

GROSS PATHOLOGY

A COLOR ATLAS

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FOREWORD BY PROFESSOR SIR THEO CRAWFORD

with 762 illustrations in full color

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FOREWORD

THE INCREASING AMOUNT of knowledge that the young doctor must acquire during his training has inevitably led to attempts to prune some of the older subjects of the classical medical curriculum. As every gardener knows, pruning is a process which, if properly conducted, leads to tidier, better organised and more fruitful growth: but if the process is crudely or ruthlessly performed the plant may wither and become unproductive. Amongst subjects of the medical curriculum that have suffered in this way Gross—or Anatomical—Pathology comes high on the list.

Those of us who were undergraduates between the wars were sated with descriptive gross pathology. We learned by rote the text book descriptions and culinary metaphors applied to a host of morbid anatomical states, for we knew—or thought we knew—that survival through a testing examination depended on this knowledge. Which of us dared to enter such an examination without being on familiar terms with such delicacies as nutmeg liver, hard-bake spleen, or bread-and-butter pericardium—or a host of other irrelevancies acquired by hours spent in the post-mortem room, at tutorials, or with the text books? It is right that much of this preoccupation with the details of gross anatomical change as a result of disease has been pruned away to find time for a more functional approach to pathological processes; but, as so often happens, there is danger of the pruning being too radical and producing a generation of medical graduates who carry in their mind's eye no mental picture of the altered anatomy of the diseased organs whose abnormality they detect by their increasingly sophisticated diagnostic tests. Such doctors are clearly at a disadvantage in acquiring a full understanding of their patients and the impact of their disease upon them. The dilemma, therefore, is to retain time for an updated version of the classical morbid anatomy and simultaneously to find time for the countless new facets of knowledge in the field of pathology with which the student now has to be acquainted.

Gross pathology is a visual subject, and like all visual subjects it is to be learned only by frequent exposure to the visual patterns involved—and, furthermore, if it is to be remembered, periodic re-exposure is essential. Traditionally these exposures have involved long hours spent in the post-mortem room and in the museum, with resort to reference books for rather indifferent black-and-white illustrations for home study or when less common conditions are being considered.

This Atlas of Gross Pathology is a serious, and—in my view—successful attempt to solve these problems. It brings together in a single volume no less than 762 technically-superb illustrations in colour of all the common and very many of the rarer pathological disturbances of anatomy found in the operating theatre and post-mortem room. These pictures have great advantages over the specimens in most museums: they look 'alive' rather than preserved, and in many instances the important features are emphasised by 'close-up' viewing. Furthermore, the clear and concise text which accompanies the illustrations gives just the amount of commentary that is needed for what is essentially a visual exercise.

Professor R. C. Curran is the natural author for such a book. His origins in Glasgow ensure a proper appreciation of the value of morphological detail in the understanding of disease: and this, tempered by his sojourns in Sheffield, London (at St. Thomas's) and Birmingham, and by his personal research in inflammation and repair, further ensures that the details of gross pathology are appropriately related to the histological features which formed the subject of Professor Curran's earlier Atlas.

Professor Curran has been fortunate and wise to enlist the aid of Dr. E. L. Jones as his co-author for this Atlas. Dr. Jones is a Senior Lecturer in Professor Curran's department in

Birmingham but, still in his early thirties, he is young enough to know by experience the pressures on the contemporary student population and on our young pathologists in training. Between them Professor Curran and Dr. Jones have solved enormously difficult problems. The technical quality of the photography is superb and is matched by the skilful selection and preparation of specimens. The result is a collection of pictures which are superior in educational value to many of the fixed specimens to be found in pathological museums. No-one would suggest that direct experience of pathological material in the laboratory and post-mortem room could ever be replaced by illustrations in the training of our future pathologists. But for the undergraduate it makes sense to short-circuit the learning process in this way: and the trainee pathologists too will find it an invaluable adjunct to the more rapid acquisition of their essential stock-in-trade of visual memory.

T.C.

AUTHORS' PREFACE

FROM THE LATE 18th century, when Morgagni published his great classic, *De Sedibus*, advances in medicine were generally based on the principle of correlating the clinical and pathological features of a disease. This was done by determining the structural changes present in the tissues and then considering how these changes might lead to disordered function and in turn to the symptoms and signs characteristic of that disease. This approach is still valid, and modern analytical techniques have extended it from the macroscopic to the subcellular and molecular levels. We believe that a sound knowledge of the macroscopic changes in the tissues is still the firmest foundation on which to build an understanding of disease and its clinical manifestations. Students are quick to recognise this. However the pressures of a crowded curriculum have tended to reduce the time available to them for studying tissues at first hand, either in the form of surgical specimens or through attendance at post-mortem examinations. It is hoped that this Atlas will help to remedy this deficiency, by presenting a balanced and comprehensive coverage of the structural derangements found in most of the diseases likely to be encountered in clinical practice.

The 'visual' content of tissue pathology is high, and to convey this in the most effective way, unfixed or lightly fixed tissues have been used wherever possible. They are shown in considerable detail, often in close-up in a way normally experienced only by the pathologist handling tissues in the laboratory or post-mortem room. Correlation of the macroscopic and microscopic abnormalities is aided by occasional brief descriptions of the main histological features. As a rule scales have been omitted from the illustrations. Instead we have included a sufficient amount of the affected organ, or shown its architecture in such detail by means of 'close-ups', as to make it easy to estimate the size of the lesion. Where more precision is required, the measurements are given in the text. The colour balance of the pictures has been carefully adjusted so as to be satisfactory for most forms of lighting, but for viewing them artificial (tungsten) light is better than daylight or light from gas discharge (fluorescent) tubes.

The concise text allows the essential features of each lesion to be readily grasped, the intention being that the student should complement this knowledge by reading the fuller descriptions available in the standard textbooks of pathology. On the other hand the Index is comprehensive to make it easy to locate a particular lesion or a macroscopic appearance. The Atlas has been planned primarily for undergraduate students but it is hoped that it will prove of interest and value not only to postgraduate students in pathology but also to those training in other branches of medicine.

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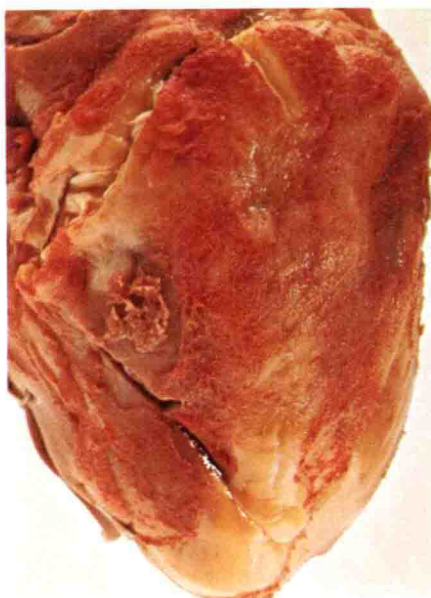
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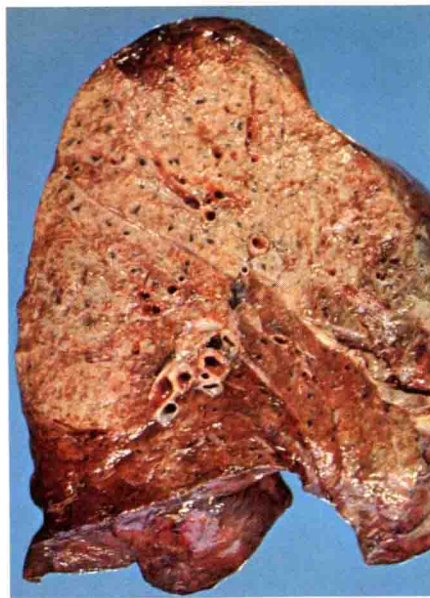
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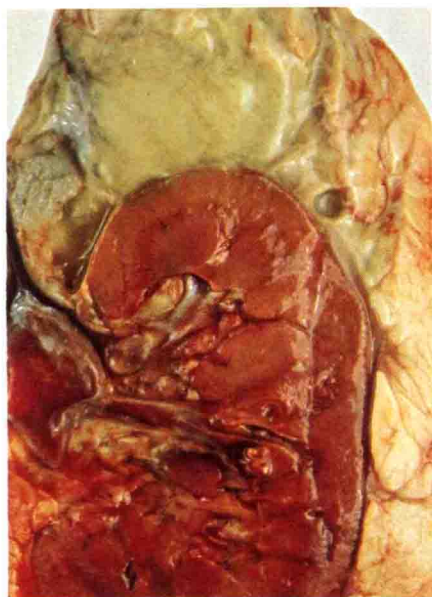
Injury to tissue initiates a series of events which tend to destroy or limit the spread or effect of the injurious agent. These events constitute inflammation. The earliest responses are mainly vascular and the later phases of repair and healing of the injured tissue may be regarded as parts of the same defensive process. The agents that injure tissues and thereby evoke an inflammatory response include bacteria and other types of microorganisms, and non-living agents such as trauma, heat, cold, radiant and electrical energy, and chemicals. The diversity of the aetiological factors is the principal reason for inflammation being one of the commonest and most important conditions in pathology. There are also immunological mechanisms which are defensive in nature but which may in some circumstances prove harmful to the host. This chapter illustrates some aspects of the inflammatory response; and examples of lesions produced by a wide variety of agents or by mechanisms of an infective, physical and immunological nature are given.



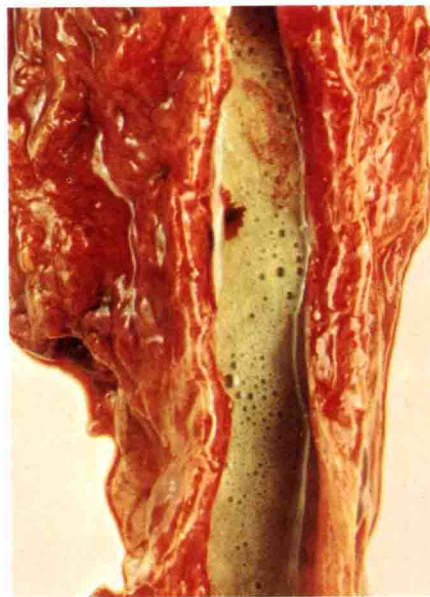
1.1 Acute fibrinous pericarditis



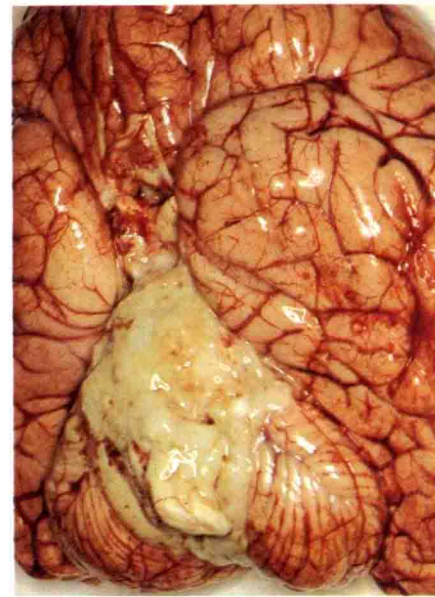
1.2 Lobar pneumonia

1.3 Suppuration and abscess formation:
branchial cyst

1.4 Perinephric abscess



1.5 Acute tracheobronchitis



1.6 Purulent meningitis

1.1 Acute fibrinous pericarditis. Fibrinous inflammation frequently involves serous membranes and the meninges. This shows the epicardial surface of the heart covered with a fibrinous exudate which is slightly blood-stained in places. A protein-rich fluid was also present in the pericardial sac. Fibrinous pericarditis may be caused by a pneumococcal infection but the most extreme forms are found in acute rheumatic fever. Fibrinous inflammation is also often seen on the pleura overlying a pneumonic process. **1.2 Lobar pneumonia.** In lobar pneumonia, a fibrin-rich inflammatory exudate fills the pulmonary alveoli in such a way as to cause one or more lobes to become solid (consolidation) and liver-like (hepatisation). This is the left lung and there is extensive consolidation of the whole of the upper lobe and apical segments of the lower lobe. The consolidated lung is pale grey, a stage of consolidation referred to as grey hepatisation. It is preceded by red hepatisation and generally occurs four to eight days after the onset of the acute illness. The cut surface of the affected lung is dry, granular and airless, and the firm consistence is shown in the way that the cut surface retains sharp straight edges. Lobar pneumonia is most often caused by a pneumococcal infection and the classical appearances of red and grey hepatisation are seen much less frequently than previously. **1.3 Suppuration and abscess formation; branchial cyst.** Two of the signs of acute inflammation are evident, viz. redness and swelling. The redness is caused by vasodilatation and the swelling mainly through the accumulation of exudate. The underlying lesion is a branchial cyst on the side of the neck

of a young girl. The cyst has become infected and acutely inflamed and pus has formed within it to produce an acute abscess. **1.4 Perinephric abscess.** Infection in the tissues around the upper pole of the kidney has caused acute inflammation which has gone on to the formation of an abundant thick green exudate of pus (purulent or suppurative inflammation). Pus contains large numbers of neutrophil polymorph leucocytes as well as necrotic cells and tissue. Initially thick and creamy, it often becomes thinner in consistence following the release of proteolytic enzymes from dying and dead polymorphs. Perinephric abscess usually complicates acute pyelonephritis, following rupture of renal abscesses into the perinephric tissues. **1.5 Acute tracheobronchitis.** Catarrhal inflammation affects mucous membranes and is characterised by copious secretion of mucus, by formation of an exudate which is at first serous and later mucopurulent, and sometimes by desquamation of the mucosal epithelium. The changes are typically confined to the superficial tissues. In this case, the mucosa of the trachea and bronchi were affected and the trachea is filled with frothy greenish mucopurulent secretion. For many years the patient had chronic bronchitis, a condition which is liable to be complicated by episodes of acute inflammation. **1.6 Purulent meningitis.** The undersurface of the brain is shown. A thick green purulent exudate fills the subarachnoid space over the brain-stem and cerebellum. The patient was treated for leukaemia and subsequently developed acute meningitis. The causative organism was *Staphylococcus aureus*.