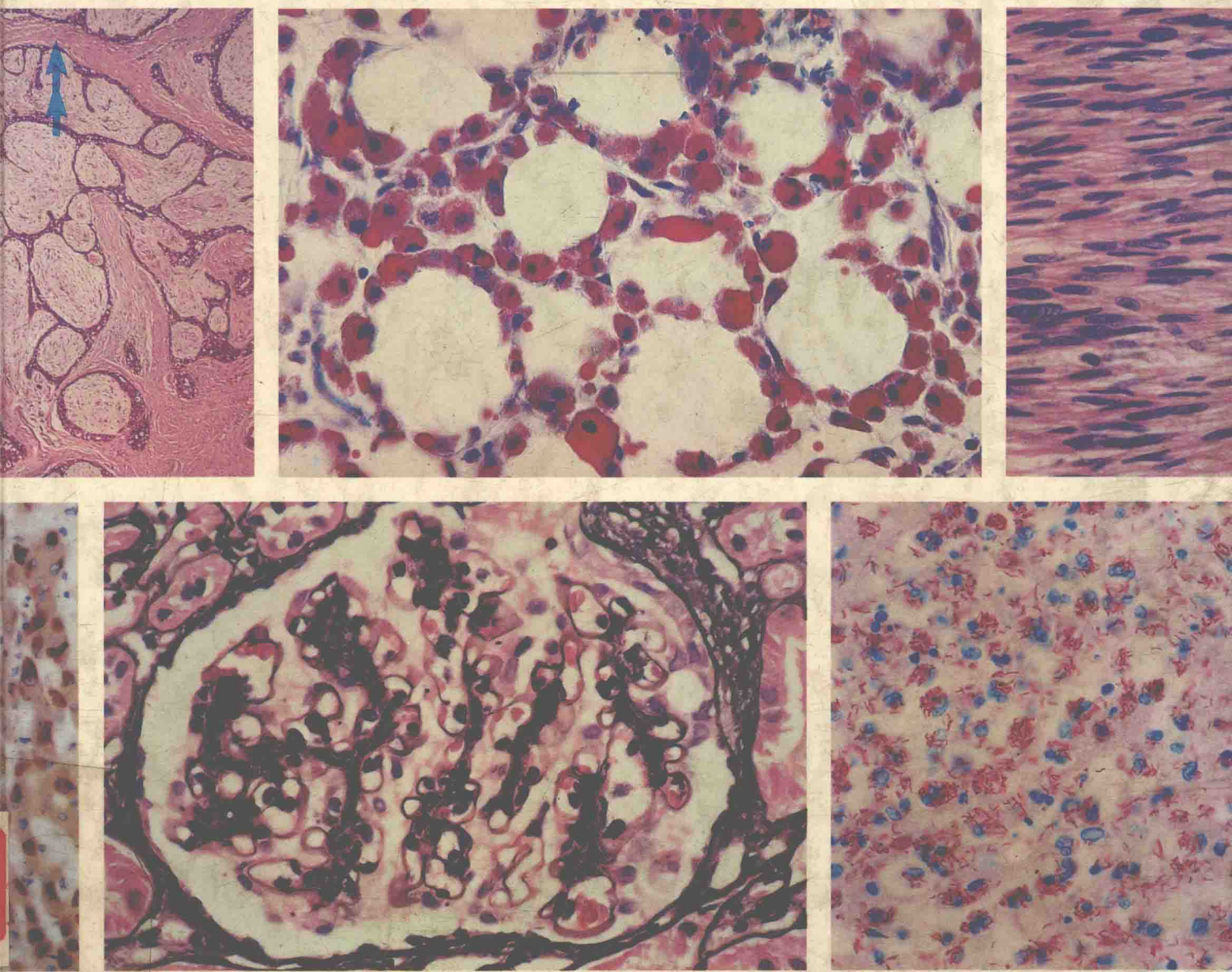
R·C·CURRAN

COLOUR ATLAS OF HISTOPATHOLOGY

THIRD EDITION ~ 804 PHOTOMICROGRAPHS



COLOUR ATLAS OF HISTOPATHOLOGY

BY R·C·CURRAN

MD·FRCP(Lond.)·FRCPath·FRS(Edin.)·FFPathRCPI

Leith Professor of Pathology, University of Birmingham,
Honorary Consultant to the Central Birmingham Health District
and the West Midlands Regional Health Authority

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WITH 804 PHOTOMICROGRAPHS

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Preface

THROUGHOUT its long history, pathology has been concerned with the study of the derangements of tissue structure and function that occur in disease, and the correlation of these changes with clinical signs and symptoms. This clinico-pathological approach, which made pathology the foundation of clinical practice, remains as valid as ever. In recent years the rate of advance of the subject has accelerated greatly with the introduction of a steadily widening range of techniques of great sensitivity, many applicable to paraffin sections, which allow the histopathologist to identify, with a high degree of specificity, cells and their products. At the same time new clinical methods (endoscopy, needle biopsy, etc.) have provided the pathologist with samples of fresh tissues and cells, sometimes repeatedly, from virtually every part of the body. The contribution that the pathologist can make to clinical practice and the understanding of disease has been enormously enhanced by these twin developments. There can be no doubt that for the foreseeable future pathology will retain its place as a basic part of the undergraduate curriculum.

Histopathology has the unique advantage of making visible the body's many complex systems and their interactions and malfunctions in disease; and the first edition of this Atlas was produced with the intention of providing the student, in a vivid and readily assimilable form, a means of acquiring a clear understanding of the basis of the many diseases that he or she will encounter in clinical practice, whether in hospital or in general practice. The Atlas was first published in 1966 and it was well received throughout the world. It was translated into six languages and it has been reprinted each year. On each occasion the question of revision was considered and in 1972 about one-twentieth of the contents were changed. However histopathology is (or was until recently) a slowly-evolving science and it was decided to wait until a thorough revision could be justified. The time for this has arrived and this third edition is virtually a new book in both format and content.

An important feature of previous editions was the large size of the page, which enabled the student to compare 18 illustrations at one time. The format of the new edition, however, allows for larger illustrations which provide increased information; and at the same time the student has the greater convenience in using, carrying and storing a smaller-sized volume. The number of pictures has been increased from 765 to 804 and two-thirds (545) are new. The area of each picture has now almost been doubled and the advantages of this will, it is hoped, be immediately obvious.

The great majority of the illustrations are based on paraffin sections stained with

hematoxylin and eosin, the method in routine use. Other techniques, in more or less common use (some using cryostat sections), have been included, where they make a specific contribution; and the potential of the newer immunohistochemical methods will be apparent from the examples included.

The general arrangement of the contents has been retained, with a chapter on each of the main systems or organs of the body. There is also an introductory chapter of a general nature which aims to demonstrate the more important reactions of the tissues in disease and at the same time teach the student the basic language of histopathology, thereby enabling him or her to read and assess the significance of changes in the tissue as revealed by microscopy. This proved to be a popular feature of previous editions and it has been extended. The text has been re-written, and along with a description of the contents of each picture a limited amount of clinical information about the lesion and its pathogenesis is given. Most of the conditions are common or fairly common diseases, but occasional examples of rare lesions have been included when they illustrate a pathological process with particular clarity. It must be emphasised however that the book remains an Atlas, the primary purpose of which is to convey information in a visual form. It is meant to complement existing textbooks, and prior study of the Atlas will, it is hoped, make the better textbooks both more intelligible and more pleasurable to read.

A comprehensive Index has been provided, and a limited number of cross-references have been inserted in the text, mainly in Chapter 1, to augment it and to integrate its contents with the rest of the book.

The book is intended primarily for undergraduate students but experience with its predecessors suggests that it is likely to prove useful to postgraduate students training in pathology or another clinical discipline.

Most of the illustrations in the Atlas are based on cases dealt with in the course of the routine hospital service provided for the Hospitals of the Birmingham Central Health District, and I wish to pay tribute to Mr. K.J. Reid, Senior Chief Medical Laboratory Scientific Officer and his colleagues for the consistently high quality of the preparations which they have produced. Mr. Sidney Whitfield, Chief MLSO, was directly responsible for much of the work and he also supplied the section of schistosomiasis of liver (**5.18**) from his personal collection. Two other individuals merit special mention: first Mrs Mary Guibarra, who prepared many of the sections of tumours; and Mr. John Gregory who performed the immunohistochemical techniques.

Some of the illustrations in Chapter 5 are from sections kindly loaned by Professor R.S. Patrick (Glasgow), and some in Chapter 12 are from slides provided by the late Dr. C.W. Taylor, pathologist to Birmingham Women's Hospital for many years. The section of the Stein-Leventhal ovary was provided by Professor J.R. Tighe (St. Thomas's Hospital, London).

A number of illustrations are based on cases referred to the Department of

Pathology and I am grateful to the following pathologists for access to these cases: Dr. T.G. Ashworth, Walsgrave Hospital, Coventry; Dr. B.W. Codling, Gloucestershire Royal Hospital, Gloucester; Dr. N.D. Gower, Hallam Hospital, Sandwell, Birmingham; Dr. P.S. Hasleton, Wythenshawe Hospital, Manchester; Dr. F. Kurrein, Royal Infirmary, Worcester; Dr. A.M. Light, Good Hope District General Hospital, Sutton Coldfield; Dr. J. Martin, Tawam Hospital, Abu Dhabi, U.A.E.; Dr. J. Rokos, Staffordshire General Infirmary, Stafford; Dr. T.P. Rollason, Maelor General Hospital, Wrexham; Dr. D.I. Rushton, Maternity Hospital, Birmingham; Dr. W. Shortland-Webb, Dudley Road Hospital, Birmingham; and Dr. Carol M. Starkie, Selly Oak Hospital, Birmingham.

The pathologist to whom the cases were referred was usually my colleague, Professor E.L. Jones. Professor Jones also gave very generously of his time and advice in many other ways during the preparation of the Atlas, and without his help it is doubtful whether this new edition would have appeared.

The manuscript was typed by Mrs. Valerie Adkins and Miss G.L. Parkinson, and I thank them for their hard work and patience.

Finally, I must pay a special tribute to Harvey and Elly Miller. As on previous occasions their advice and professional skills were invaluable at all stages of preparation of the volume and I am pleased to have this opportunity to acknowledge my indebtedness to them.

Methods

Brooke's stain: stains growth hormone-containing (acidophil) cells in the pituitary orange and prolactin-containing cells red.

Dialyzed iron method: stains acid mucins, and particularly those of the connective tissues, blue.

Gough–Wentworth technique: thin (300 μm) unstained slices of whole lung (inflated and fixed).

Grimelius's silver method: stains argyrophil materials (such as 5-hydroxytryptamine) black.

Grocott's methenamine silver method: stains fungi black.

Hematoxylin and eosin (HE): combination of basophil (bluish-purple) nuclear stain and acidophil (pink) cytoplasmic stains in routine use.

Indirect antibody immunoperoxidase method: for detecting antigens in cryostat or paraffin sections by means of polyclonal or monoclonal antibodies. Examples shown are based on the peroxidase-antiperoxidase (PAP) sequence and a brown reaction product indicates the site of the antigen in the tissues.

Loyez (iron hematoxylin) method: stains myelin blue-black.

Periodic acid-Schiff (PAS) sequence: stains carbohydrates, and particularly epithelial mucin and glycogen, purplish-red (magenta).

Periodic acid-silver (PA silver) method: stains basement membrane (particularly in kidney) and fungi, black.

Perls' method (Prussian Blue reaction): stains ferric iron, and particularly haemosiderin, blue.

Thin resin-embedded (½–1 μm) sections: facilitates study of the finer details of cell and tissue structure.

Gordon and Sweet's method: silver deposition method staining fine connective tissue fibres (including basement membranes and newly-formed collagen), black.

Solochrome cyanin: stains myelin blue.

Sudan IV: stains lipid orange-red.

Toluidine blue: reveals periodic striations in muscle cells (e.g. to confirm that a tumour is a rhabdomyosarcoma).

Van Gieson: stains collagenous fibrous tissue purplish-red. Sometimes combined with Weigert's method for elastic fibres (Weigert–van Gieson).

Von Kossa: stains bone salts black (in sections of undecalcified tissue).

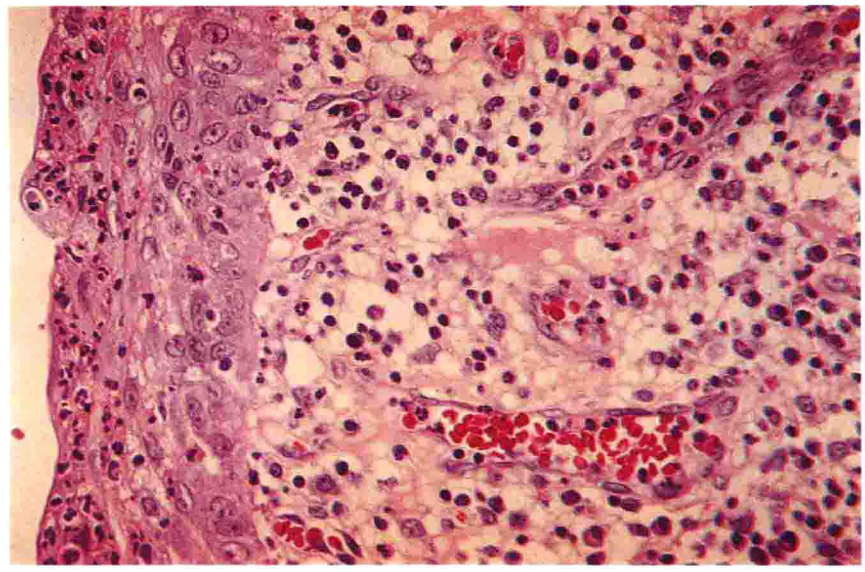
Weigert's method (Miller's modification); stains elastic fibres blue-black.

Ziehl-Neelsen (ZN) technique: stains mycobacteria, (e.g. *M. tuberculosis)* purplish-red.

Atlas of Histopathology

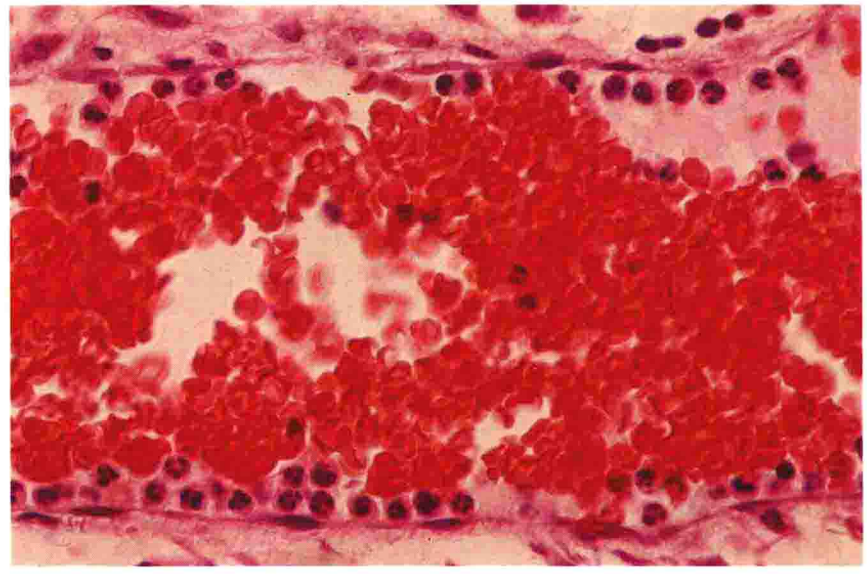
1.1 Acute inflammation

This is an acutely inflamed mucous membrane. The patient had acute inflammation of the colon (ulcerative colitis) for some months, with recent involvement of the anal canal. The anal canal (left) is lined with stratified squamous epithelium. There are many polymorph leukocytes within the epithelium, most of them close to the surface. The submucosa (centre and right) is hyperemic, with marked dilatation of the small blood vessels. Many polymorphs are visible within these vessels, adjacent to the endothelium. The connective tissues between the vessels are pale-staining, from the presence of inflammatory exudate (e.g. centre). In addition to many polymorphs, lymphocytes and plasma cells are present, showing that the acute inflammatory reaction is superimposed on a subacute or chronic lesion. HE ×230



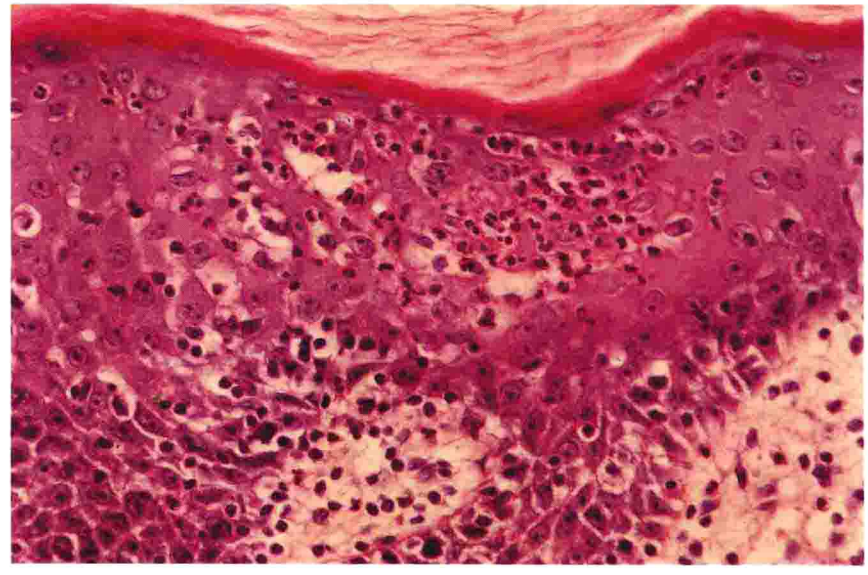
1.2 Acute appendicitis

In acute inflammation the small blood vessels dilate and the blood flow to the inflamed area is greatly increased. The blood flow eventually slows and the polymorphs move towards the periphery of the stream, where they come into contact with the endothelium to which they tend to adhere. The small vessel shown here, a capillary or postcapillary venule in the mesentery of an acutely inflamed appendix, has dilated enormously and an almost continuous layer of polymorphs is adherent to the endothelial surface – 'pavementing' of the endothelium. These polymorphs migrate through the wall of the blood vessel between the endothelial cells, and then through the surrounding tissues towards the cause of the inflammation (chemotaxis). This vessel is so dilated that the blood flow had probably stopped i.e. stasis had developed. HE ×335

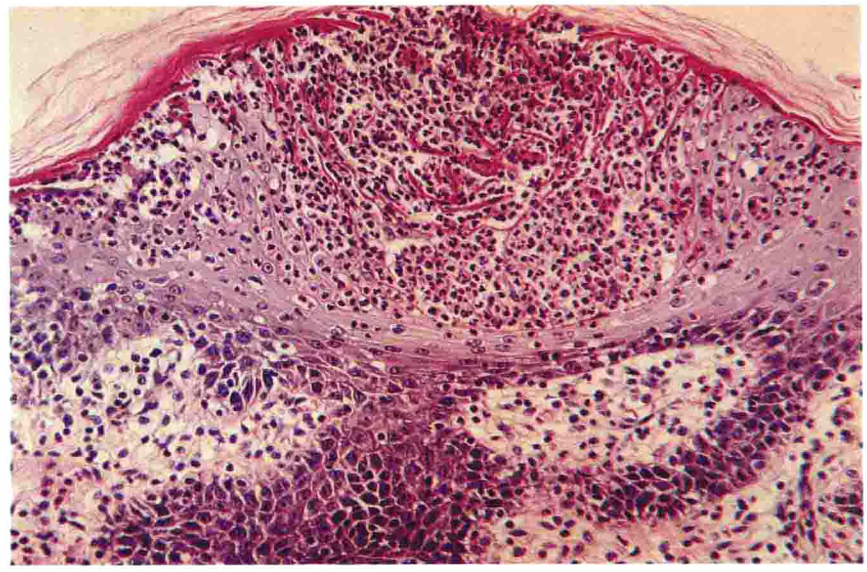


1.3 Pustule: skin

When the agent causing an acute inflammatory reaction is persistent (e.g. pyogenic staphylococci) emigration of polymorphs to the site of the inflammatory stimulus (often bacterial) may continue until they form a mass in the tissues. Some surrounding tissue is invariably killed (necrosis) by the inflammatory agent and enzymes released by polymorphs. An abscess thus forms containing polymorphs, necrotic tissue elements and serous fluid. When the collection forms in the skin it constitutes a pustule (pus-filled vesicle). This is an early stage in the formation of a pustule. Large numbers of polymorph leukocytes have migrated from the small blood vessels in the dermis (bottom) into the stratified squamous epithelium of the skin, and they are starting to form a collection within the epithelium (above centre). HE ×335

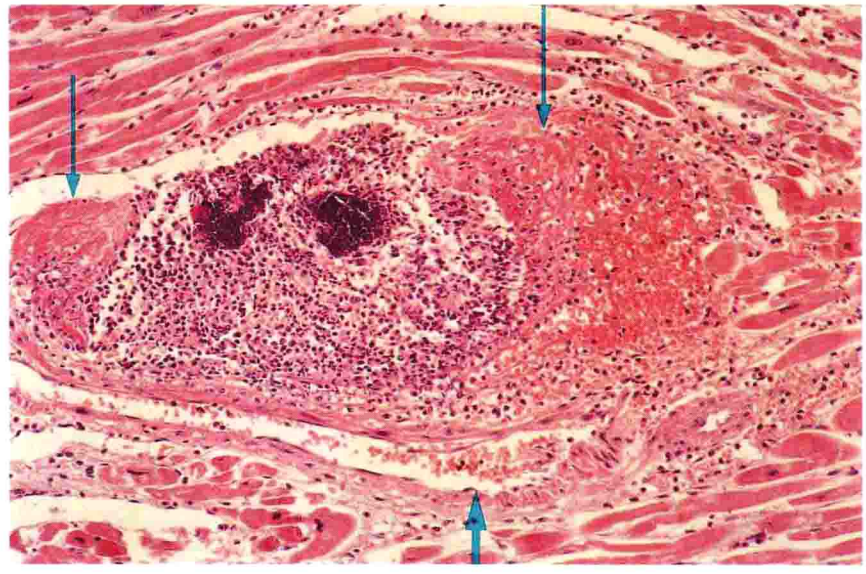


1.4 Pustule: skin



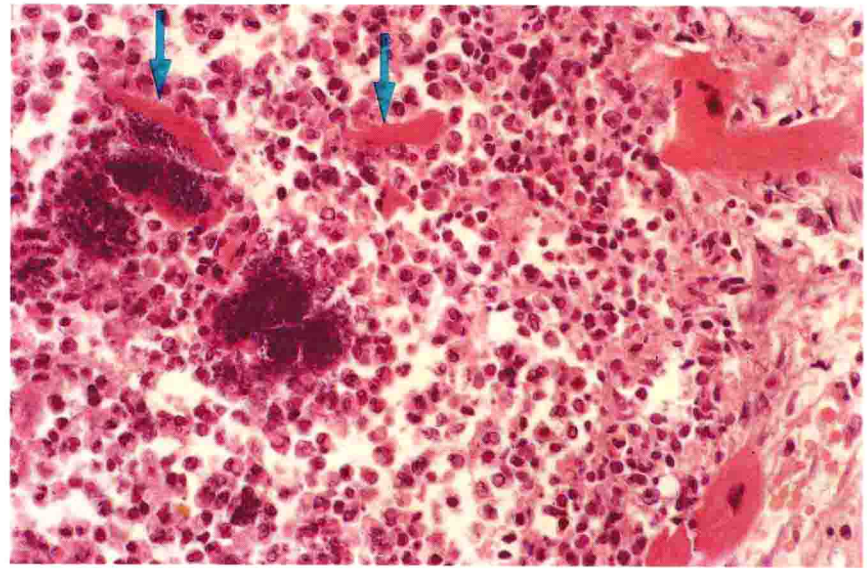
This is a fully-formed pustule. It is an ovoid abscess (centre top) within the upper epidermis and it is causing the surface to bulge upwards. There is a fairly thick layer of keratin on the surface of the skin over the pustule but it is starting to break down over the centre of the pustule. Polymorphs are the main constituent of the pustule but there are also many eosinophilic keratinous 'squames' mingling with the polymorphs. At higher magnification necrotic epithelial cells were also found to be present. The connective tissues of the dermis (below) are also inflamed and infiltrated with inflammatory cells. HE ×150

1.5 Pyemic abscess: myocardium



In pyemia, pyogenic organisms are present in significant numbers in the bloodstream, and, in addition to the primary focus, there are pus-filled abscesses in various tissues. An abscess is always accompanied by destruction of tissue. This abscess is in the wall of the left ventricle. It is an ovoid mass (centre) destroying and displacing the fibres of the myocardium. The muscle fibres nearest the abscess are necrotic with no nuclei, having been killed by the toxins from the two deeply basophilic colonies of *Staphylococcus pyogenes* in the centre of the abscess which contains also degenerate polymorphs and macrophages, fibrin (thin arrows) and red cells. There is also an infiltrate of acute inflammatory cells and red cells in the interstitial tissues of the myocardium (top). The small blood vessel (thick arrow) is dilated. HE ×150

1.6 Pyemic abscess: myocardium



This is another of the many small abscesses that were present in the wall of the ventricle. The pus consists of two deeply-stained colonies of *Staphylococcus pyogenes* (left), polymorph leukocytes (most of which are necrotic and disintegrating) and macrophages. Fragments of necrotic heart muscle (arrows) killed by the toxins from the bacteria and the polymorph enzymes are also present. The serous fluid component of the pus is unstained. The small blood vessels (right) adjacent to the abscess are dilated. Although the myocardial fibres at the edge of the abscess retain their nuclei, they are probably necrotic. Pyemia is usually a very serious illness, the patient surviving for only a few days; and there is no evidence of encapsulation of the lesion by granulation tissue or fibrous tissue. HE ×235