat by the skin is under the control of a heat regulation centre and in fevered states this thermostatic control is set for a higher level than normal.

LEUCOCYTOSIS. The rise in the number of polymorphonuclear leucocytes circulating in the blood in most pyogenic infections is necessary to provide the large number required at the site of bacterial invasion. This leucocytosis must be brought about by stimulation of the bone marrow, but whether by the circulating bacterial toxins or by some other substance produced by the damaged body cells is not known.

PRODUCTION OF ANTIBODIES. The question of antigens and antibodies cannot be fully discussed here, but a word about the production of antibodies is necessary. In microbic infection the bacteria and their toxins act as antigens and stimulate the production of antibodies. The antigens are prepared or modified by the cells of the reticulo endothelial system while the actual production and transportation of the antibodies is the function of the plasma cells and lymphocytes. The lymph glands regional to infection are specially active in the production of antibodies. In response to infection antibodies are soon to be found in the blood, lymph fluid and inflammatory exudate.

Antibodies can act in the body by neutralizing toxins, by opsonic effect and by bacteriolysis. Precipitation and agglutination, which are produced by antibodies reacting with antigens in test tubes, are of doubtful significance in the body.

TOXÆMIA. As generally employed this term denotes a group of non-specific symptoms and signs which we associate with any infection and which is produced chiefly by the bacterial endo-toxins. Pyrexia, increased pulse rate, headache, pains in the limbs, nausea, vomiting, and delirium may all occur.

If this condition persists for any length of time, or if it is severe from the onset, the bacterial toxins may damage the parenchymatous cells of the liver, kidneys, heart, and other viscera, producing "cloudy swelling" or "fatty degeneration." Amyloidosis is a less common result of long-standing bacterial infection.

Principles of Treatment

The principles that should guide the surgeon in the management of patients suffering from pyogenic infections are the promotion of the natural defence mechanism in all its phases and the avoidance of any treatment which harms it.

General Treatment. The vast majority of pyogenic infections can now be treated on an ambulatory basis. Few require any very strict regime. It is obvious that if the patient is debilitated, is suffering from a definite systemic disease such as diabetes or anæmia, or has marked systemic effects arising from his infection, suitable counter measures must be adopted.

Chemotherapy is now an essential part of the treatment of many pyogenic infections. In view of the increasing number of strains of organisms resistant to the antibiotics, especially penicillin, the indications for the use of antibiotics should be given careful consideration. There has been a tendency in the immediate past to administer penicillin in even the simplest of pyogenic infections, infections which in the past were adequately cared for by the unaided natural defence mechanism. None the less, when the infection is giving rise to considerable systemic upset, is locally severe, or by reason of its position is threatening function, the antibiotics should not be withheld. Until the invading organism, its type, and antibiotic sensitivities can be determined by culture of the discharge from the presenting lesion, treatment should be instituted by the administration of penicillin. On the other hand, if by reason of the site of the infection or the nature of the discharge, the presence of an organism other than one known to be sensitive to penicillin is suspected, the appropriate antibiotic should be administered.

Most pyogenic infections give rise to considerable pain and sedation is, therefore, an essential item in treatment.

Local Treatment. The first phase of defence is aimed at increasing the blood supply to the area invaded by organisms. All local treatment should at first be directed towards promoting this increased vascularity. The application of heat and splinting of the part can best achieve this. The value of a poultice of the kaolin type is that it provides both heat and rest to the part.

Because tourniquets and local anæsthetics diminish the blood supply to an infected area their use should be limited.

The initial phase of increased vascularity is followed by the phase in which the infection is localized by the building up of an inflammatory barrier. In this phase, rest is of paramount importance. To avoid movement and trauma the part should be splinted and in the upper limb a sling is invaluable. It is in this early phase that all attempts to express small beads of pus from the centre of an indurated inflammatory lesion should be scrupulously avoided.

Surgical Measures. The scalpel has no place in the treatment of early, non-localized, pyogenic infections. By early incision of an inflammatory area where no stable localization has occurred, the surgeon will only damage the natural defensive barrier, allow invading organisms a quick entry to tissues not yet mobilized for defence, and by the production of a wound make new demands on an already over-strained blood supply.

The only regular exception to this rule is where the infection is situated in an organ which can be readily dispensed with in its entirety—viz. the appendix. Tiny lesions in other parts could theoretically be treated by excision, but if left alone nature will cope with these with much less loss of tissue and with far less disturbance to both patient and surgeon. At one time excision of such a lesion as a carbuncle was advocated, but this resulted in very considerable loss of tissue and later gross scarring and contracture.

Once the natural defences, aided by chemotherapy, heat, and rest, have succeeded in localizing the infection and when there is definite clinical evidence of pus formation,