#### MACFARLANE AND THOMAS

# Textbook of Surgery



E. & S. LIVINGSTONE LTD. EDINBURGH AND LONDON

## TEXTBOOK OF SURGERY

EDITED BY

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WITH A FOREWORD BY

NORMAN C. TANNER

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1964

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#### TEXTBOOK OF SURGERY

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#### FOREWORD

In such a live science and art as surgery, new ideas, technical innovations and the need to discard discredited concepts all demand a selective but voluminous output of medical literature. This in turn leads to a demand for the steady production of new editions of the standard textbooks or of new books. For the student, and particularly the undergraduate student's textbook, it is unnecessary to reflect every passing surgical fashion or to be in the vanguard of surgical thought. The student reader, with his ever more crowded curriculum, requires the help of writers who can provide a readable and sound basis of surgery, in a book which will also function as a reference book. Such a book must be firmly pruned of outworn or discarded ideas and should not contain the minutiae of technique required by the specialist.

It is surprising that some textbooks have succeeded in achieving these ends, and at the same time retained a freshness of outlook from one edition to another. Unfortunately, however, old favourites for one reason or another may eventually lose their appeal and effectiveness as teaching agents, and so it is desirable that entirely new books should be produced to take their place. The student in the mass is the one who in the final count will judge the teaching value of the book.

This book is an entirely new production and the editors and writers are surgeons who are practised in undergraduate and postgraduate student teaching. They are young enough to remember the problems they encountered as students and to have the physiological foundations of modern surgical treatment foremost in their minds.

It is hoped that this book will act as the student's friend, not only when he is confronted with classical surgical conditions in his final clinical examination, but also when he is at the bedside or in the out-patients' department.

Above all it is hoped that by its variations in emphasis, it will reinforce the efforts of the clinical teachers to impart a balanced and sound surgical judgment to the student.

NORMAN C. TANNER.

#### PREFACE

It is a well-recognised but regrettable fact that a considerable number of medical students do not possess a textbook of surgery. The cause may lie in more extensive use of library facilities or it may be related to the increasing cost of book production. The appearance of many excellent monographs on special subjects may be a further reason for failure to own such a book. It is desirable that a composite general volume should be available for reference and study on all occasions. A plea frequently made to one of us for a suitable book published at a moderate figure has been the reason for the initiation of this particular work.

The aim has been to provide surgical information in a concise and didactic form that will enable the student to both understand and retain the essentials in logical sequence, and thereby provide a sound basis for future surgical practice. In this respect the qualified practitioner or postgraduate student may find the book a satisfactory introduction to further study.

Consideration of how this aim could best be achieved led us to the belief that homogeneity and awareness of student requirements were essential. In consequence the number of contributors was kept small, all were actively engaged in undergraduate teaching in a number of different medical schools, and all were of a seniority which was sufficiently near to that of the student to understand his needs. Frequent discussion by members of the team and exchange of ideas in a friendly, lively and constructive manner, are considered to have been of benefit to the final form. The style and composition have been adapted in an attempt to produce the desirable conformity which a single-author book possesses. Ease of reading rather than tabulation has been an aim, and technical operative detail has been largely omitted as this field is considered to be one for postgraduate study.

The text covers the accepted field of general surgery, and the specialties of ophthalmology and oto-rhino-laryngology have been omitted. It is maintained that fractures and orthopaedic surgery not infrequently come within the province of the general surgeon, and that it would therefore be an advantage to include these features.

It was further considered that student needs were best provided by illustrations consisting of line and shaded drawings. These viii PREFACE

emphasise important practical points and it is hoped they will enable the retention of the essential aspects. A further advantage is to reduce the cost of production.

It is a pleasure to record our thanks to those who have played a part in this undertaking. Mr Norman Tanner, with whom one of us was formerly associated, has shown great interest in the work and offered helpful criticism. He has previously influenced our writing and much of our surgical thought, and for this and for kindly consenting to write a foreword, we are greatly in his debt. In the earlier phases the late Professor Lambert Rogers gave us constant encouragement. We should like to take this opportunity to pay our respects and record the appreciation of two of his students. Our contributors have fully co-operated in an enthusiastic and industrious manner and their loyal support has been a stimulation without which this book could not have been completed. Mr Adrian Fox, F.R.C.S., has been responsible for compiling the index and has dealt with this important section in a careful and assiduous manner. He has also read and assisted in the correction of all proofs and we are grateful to him for his meticulous attention to detail. Miss Valerie Sweet, our artist, has drawn all the illustrations and we are indebted to her for the high standard of her skill and for her co-operation in a long and laborious task. The complete book has been typed by Miss Linda Rogers, whose help and efficiency have contributed in no small manner to its completion. Messrs E. & S. Livingstone have been indulgent and patient publishers and we are greatly appreciative of the understanding and advice which we have received from the managing director, Mr Charles Macmillan. Lastly we wish to express our thanks to our wives for their patience and encouragement throughout the production of this book.

London, 1964

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#### CHAPTER I

#### SURGICAL ASPECTS OF INFECTION

#### **INFLAMMATION**

INFLAMMATION is the response of living tissues to injury. Although usually bacterial in origin, comparable local tissue changes result from physical or chemical trauma, including wounds, irradiation injury, frostbite and thermal or chemical burns.

#### Local Changes in Inflammation

These were described by Celsus in the first century A.D. in his aphorism "turgor, dolor, rubor, calor," and swelling, pain, redness and heat form these local features together with loss of function of the part.

The inflammatory response, as revealed by studies of translucent tissues such as the foot-web of the frog or the ear chamber preparation in the rabbit, comprises dilatation of and stasis within the small blood-vessels, margination, and finally migration of polymorph leucocytes into the surrounding tissues. A severe inflammatory process may also be accompanied by the migration (diapedesis) of red blood corpuscles through the vessel walls. This vascular dilatation in the inflamed area accounts for the redness and increased heat; the local swelling is produced by exudation of plasma into the injured tissues, due to increased capillary permeability and filtration pressure.

The mechanisms of the vascular changes have been explained to a considerable extent by the work of Lewis. Vessel dilatation results partly from the liberation of a histamine-like chemical from injured tissues (the "H-substance" of Lewis), and partly from an axone reflex, the latter accounting for the red "flare" which develops around the traumatised area. This axone reflex is abolished in skin which has been denervated and in which the nerve fibres have degenerated. The mechanism of the pain is less well understood; it may be related to the release of nervestimulating substances from the damaged cells, changes in tissue pH, or to the stretching of nerve endings in the inflamed and swollen tissues, as suggested by the almost immediate relief of pain on incising an abscess.

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The migration of polymorphs into inflamed tissues has been investigated by Menkin, who showed that a polypeptide may be extracted from the inflammatory exudate which will induce this cell migration on injection into normal tissues. Other fractions obtained from inflamed tissues will reproduce the pyrexia and leucocytosis which are the well-known systemic manifestations of the inflammatory process.

#### **Bacterial Infections**

Only a few of the vast number of bacteria cause injury to man. Their effect is the result of the production by these organisms of toxins, either from breakdown of the bacteria (endotoxins) or from an actual secretion of toxin into the tissues or blood (exotoxins). The clinical picture and the course of an infection depend on the nature of the bacteria and on the resistance of the host. The latter is related to the general condition, the natural or acquired immunity and the presence of antibiotics in the tissues. Certain organisms, such as the staphylococcus, produce an intense local infection and disseminate only infrequently; others, such as Clostridium tetani and Corynebacterium diphtheriae, remain at the site of inoculation but produce widespread effects by means of their exotoxins. The  $\beta$ -haemolytic streptococcus is very invasive and tends to spread rapidly in the tissues, the lymphatics and the blood stream.

#### The Defence Mechanisms of the Host

These include the intact epithelia of the skin and of the respiratory, alimentary and urinary tracts, the local tissue reactions of acute inflammation and immunological defences. Chemotherapeutic agents can assist the natural defence mechanism.

1. The Intact Epithelia. It is improbable that the completely intact skin can be invaded by an organism, but a small and unnoticed abrasion may be a sufficient portal of entry. The skin possesses an active as well as a passive defence; constant shedding of squames and flushing with sweat remove organisms, whilst the highly acid skin gland secretions kill bacteria. It has been shown that haemolytic streptococci disappear after several hours when inoculated on to skin, although they persist in control smears on glass. The alimentary tract is guarded by the acid barrier of the stomach, but despite this the virulent organisms of typhoid, dysentery and cholera can reach the vulnerable intestine. The

lower respiratory tract is protected from organisms by a mucous stream propelled upwards by cilial action which continuously removes invading bacteria.

2. The Local Tissue Reactions of Acute Inflammation. The inflammatory process constitutes an important means of containing and destroying invading organisms. The clotting of tissue exudate produces a fibrin barrier to the spread of bacteria. This is of particular importance in serous membranes, and is well demonstrated in the fibrinous adhesions which form around an acutely inflamed appendix.

Streptococcus haemolyticus and Cl. welchii both secrete an enzyme, hyaluronidase, capable of depolymerising the long chains of hyaluronic acid, a mucopolysaccharide which is an important component of intercellular ground substance. By this means both these organisms are capable of widespread tissue dissemination. The streptococcus also produces streptokinase which breaks down the fibrin barrier produced by the tissues in response to infection. Actual destruction of invading organisms is partly due to immunological effects of the tissue exudate and partly to phagocytosis of bacteria by the polymorph leucocytes, which invade the inflamed tissues through an increasedly permeable capillary wall.

3. Immunological Defences. The body reacts to foreign protein by the production in the reticulo-endothelial system of specific antibodies contained in the globulin fraction of the blood plasma. Within a few days of infection antitoxins, precipitins and agglutinins can be detected in the blood and in the inflammatory In many bacterial and virus infections previous inflammation will produce complete or relative immunity due to the presence of residual antibodies in the blood. Immunity, which may be active or passive, can also be induced artificially. Active immunisation makes use of killed organisms such as T.A.B. vaccine for typhoid, attenuated living organisms as in B.C.G. vaccine for tuberculosis, or inactivated toxins called toxoids as exemplified by formalin-inactivated tetanus toxoid. Passive immunisation makes use of the antiserum produced by injecting the organism or toxin into an animal, for example, tetanus and diphtheria antitoxins. An immunological mechanism is necessary for phagocytosis to occur. It can be shown that polymorphs washed free from plasma are unable to ingest organisms; these must be coated with specific opsonin before phagocytosis can occur. Opsonins are present in

small amounts in serum as a result of previous infection, and their titre rises rapidly on fresh bacterial invasion.

Chemotherapeutic agents may considerably modify the defence mechanisms of the body. This can be achieved by bacteriostatic agents, which by preventing the multiplication of invading organisms enable the body to deal with the bacteria by phagocytosis and antibodies, or by bactericidal agents which act by destroying the organisms. The sulphonamides, chloramphenicol and the tetracyclines are examples of the first group, and penicillin, erythromycin and streptomycin are examples of the second. Sulphonamides and antibiotics are dependent on an adequate blood supply to carry them to the affected tissues, and are highly efficient in dealing with sensitive organisms free in the blood stream as in septicaemia, the tissues as in cellulitis or pneumonia, or the serous surface such as in diffuse pleurisy or peritonitis. Where bacteria are walled off within slough or in an abscess cavity chemotherapeutic agents can only penetrate by slow diffusion, and in addition the sulphonamides are inhibited by the action of pus. The contained organisms are thus unlikely to be destroyed, the mass of dead and infected tissues will persist as an irritant to surrounding structures, and absorption of breakdown products will continue to cause toxaemia. This difference is of considerable practical importance; while bacteria are in the invasive and free phase of a cellulitic or septicaemic process chemotherapeutic agents are extremely valuable and have revolutionised the course of such illnesses, but when abscess formation has occurred chemotherapy becomes of little value and surgical drainage will be needed in most instances.

In addition to such specific mechanisms there are certain general factors which are operative in the defence of the host. These include the age of the patient, the nutritional state, the presence of anaemia, fatigue or protein deficiency and the occurrence of severe systemic disease such as diabetes or nephritis. In any of such associated conditions and in the extremes of age infection is more likely to be serious.

#### The Bacterial Response

In an effort to overcome the defence mechanism of the host, the following factors in the bacterial attack are important.

1. The Size of the Bacterial Invasion. It can be shown experimentally that increasing doses of a particular strain of

bacteria produce a corresponding increase in mortality in animal hosts. In practice it is not feasible in the course of a long operation to prevent pathogenic organisms from entering the wound, but ill-effects are only encountered when a sufficiently large number of bacteria contaminate the wound.

- 2. The Virulence of the Bacteria. This may be measured as the capacity to produce disease and consists of two components: (a) toxicity, the power of the organism to injure the tissues of the host; and (b) invasiveness, the ability to attack and spread in the body. There is considerable variation in the degree of virulence in different bacterial species and in varying strains of the same species. Dissimilar strains of Clostridia have different lethal doses in experimental animals, and there is evidence that penicillinresistant staphylococci may be more aggressive than sensitive forms. Organisms may be highly lethal although their invasiveness is low. Cl. tetani, despite remaining localised at the site of inoculation, produces serious effects by the formation of an exotoxin.
- 3. The Environment. Bacteria become established only with difficulty in healthy tissues with an abundant blood supply. Clostridia only thrive in anaerobic conditions, and inoculation of the most virulent strain of gas gangrene organisms on a healthy, vascular granulating surface has no effect. In contrast, haematomas, necrotic tissue and serous collections all form an excellent nidus for bacterial infection.

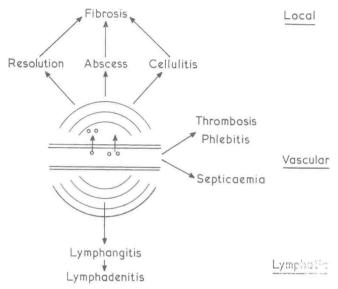
#### ACUTE INFECTION

#### Cellulitis

Cellulitis is a spreading inflammation of connective tissues. The term usually refers to subcutaneous infection, but may also be applied to pelvic, retroperitoneal, perinephric, pharyngeal or intra-orbital connective tissue infection. The common organism is the  $\beta$ -haemolytic streptococcus which usually gains entrance through a scratch or prick. The skin becomes dusky red around the site of inoculation and there is local oedema, heat and severe pain. Vesicles may appear in the involved area of skin, and in advanced cases there may even be cutaneous gangrene.

Cellulitis is frequently accompanied by *lymphangitis* and *lymphadenitis*, red streaks being seen along the lymph channels

and the regional glands becoming enlarged and tender. Not uncommonly there is an associated *septicaemia* which originates either from a septic thrombophlebitis in the affected area or from spread of bacteria from the lymphatics to the blood stream by way of the thoracic duct (Fig. 1). The spreading invasiveness of *Str. pyogenes* is due, at least in part, to its ability to produce



 $$\operatorname{Fig.}$\ 1$$  The course and complications of acute infection.

hyaluronidase which dissolves the intercellular matrix, and a fibrinolysin called streptokinase which is able to destroy the fibrin inflammatory barrier to bacterial spread.

**Treatment.** Immobilisation of the affected part is essential and may necessitate bed rest. Antibiotics will often resolve an infection. Local heat is frequently soothing and can best be achieved by short-wave diathermy which does not macerate the skin. Kaolin poultices and hot fomentations are sometimes used if this is not available. Incision is required to evacuate a localised collection of pus.

#### Erysipelas

Erysipelas is a diffuse streptococcal infection of the skin and its underlying lymphatics. It is a notifiable infectious disease. The

organisms may enter through a wound, burn or ulcer, but usually invade through a minute breach in the skin. The disease is usually encountered on the scalp, face and neck, the streptococci spreading from the nose or sinuses. Pain, stiffness and redness of the skin are noted and the area is raised, this often being better felt than seen. The margin is irregular and vesicles may form on the surface. When infection has subsided the area often becomes pigmented. Pyrexia and toxaemia usually accompany the local signs. Resolution of the infection generally follows antibiotic therapy.

#### Abscess

An abscess is a localised collection of pus and is usually, but not always, the result of invasion by pyogenic organisms. Polymorphs ingest and kill these organisms, and in dying liberate enzymes which liquefy the resulting dead tissue. The pus becomes surrounded by a "pyogenic membrane" of living polymorphs embedded in fibrin and granulation tissue, which in turn is contained by a zone of intensely hyperaemic tissue infiltrated with polymorphs. Septic thrombosis occurs in adjacent small bloodvessels, and the detachment of fragments of infected blood clot accounts for the septic emboli which may complicate a relatively insignificant abscess.

An abscess may result from a number of causes. There may be direct implantation of organisms, as occurs in the infection of a clean surgical incision, or that which results from inoculation of the subphrenic spaces with bacteria after perforation of a peptic ulcer. Lymphatic spread of infection may occur to regional nodes which can then undergo suppuration. Haematogenous spread may take place from even a small septic focus such as a boil. This is exemplified in osteomyelitis or in the production of a perinephric, cerebral or lung abscess. Pus formation is not entirely dependent on bacterial invasion, and may occur wherever there is intense local tissue death; thus inadvertent injection of caustic fluids, thiopentone or other irritants into the soft tissues will result in an abscess, the contents of which are sterile. Clinically an abscess first manifests itself as a hard, red, painful swelling which later softens and becomes fluctuant. If not drained at this stage it may discharge spontaneously through the skin or internally into a viscus or serous cavity. There are the associated features of bacterial