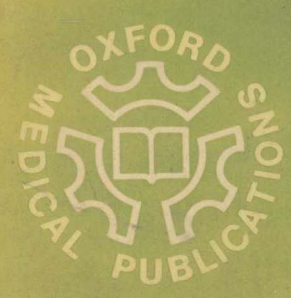

Tropical Gastroenterology

G. C. Cook

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OXFORD
OXFORD UNIVERSITY PRESS
NEW YORK DELHI
1980

Oxford University Press, Walton Street, Oxford OX2 6DP
OXFORD LONDON GLASGOW
NEW YORK TORONTO MELBOURNE WELLINGTON
KUALA LUMPUR SINGAPORE HONG KONG TOKYO
DELHI BOMBAY CALCUTTA MADRAS KARACHI
NAIROBI DAR ES SALAAM CAPE TOWN

British Library Cataloguing in Publication Data
Cook, G C
Tropical gastroenterology. – (Oxford medical publications).
1. Digestive organs – Diseases
2. Tropical medicine
I. Title II. Series
616.3'00913 RC802 79-41545

ISBN 0-19-261228-X

Phototypeset in V.I.P. Times by
Western Printing Services Ltd, Bristol

Printed in Great Britain
at the University Press, Oxford
by Eric Buckley
Printer to the University

Preface

I have written this book because I have for several years felt that a text devoted to tropical gastroenterology was long overdue. I have endeavoured to write first for the clinician and clinical student working in the tropics, who is engaged with gastroenterological problems, which form a substantial part of a subject for long known as 'tropical medicine'. Secondly, I have written for the physician working in a temperate climate, who with the vast increase in rapid population movement is seeing more and more patients with gastroenterological diseases, some of which he has not even heard of, which have been acquired in tropical countries.

I have been guided by much personal knowledge and experience gained from some twelve years in tropical countries. Most of that time has been spent in academic posts in third-world universities and medical schools: two years (1960–2) at Lagos, Nigeria; two years (1965–7) at Makerere University, Uganda; five years (1969–74) at The University of Zambia; one year (1964–5) at Riyadh University, Saudi Arabia; and two years (1978–80) at The University of Papua New Guinea.

The longer one spends in tropical countries the more one appreciates that 'tropical medicine' is a non-entity. Medicine is basically the same the world over, but with some geographical and ethnological variations. Disease patterns depend not so much on ambient temperature as on public health standards and socioeconomic conditions. Much of the medicine in the third world of 1980 is precisely the same as that which existed in the United Kingdom and other 'developed' countries in the eighteenth and nineteenth centuries. Ideally therefore, demands for a book such as this should have a limited duration, as health standards in the third world improve.

For much of internal medicine in the tropics therefore, western texts are sufficient. However, there are a number of diseases which assume a greater importance in third world countries, and which are not dealt with to an adequate depth in such works; many of them carry a vast morbidity and mortality. I have attempted to cover them in greater detail. I have not however tried to cover again all that is contained in well-known texts on gastroenterology and liver disease, such as those written by Sir Francis Avery Jones and his colleagues, and Professor Dame Sheila Sherlock, respectively.

During the last few years some important advances have been made in the field of tropical gastroenterology. Work on absorption has led to the greatly improved management of cholera and other acute diarrhoeal diseases. Demonstration of bacterial colonization of the small intestine in postinfective tropical malabsorption ('tropical sprue'), and recognition of the importance of clostridial toxins in jejunitis necroticans ('pig bel' disease), necrotizing colitis, and antibiotic-associated colitis are other examples. In the field of hepatology, work on HB_sAg and alpha-feto-protein has advanced substantially our understanding of cirrhosis and hepatoma in the tropics.

Comparison of incidence rates of many diseases in various parts of the world must surely hold the key to their aetiology; large intestinal cancer, non-specific ulcerative colitis, and Crohn's disease, are three examples.

There will doubtless be omissions from this text because my personal knowledge of the third world contains deficiencies.

If my readers have suggestions for inclusion of other diseases should a further edition become desirable or possible or if they have any other criticism of a constructive or destructive nature, I shall be most grateful if they will write to me.

I am extremely grateful to Mrs Raka Natera for typing the whole of the manuscript, including the drafts from my long-hand; she has been exceedingly tolerant and generous with her time. I am also indebted to Doctor J. K. A. Clezy, Professor L. W. Deubert, and Doctor D. S. Pryor for reading and criticizing some of the chapters. I am grateful also to Doctors G. H. Aiken and J. C. Muirden for providing some of the pathology illustrations and radiographs, respectively. Finally I should like to thank the staff of Oxford Medical Publications for their help and kindness during the preparation of this work.

Port Moresby,
Papua New Guinea

G. C. Cook

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Section 1 : Upper gastrointestinal tract

1

The mouth, jaws, and salivary glands

BECAUSE it is one of the few accessible parts of the gastrointestinal tract, and because most gastrointestinal investigations are not easy to perform in tropical countries, a full examination of the mouth is often extremely rewarding (Tyldesley, 1969). A systematic inspection with a spatula and a good light is essential.

Oral diseases are extremely important in a tropical context; when the mouth is diseased, eating becomes difficult and a poor nutritional status may be worsened.

1.1. The lips, tongue, and buccal mucosa

1.1.1. Pigmentation of fungiform papillae of the tongue

This condition is a result of melanin deposition in the papillae of the tongue. It increases with age in many dark-skinned people. About 26 per cent of children in east Africa are affected, and it seems to be completely unassociated with malnutrition (Beet, 1948); the condition is present in about 50 per cent of adult Africans (Raper, 1948).

1.1.2. Viral infections

Virus infections involving the mouth and fauces are common in tropical countries. The sore throat associated with infectious mononucleosis is rarely encountered. Oral manifestations of smallpox are no longer seen.

Herpes simplex. Herpes simplex infection involving the lips (herpes labialis), tongue, buccal mucosa, and palate is common in children in tropical countries, especially those with kwashiorkor and measles, in whom a severe stomatitis may also occur. Herpes simplex is an important aetiological agent in cancrum oris (see below). It is often associated with pneumococcal lobar pneumonia and acute malaria. Vesicles of 3–5 mm diameter contain clear fluid at first and then become pustular. Permanent scarring is unusual. Multinucleated giant cells can be identified in smears stained with Geimsa. A specific herpes antiserum and an indirect immunofluorescent technique on cell scrapings can be used to make a rapid diagnosis (Gardner, McQuillin, Black, and Richardson, 1968). Rising antibody titres, and neutralization and compliment fixation tests are valuable in diagnosis.

Enteroviruses. Various members of this group—poliomyelitis, Coxsackie, Echo, and Reo viruses—may produce vesicular stomatitis and herpangina.

Behçet's syndrome. Oral ulceration is present in nearly 100 per cent of patients with this syndrome which is common in the Middle east, Cyprus, and Japan (James, 1979).

1.1.3. Bacterial infections

Acute infection. Such infections are common. Acute tonsillitis with recurrent sore throats is a frequent problem and may be associated with acute rheumatic fever; however it is the chronic manifestations of that disease which are encountered more often than the acute condition. A common practice in some parts of Africa is to remove the uvula (uvulectomy); sepsis, haemorrhage, and gross scarring may result.

Tuberculosis. This disease is not common in the oral cavity. Ulceration of the tongue and buccal mucosa may be secondary to pulmonary tuberculosis. It may consist of a solitary lesion or be part of the widespread disease; mode of infection is not known, but is probably haematogenous. The lesions may be proliferative, granulomatous, or ulcerated (Wilkinson, 1972; Laws, 1976; Rao, Satyanarayana, Sundareshwar, and Reddy, 1977). Presentation may be with a single patch, which has an appearance not unlike lichen planus (Tyldesley, 1978). Tuberculoma of the tongue is an unusual event.

Syphilis. This is a rare infection of the oral cavity. Intraoral chancres are rare. Condylomata may be present on the lips. Oral ulcers can occur in the secondary disease.

Yaws. The primary sore of this infection, caused by *Treponema pertenue*, may appear on the lips. The disease has now been eliminated from most tropical countries.

Leprosy. Oral lesions are unusual, but lepromas, small tumour-like masses, may appear on the tongue, lips, or hard palate.

Sulphonamides, which are widely used in the treatment of shigellosis (bacillary dysentery) (Chapter 24) occasionally give rise to the Stevens–Johnson syndrome in which there is extensive ulceration of the buccal and genital mucosa, and in addition erythema multiforme; the syndrome also occurs after virus and bacterial infections, and other therapeutic agents.

1.1.4. Mycotic infections

Moniliasis (Candidiasis). This is caused by *Candida albicans* and is extremely common in children. It causes white plaques on the oral mucosa often known as 'thrush'. Although usually a localized disease it may extend to the lungs with a fatal outcome. It seems probable that some of the oral changes described in tropical sprue in the nineteenth and early twentieth centuries were due to this organism (see below). It occurs especially in malnourished and debilitated patients and also after antibiotics, corticosteroids, and immunosuppressive agents. Monilial infections respond to nystatin.

Histoplasmosis. This is principally a disease of the reticuloendothelial system, and is caused by *Histoplasma capsulatum*. It is present throughout the tropics and subtropics. The source of infection is usually the faeces of chickens, bats, dogs, and cats. Children are more often affected than adults. Primary lesions

may occur on the lips and mucous membranes of the mouth; however the lungs are usually the most severely affected organs. The fungus can be demonstrated in smears stained with Giemsa. A compliment-fixation test or mouse-culture technique may be used to confirm the diagnosis.

South American blastomycosis. This disease, caused by *Paracoccidioides brasiliensis*, produces ulcerative granulomatous lesions of the buccal mucosa. It occurs frequently in Brazil and is seen in most South American countries (Peña, 1967). It is probably spread by twigs used for cleaning the teeth which are contaminated with the fungus. The granulomatous ulcers spread slowly but extensively, and regional lymph glands become involved. The yeast cells are seen in haematoxylin and eosin preparations of the pus and crusts from superficial lesions.

Coccidioidomycosis. *Coccidioides immitis* is the cause of this disease which is limited to southern America. Extensive ulceration of the lips and face, with secondary infection, is an unusual complication. Amphotericin B is the most effective proven form of treatment for the systemic mycoses (Wilcocks and Manson-Bahr, 1972).* Various other systemic fungal (mycotic) infections can cause lesions of the upper lip in parts of the tropics (Edington and Gilles, 1976). Subcutaneous phycomycosis caused by a species of *Basidiobolus*, and rhino-entomophthoromycosis caused by *Delacroixia coronata* (Chapter 2) occasionally involve the lips.

1.1.5. Malnutrition and deficiency disease

Oral changes in severe protein-calorie deficiency are multiple, but are usually dominated by one or more infections (see above) with added evidence of vitamin and iron deficiencies.

Hypochromic anaemia. In iron-deficiency (hypochromic) anaemia, which is extremely common in all parts of the third world and is usually caused by hookworm infection, smoothness and pallor of the tongue are common. However, the incidence rates of glossitis, stomatitis, and dysphagia are surprisingly low when the frequency of anaemia is taken into account.

Riboflavin and other B vitamin deficiencies. A deficiency of these vitamins produces cheilosis, a condition in which the lips are cracked, *angular stomatitis*, grey-white fissures at both angles of the mouth, and *glossitis*, a sore tongue which is often abnormally deep red in colour. These changes are very common in pellagra.

Scurvy. This is an uncommon disease in the tropics; even in areas with a long, dry season during which time there are few fresh vegetables and fruits, it is rarely seen. Produced by a lack of dietary ascorbic acid, it produces severe gingivitis and loosening of the teeth.

1.1.6. Tropical malabsorption (TM) ('tropical sprue')

Oral changes in tropical malabsorption (TM) seem to be much less usual today

* Recent evidence suggests that miconazole gives favourable results in various tropical mycotic infections including coccidioidomycosis and South American blastomycosis (*Lancet*, 1979).

than they were in the past (Cook, 1978). That is probably because cases seen today are not as long-standing or severe as many of those described in the classical reports. Severe cases of TM seen at present occur mainly in expatriates following holidays or tours in Asia. In most eighteenth and nineteenth century reports, aphthous ulceration involving the tongue and buccal mucous-membrane was considered to be very common in the 'white flux' and 'hill diarrhoea' (TM) (Cook, 1978) (Chapter 20). In describing the oral changes associated with TM, which dominated most early accounts of the disease, Hillary (1766) described 'little small pustules, or pimples, filled with a clear acrid lymph at the end and sides of the tongue, which gradually increase in number and slowly spread to other parts of the mouth; soon the thin skin slips off and the tongue looks red and a little inflamed, and is almost raw like a piece of raw flesh and is tender and sore'. Writing of the 'white flux', Martin (1856) described 'anaemic ulcerations (chronic aphthae) of the mucous digestive surface'. The term 'tropical sprue' was first used by Manson (1880) to describe TM; he adopted the term sprue from the Dutch word *spruw* which was then in use to describe oral aphthous ulceration in children. Good descriptions of the oral changes seen in sprue or psilosis, were given by Thin (1890; 1897). One hundred and one of 150 cases of 'tropical sprue' reported in London by Low (1928) had tongue lesions when first seen and 44 had other oral signs as well. In some of the 200 cases reported by Manson-Bahr and Willoughby (1930) buccal changes were considered to have preceded the diarrhoea. Rogers (1913) felt that the oral changes of the disease were dependent on the length of history of systemic symptoms. Bahr (1915) however felt that exactly similar aphthae to those seen in 'tropical sprue' occurred in normal Europeans; he considered that aphthous ulceration was sometimes associated with secondary infection, possibly monilial in origin, which was superimposed on a progressive nutritional deficit. Brown (1908) was of the opinion that the oral lesions were similar to those of 'thrush'. It is now clear that aphthous ulceration can occur after long-standing malabsorption of other causes; a high incidence has for example been reported in gluten-induced enteropathy (Ferguson, Basu, Asquith, and Cooke, 1975; Wray, Carmichael, Ferguson, Lee, and Russell, 1978).

Today those lesions are rarely seen except in very severe prolonged cases of TM. Painful, burning sensations of the tongue and oral mucosa are sometimes a problem in a severe case; small painful vesicular erosions may occasionally be seen. Rarely there may be a severe glossitis with atrophy of filiform papillae; the fungiform papillae often persist for some time, however, on the atrophic surface.

1.1.7. Oral submucous fibrosis (sclerosing stomatitis, atrophia idiopathica [trophica] mucosae oris)

This is a chronic disease of unknown aetiology which may affect any part of the oral cavity (Joshi, 1953; Lal, 1953; Pindborg, 1965); a slow onset of fibro-elastosis of the submucous tissues with epithelial atrophy occurs. Leukoplakia is frequently associated with it. The condition is probably premalignant (Paymaster, 1956; Pindborg, 1965; Pindborg, Mehta, Gupta, and Daftary, 1968). Most reports are from India, or concern Indians resident in east Africa