GENERAL PATHOLOGY AND BACTERIOLOGY

FOR DENTAL STUDENTS

BY

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BRISTOL: JOHN WRIGHT & SONS LTD.

1958

PRINTED IN GREAT BRITAIN
BY JOHN WRIGHT & SONS LTD.
AT THE STONEBRIDGE PRESS,
BRISTOL

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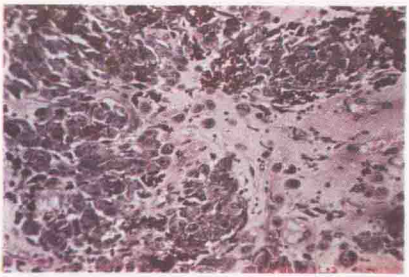
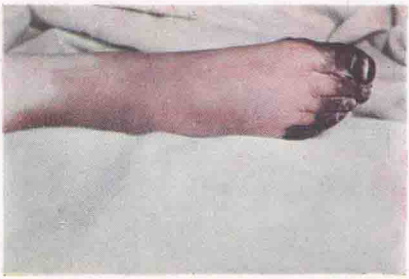
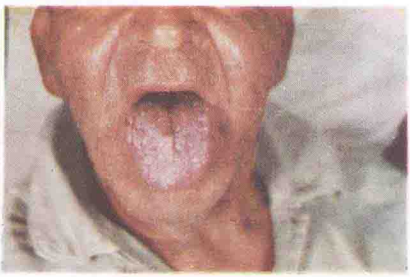


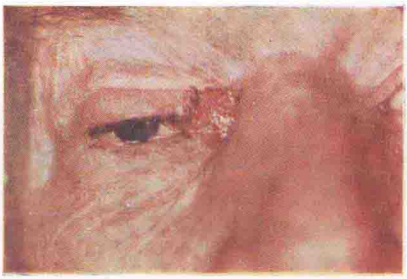
A. Malignant melanoma. (Hæmatoxylin and eosin.) (× 75.)



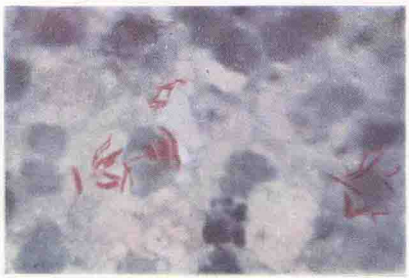
B. Dry gangrene.



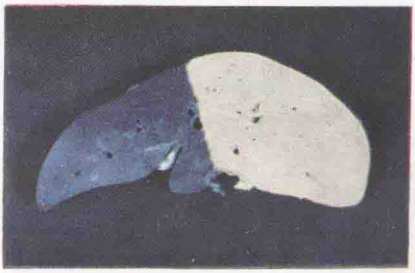
C. Leucoplakia of tongue.



D. Rodent ulcer.



E. Tubercle bacilli in urinary deposit. (× 750.)



F. Pernicious anæmia. Liver showing iron reaction.

PREFACE

THIS book is based on the routine teaching given by the author to dental students over a period of twelve years and has been prepared at the request of the students themselves. The unusual arrangement of the chapters is intentional and is designed to link pathology with bacteriology as much as possible, for it is felt that in many Schools there is an unfortunate tendency to treat them as separate subjects.

It must be stressed that pathology, like clinical subjects, cannot be learned from a text-book alone and study of specimens in a pathology museum is an essential supplement to reading. Dental students in Bristol are fortunate in having a general hospital opposite their Dental School and they are encouraged to attend the daily autopsy demonstrations given by the pathologists. It is at such demonstrations that the student can appreciate the effects of disease on the body as a whole ; the clinicians who are present to discuss the case histories serve as a reminder that diagnosis, prognosis, and sometimes treatment are founded on a sound knowledge of the pathology of disease.

Details of staining methods and other practical procedures are best acquired in the laboratory and therefore are omitted from the text.

I am deeply indebted to Professor T. F. Hewer who kindly read the original manuscript and made valuable criticisms and suggestions. Advice and information on certain topics were freely given by many colleagues, and special thanks are due to Professor A. I. Darling, Dr. Paul Mann, and Dr. C. N. Iland. I am grateful to Professor K. E. Cooper for the use of *Figs.* 26, 62, 66, and 69 ; to Dr. D. B. Peacock for *Fig.* 29 ; and to Messrs. G. W. Griffin and D. N. White for their skill in preparing most of the photomicrographs. I must also record the great help given to me by my wife, Kathleen, in typing the original manuscript.

To my publishers, Messrs. John Wright & Sons Ltd., I owe much, for they have been models of patience, courtesy, and co-operation.

November, 1958 R. L. B.

CORRIGENDA

p. 51, l. 3 *for* Fig. 16 *read* Fig. 18

p. 151, l. 24 *for* Löwenstein-Jenson media *read* Löwenstein-Jensen medium

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GENERAL PATHOLOGY AND BACTERIOLOGY FOR DENTAL STUDENTS

CHAPTER I

INTRODUCTION

PATHOLOGY is the science concerned with the causes, evolution, and effects of disease and is an essential background to clinical diagnosis. Morbid anatomy deals with the naked-eye or macroscopic changes, whilst morbid histology is concerned with the microscopic changes; the abnormal structural changes are termed 'lesions'. Some conditions are amenable to treatment by surgery and examination of the excised tissue or organ gives information about the local lesion, showing it to be an entirely local disease such as a simple tumour or perhaps indicating that it is part of a more widespread disease. Sometimes the surgeon removes a portion of tissue merely for diagnostic purposes and this is known as a biopsy. For example, a patient may have enlarged lymph-glands in the neck without an apparent cause and a biopsy of one of the superficial glands might show the lesion to be a harmless condition such as a trivial inflammation or it could lead to the diagnosis of a serious condition such as cancer in one of the internal organs. A specimen of bone-marrow is an essential aid to the elucidation of many blood diseases and marrow biopsy can be carried out without undue inconvenience to the patient, but in spite of the extension of biopsy methods to the brain, liver, and other organs by inserting wide-bore needles and aspirating tissue, it is not always reasonable to subject ill patients to biopsy procedures. Examination of more readily obtainable material, i.e., blood, urine, and fæces, can often be of great assistance in diagnosis, and chemical pathology is concerned with the biochemical changes in disease; it may be the main approach to the confirmation of such conditions as diabetes and nephritis. Hæmatology is the branch of pathology dealing with blood disorders and involves mainly the examination of blood and marrow biopsy.

Pathology, however, is not concerned with diagnosis alone but embraces also the natural history of disease, and pathogenesis is the sequence of events in the progress of lesions, i.e., their evolution. Much of this information is obtained from post-mortem examinations (autopsies) which also enable us to investigate the effect of disease on the body as a whole. Experimental pathology attempts to verify theories of the causation of various diseases by reproducing them in animals, and when successful it gives valuable information regarding the natural history, because, unlike human beings, the subjects can be killed at selected intervals in the course of the disease. Although the precise cause of diabetes is still not clear, we are indebted to animals for our present knowledge and treatment of the condition, for without the earlier animal experiments the secretion of insulin by the islets of Langerhans could not have been discovered ; experimental physiology therefore makes contributions to pathology.

Aetiology concerns the factors which play a part in the onset of a disease and can be divided into predisposing and precipitating causes. For example, in the aetiology of a fracture the predisposing cause could be a tumour in the bone and the precipitating cause a relatively mild injury insufficient to fracture a normal bone.

Disease may be acquired owing to factors operating after birth or may be congenital as the result of disturbance before or during birth. A congenital lesion is present at birth but is not necessarily inherited. An inherited condition is usually the result of transmission from one or both parents of particular factors in the genes of the chromosomes and is therefore potentially present from the moment of fertilization of the ovum. Thus albinism and hæmophilia are inherited conditions, but whilst the former is obvious at birth as a congenital abnormality, hæmophilia does not usually reveal itself until a year or two after birth. Some infections can be transmitted from the mother through the placenta to the child and the resultant disease is inherited without a genetic basis, e.g., congenital syphilis. In contrast, there is evidence that maternal infection can produce a congenital disease which is not strictly the same infection : German measles occurring in the mother during early pregnancy is liable to cause developmental abnormalities of the heart, cataract, or other congenital lesions in the child (Gregg, 1941 ; Logan, 1951).

Following the onset of a disease there are sequelæ and perhaps complications. A sequel is one of the usual effects of a particular lesion, whereas a complication is superimposed on this natural course and is a not inevitable process predisposed to by the initial lesion. As an example in everyday life, a marriage certificate is a

sequel of the marriage ceremony and a subsequent birth certificate is a complication.

Bacteriology is the study of microscopic organisms, and this relatively new science demonstrates the importance of aetiology; many of the bacterial diseases can now be successfully cured or even prevented because the causal agent was found and studied. The marked prolongation during recent years of our expectation of life must be attributed mainly to the lower incidence of deaths from infections, a triumph which would have been impossible without bacteriology, whilst great advances in surgery, with resultant immediate saving or prolongation of life, followed the institution of asepsis and antiseptics.

The routine bacteriology laboratory is engaged largely in studying the characteristic features of organisms so that they may be identified, but frequently it is only by correlation with clinical findings that their importance can be assessed. Micro-organisms are ubiquitous and are widely distributed in nature, particularly in and on our bodies. Fortunately, only a few of them are capable of producing disease and these are known as pathogens. The majority of bacteria live on dead organic matter derived from plants or animals and are termed 'saprophytes', but only a few are pathogens. The others, the parasites, are adapted to life on living tissues and present variations in their behaviour. Some parasites are always pathogenic when introduced in sufficient numbers into a person or animal (the host) lacking immunity to the particular organism; thus the causal agent of syphilis is a strict pathogen in man. Most of the parasites living on the body surface and in the body cavities are harmless and are termed 'commensals', but some can become pathogenic under certain conditions; e.g., due to local trauma or impaired body resistance, previously harmless commensals in the throat or buccal cavity frequently become pathogenic and cause inflammation. Some parasites are able to exist for varying periods on inanimate objects (fomites), such as dust and clothing, but the remainder, the strict or obligate parasites, being more delicate and usually more specialized, can live and multiply only in the presence of the living tissue of a host.

Some species of organisms enhance the growth of another particular species and this phenomenon is known as 'symbiosis'; occasionally a species is frankly antagonistic to another (antibiosis). In the main, however, different species compete with each other for nutritional requirements and an equilibrium, depending on the environment, is reached.

A disease which is always present in a particular geographical area is 'endemic' in that locality, whereas the occurrence of a few isolated

cases of a disease with intervals of freedom from it is termed a 'sporadic' outbreak, e.g., measles is endemic in Great Britain, but there are only sporadic cases of small-pox. An 'epidemic' is a rapid increase in the incidence of a disease and a 'pandemic' implies a nation-wide or world-wide epidemic.

Carriers play an important role in the epidemic, endemic, and sporadic aspects of a disease. A 'carrier' is a host who, apparently not suffering from a particular infectious disease, harbours the causal pathogen and transmits it to other hosts, with resultant infection in those susceptible. An 'incubatory carrier' is infectious in the interval between acquisition of the organism and its clinical manifestation. A 'convalescent carrier' is one who has overcome the infection but has not completely eradicated the organism; if the latter persists for an indefinite period as a parasite, now less pathogenic to the host because of recently acquired immunity, the convalescent carrier becomes a 'chronic carrier'. Chronic carriers therefore usually have a residual low-grade inflammatory lesion in which the organism persists. The importance of a chronic carrier from an epidemiological point of view depends on the type of organism and its site, e.g., a chronic carrier of typhoid fever only intermittently discharges the organism from an internal source into the fæces or urine. A host with pre-existing specific immunity may come into contact with a pathogen and harbour it for a variable period; because no lesions are produced the host is a healthy or contact carrier. In the intervals between outbreaks, endemic infectious diseases are maintained in a community by healthy carriers. An endemic disease may assume epidemic proportions because of increased virulence of the organism or lack of sufficient immunity in large numbers of the population at risk. A community usually has little or no immunity to diseases which are not endemic: simply because there is little opportunity to acquire any immunity, e.g., some of our simple childhood infectious diseases are disastrous when introduced by carriers into tribes who lead an isolated existence normally free from these infections. Fortunately, prompt public health measures are frequently able to confine imported diseases to sporadic outbreaks, and in this control particular attention has to be paid to tracing and isolating the carriers. During the development of an epidemic the number of carriers increases and there is therefore greater dissemination of the disease.

Environment is an important factor in the aetiology of disease and the incidence of most infectious diseases is related to the living conditions of the susceptible community. Overcrowding and poor ventilation increase the risk of infection from clinical cases and carriers;

communal food or water supplies may become contaminated by certain pathogens, but good public health administration reduces these risks to a minimum. Environment should be considered in diseases other than infections. Many occupations predispose to or even cause disease, and preventive measures can often be practised when the association is discovered, e.g., silicosis and tuberculosis in miners due to inhalation of dust; cancer of the skin in trades involving certain chemicals. Racial tendencies to various diseases are often due not to heredity but to customs or diets peculiar to the race.

The following chapters are concerned with the aetiology and pathogenesis of disease processes. The latter conforms to certain patterns, but one fundamental point must be stressed, namely: Nature is unpredictable and all pathological processes have their extremes in severity, the variations depending on the individual's personal response to the disease, and also on the intensity of the aetiological factors.

CHAPTER II

TISSUE RESPONSES

NECROSIS

NECROSIS is the local death of tissues or cells in the living body. Massive necrosis involving a large volume of an organ or tissue is termed 'gangrene' and is often accompanied by bacterial putrefaction.

Aetiology.—According to the intensity of the causal agent necrosis may take place suddenly or it may be preceded by degenerative changes. There are four main groups of causes :—

1. Deprivation of blood-supply by thrombosis, embolism, arterial spasm, or pressure on blood-vessels will prevent the essential oxygenation of the affected tissues. The brain and other highly specialized tissues with very active metabolism are obviously more sensitive to oxygen lack than are the relatively inert cells of connective tissue.

2. Some bacterial poisons (toxins) are capable of producing necrosis, but this depends on the concentration of the toxins and on tissue sensitivity. In diphtheria, for example, there is necrosis of epithelium in regions infected by the organism because the toxin concentration is high at the site of bacterial activity ; dilution of the diphtheria toxin by the blood-stream protects similar epithelium elsewhere in the body. Some tissues, although not invaded by the organism, may be susceptible to its toxins in the blood, and in diphtheria necrosis is sometimes seen in the suprarenals.

Chemical poisons act also according to their concentration and tissue susceptibility. The concentrating action of the tubules in the kidney predisposes this organ to damage by some chemicals. Ingested poisons may affect the stomach if in sufficient quantity, and following their absorption into the portal vein the liver is the next organ to receive a high concentration. The liver is therefore prone to necrosis by alimentary poisons, and by bearing the full brunt of the absorbed poison tends to protect other organs.

3. Trauma, frost-bite, burns, scalds, X rays, sustained pressure, and other physical agents cause necrosis by direct destruction of living cells.

4. Interference with innervation may give rise to peripheral necrosis, and such trophic ulceration is seen in the perforating ulcer

on the ball of the foot in tabes dorsalis. A similar type of necrosis occurs in the tips of the fingers and toes in the anæsthetic or nervous form of leprosy.

Macroscopic Changes.—The appearance of necrotic tissue is dependent on the type of tissue involved.

Coagulative necrosis is a form encountered in infarcts of the kidney, spleen, and myocardium. The dead region is yellowish and hard because of coagulation of the cell contents and the pericellular fluid.

Colliquative necrosis is seen in the brain where necrotic tissue does not coagulate, but is softened or liquefied by autolytic enzymes—a process of autodigestion.

Microscopical Changes.—The cytoplasm of necrotic cells usually stains deeply with eosin and has a granular appearance. The nuclei show various changes before their ultimate disappearance. The first change is condensation of the chromatin content so that the nucleus is smaller and stains darkly (pyknosis). It then breaks up into multiple fragments (karyorrhexis) which still stain densely with basophil dyes. At a variable period later the chromatin fragments disappear by solution into the surrounding necrotic cytoplasm (karyolysis). These nuclear changes proceed with greater rapidity in some organs compared with others ; in the less specialized connective-tissue cells the change takes place slowly.

Caseation is a form of necrosis encountered in tuberculosis and syphilis. The necrotic material has a cheesy appearance and histologically consists of amorphous granular material stippled with fine droplets of fat due to breakdown of fat-protein complexes in the cells.

Fat Necrosis occurs in two conditions. In acute pancreatitis there is liberation of active pancreatic enzymes into the pancreas, omentum, and mesentery, where fat is then split by the lipase into fatty acids and glycerol. The glycerol is absorbed, but the fatty acids become converted into soaps ; calcium soaps are eventually changed to calcium carbonate and phosphate. The areas of fat necrosis are seen as small, white, opaque plaques in the affected fatty tissue.

Traumatic fat necrosis is sometimes encountered in the breast, where direct injury may produce necrosis of the fat cells which then liberate their fat content. Macrophages, which are large phagocytic cells, engulf tiny droplets of this free fat, but some break down to fatty acids which act as irritants and evoke a chronic inflammatory reaction. The condition is important clinically because it may be mistaken for a cancer.

Sequelæ of Necrosis.—If an area of necrosis is near to the surface of the skin or a mucous membrane, the overlying epithelium usually loses its blood-supply and when it is shed leaves an ulcer. Dead tissue stimulates the surrounding healthy tissue to effect the process of repair. Sometimes the necrotic cells are removed and replaced by regeneration of neighbouring surviving cells. The epithelium of the skin and mucous membranes has great powers of regeneration : this is fortunate because such sites are repeatedly being damaged by trauma. Other tissues such as the nervous system are not so well endowed with regenerative ability. In massive necrosis, such as occurs in infarcts, regeneration does not usually occur and the dead tissue is either replaced by scar tissue or it becomes encapsulated by fibrous tissue and the inspissated contents later calcify.

REPAIR

In the majority of pathological processes there is an attempt by the body to repair damaged tissues by either regeneration or scarring. The process of repair is best illustrated in the healing of a wound, and there are two possible methods.

Healing by First Intention.—The healing of an aseptic, sutured operation wound is an example of repair with restoration almost completely to normal. The adjacent edges of the wound are held in very close apposition by sutures. Blood and lymph escape from the sides of the incision into the intervening small space and the resulting sticky coagulum joins the wound edges. Capillaries bud out from the marginal vessels of the wound and grow into the coagulum accompanied by fibroblasts. The latter are derived from connective-tissue cells which swell and then divide. If there is no infection of the wound there are very few capillaries and the main reaction is fibroblastic. The capillaries and fibroblasts merge with those growing in from the opposite side and arrange themselves at right angles to the surface. Polymorphonuclear leucocytes simultaneously invade the area and their proteolytic enzymes liquefy the coagulum, which is then absorbed by the lymphatics. Meanwhile, the epithelial surface grows from the sides to cover the top of the wound. Eventually the fibroblasts lay down collagen and the mature fibrous tissue contracts ; this compresses the capillaries and they gradually disappear, leaving an avascular scar of fibrous tissue. The closer the edges of the wound are held in apposition, the less obvious is the scar externally. The epithelium covering the scar appears white because of the underlying avascular fibrous tissue.