

Harold Ellis
Roy Yorke Calne
**Lecture Notes on
General Surgery**

Fifth Edition, Second Printing

1899
~~I.H.E.~~
LECTURE NOTES ON

General Surgery

R6-42 E47No.5

HAROLD ELLIS

M.A. D.M. M.Ch. F.R.C.S.

Professor of Surgery
Westminster Medical School, London

&

ROY YORKE CALNE

M.A. M.S. F.R.C.S. F.R.S.

Professor of Surgery
University of Cambridge

FIFTH EDITION.



BLACKWELL SCIENTIFIC PUBLICATIONS

OXFORD LONDON EDINBURGH MELBOURNE

1965, 1968, 1970, 1972, 1977
© Blackwell Scientific Publications
Osney Mead, Oxford OX2 0EL
8 John Street, London WC1N 2ES
9 Forrest Road, Edinburgh EH1 2QH
P.O. Box 9, North Balwyn, Victoria, Australia

All rights reserved. No part of this publication
may be reproduced, stored in a retrieval system,
or transmitted, in any form or by any means,
electronic, mechanical, photocopying, recording
or otherwise without the prior permission of
the copyright owner.

FIRST PUBLISHED 1965
REVISED REPRINT 1966
SECOND EDITION 1968
GREEK EDITION 1968
THIRD EDITION 1970
FOURTH EDITION 1972
REPRINTED 1974
REVISED REPRINT 1976
FIFTH EDITION 1977
REPRINTED 1979

British Library Cataloguing in Publication Data
Ellis, Harold, b.1926
Lecture notes on general surgery. — 5th ed.
I. Surgery
I. Title II. Calne, Roy Yorke
617 RD31
ISBN 0-632-00496-7

Distributed in U.S.A. by
Blackwell Mosby Book Distributors
11830 Westline Industrial Drive
St. Louis, Missouri 63141
and in Canada by

Blackwell Mosby Book Distributors
86 Northline Road, Toronto
Ontario, M4B 3E5

Printed and bound in Great Britain
by Billing and Sons Limited,
Guildford, London and Worcester

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 widening knowledge and expanding syllabus for a book which will set out briefly the important facts in general surgery which are 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 attempts 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 ENT are already dealt with by lecture notes published by our colleagues, Mr Trevor Roper and Mr Miles Foxen, at Westminster Hospital.

The need for a 5th edition has enabled us to carry out a detailed revision of the whole text and to add a number of new illustrations.

HAROLD ELLIS
ROY YORKE CALNE

Westminster Medical School, London
Addenbrooke's Hospital, Cambridge
1977

Acknowledgments

We are grateful to our colleagues—registrars, housemen and dressers at Westminster Hospital—who have read and criticized this text during its production and to many readers and reviewers for their constructive criticisms.

We have received particular help in this edition from Mr John Gleave FRCS and Mr Ben Milstein FRCS of Addenbrooke's Hospital, and Mr Leslie Oliver FRCS of Westminster Hospital in revising the sections on thoracic surgery and neurological surgery. We thank Professor R.G. Harrison for permission to use Figures 23 and 28 which are taken from his *Textbook of Human Embryology*.

Finally, we should like to acknowledge the great and continued help given by Mr Per Saugman and his staff at Blackwell Scientific Publications.

H.E.
R.Y.C.

Contents

Introduction	vii
Acknowledgments	viii
1 Acute Infections	1
2 Specific Infections	4
3 Shock	10
4 Burns	13
5 Post-operative Complications	18
6 Tumours	29
7 Chest and Lungs	33
8 The Heart and Great Vessels	47
9 The Peripheral Arteries	62
10 The Veins of the Legs	77
11 The Lymph Nodes and Lymphatics	83
12 The Brain and Meninges	86
13 Head Injuries	97
14 The Spinal Cord	110
15 Peripheral Nerve Injuries	121
16 The Mouth and Tongue	128
17 The Gums and Jaws	141
18 The Salivary Glands	144
19 The Oesophagus	150
20 The Diaphragm	159
21 The Stomach and Duodenum	165
22 Mechanical Intestinal Obstruction	187
23 Specific and Special Forms of Obstruction	193

24	The Small Intestine	202
25	Acute Appendicitis	206
26	The Colon	212
27	The Rectum and Anal Canal	225
28	Peritonitis	240
29	Paralytic Ileus	249
30	Hernia	252
31	The Liver	263
32	The Gall Bladder and Bile Ducts	281
33	The Pancreas	291
34	The Spleen	300
35	The Kidney and Ureter	303
36	The Bladder	322
37	The Prostate	329
38	The Male Urethra	340
39	The Penis	343
40	The Testis	347
41	The Neck	357
42	The Thyroid	360
43	The Parathyroids	375
44	The Adrenal Medulla	378
45	The Adrenal Cortex	380
46	The Thymus	384
47	The Breast	386
48	The Skin and its Adnexae	401
49	Transplantation Surgery	420
	Index	426

CHAPTER 1

Acute Infections

There is an important general principle in treating acute infections anywhere in the body: antibiotics are invaluable when the infection is spreading through the tissues (e.g. cellulitis, peritonitis, pneumonia), but drainage is essential when abscess formation has occurred.

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. If a local abscess forms, this must be drained.

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, although 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 toxæmia.

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 body 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 toxæmia 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 usually 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 septicæmia and be responsible for 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. Recurrent crops of boils should be treated by improving the general hygiene of the patient, and by the use of ultra-violet light and hexachlorophene 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 excision and a skin graft. Diabetes, if present should be controlled.

CHAPTER 2

Specific Infections

TETANUS

Pathology

Tetanus is caused by the clostridium tetani; an anaerobic, flagellated, exotoxin-secreting and gram-positive bacillus, which forms a characteristic terminal spore ('drumstick'), and 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 CNS and which is probably conveyed along the peripheral nerves directly from the affected part.

Tetanus follows the implantations 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', or 'lock-jaw' to the layman) 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 24-72 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 two initial injections of tetanus toxoid (formalin treated exotoxin) at an interval of 6 weeks. Booster doses are given at intervals of not more than 7 years, or 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 (1500 units of anti-tetanus serum given intramuscularly) has recently come under severe criticism. There has never been a controlled trial of the value of ATS, severe reactions may occur, particularly if 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 ATS 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. Human gamma globulin from fully-immunized subjects is now becoming available and is theoretically ideal for passive protection.

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 period 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 ATS (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. Human gamma globulin is safe, effective, but is at present in short supply.

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 amputation of an ischaemic lower limb, or follows 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. Penicillin is given in all heavily contaminated wounds and to patients with atherosclerosis undergoing amputation of the leg.

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. Hyperbaric oxygen therapy, to eliminate the anaerobic environment, is theoretically sound but as it is combined with all the other modalities of treatment, its efficacy cannot be judged. If a hyperbaric chamber is available, it should certainly be employed.

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

A vaccine is available for workers at risk of anthrax. Penicillin in large doses is now the treatment of choice, and since the antibiotic era, the mortality is under 1 per cent.

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, as was once taught, 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-caecal region and follows 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, honey-combed 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. 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 dyspnoea, thirst, nausea or vomiting. The patient may be confused and restless or be semi-conscious.

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 toxæmia*, as in peritonitis, septicaemia (particularly gram-negative organisms) or pancreatitis. Here there is a combination of fluid loss into the extravascular space, pain and the effect of chemical or bacterial toxins on the heart; once again the shock picture is 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