



Tropical Gastroenterology

G. C. Cook

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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,
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Contents

Section 1 : Upper gastrointestinal tract

1. The mouth, jaws, and salivary glands	3
1.1 The lips, tongue, and buccal mucosa	3
1.2 The gums and teeth	8
1.3 The jaws	10
1.4 The salivary glands	12
2. The pharynx	17
2.1 Pharyngeal infections	17
2.2 Other inflammatory lesions of the pharynx	19
2.3 Pharyngeal tumours	19
3. The oesophagus	24
3.1 Oesophageal trauma	24
3.2 Carcinoma of the oesophagus (CO)	25
3.3 Oesophageal varices	31
3.4 Infections of the oesophagus	31
3.5 Miscellaneous conditions involving the oesophagus	33
4. The stomach and duodenum	36
4.1 Gastric physiology	36
4.2 Gastritis and widespread mucosal disease	37
4.3 Specific infections of the stomach	38
4.4 Gastric ulcer (GU)	39
4.5 Gastric carcinoma (GC)	42
4.6 Other malignant conditions of the stomach	44
4.7 Duodenal ulcer (DU)	44
4.8 Other complications of peptic ulcer disease	52

Section 2 : The liver and biliary system

5. The tropical liver: effect of chronic intestinal and reticuloendothelial infections	59
5.1 Effect of chronic intestinal infections	61
5.2 Effect of chronic reticuloendothelial infections	62
5.3 Hepatic granulomas	64

6. Hepatic abnormalities associated with systemic bacterial infection	67
6.1 Nature of the jaundice and hepatic dysfunction	67
6.2 Clinical aspects of the jaundice of systemic infection	70
6.3 Other forms of jaundice precipitated by systemic infection	71
7. Acute hepatic injury of viral, bacterial, and parasitic origin	73
7.1 Viral hepatitis	73
7.2 Other acute viral diseases involving the liver	82
7.3 Bacterial infections associated with acute hepatic injury	88
7.4 Parasitic infections associated with acute hepatic injury	90
8. Chronic hepatic injury (excluding alcoholic liver disease)	95
8.1 Active chronic hepatitis (ACH)	95
8.2 Chronic persistent hepatitis	99
8.3 Macronodular cirrhosis (MC)	99
8.4 Other forms of cirrhosis	105
8.5 Other chronic diseases without an infective basis	112
8.6 Other chronic diseases caused by infections	113
8.7 Mechanism of hyperglobulinaemia in chronic hepatic disease	116
9. Hepatocellular carcinoma (HCC) (hepatoma) and other hepatic malignancies	122
9.1 Distribution of HCC	122
9.2 Aetiology of HCC	123
9.3 Presentation of HCC	128
9.4 Pathology of HCC	129
9.5 Investigation of HCC	131
9.6 Treatment of HCC	135
9.7 Other hepatic malignancies in tropical countries	136
10. Schistosomal involvement of the liver	141
10.1 Hepatic schistosomiasis (HS)	141
10.2 Other diseases related to hepatic schistosomiasis	147
11. The portal venous system and portal hypertension (PH) in the tropics	150
11.1 Aetiology of PH	151
11.2 Presentation of PH	153
11.3 Investigation of PH	154
11.4 Treatment of PH	154
12. Space-occupying hepatic lesions: amoebic and pyogenic abscess, and hydatid disease	158
12.1 Amoebic abscess of the liver (AAL)	158
12.2 Pyogenic abscess of the liver (PAL)	164

12.3 Hydatid disease of the liver (HDL)	166
12.4 Other space-occupying lesions of the liver	169
13. Effect of undernutrition and alcohol on the liver	173
13.1 Undernutrition and the liver	173
13.2 Alcoholic liver disease (ALD)	176
14. Diseases of the gall-bladder and biliary system	182
14.1 The gall-bladder in the tropics	182
14.2 Bile duct disease	184
14.3 Biliary cirrhosis	189

Section 3 : The pancreas

15. The pancreas in the tropics	193
15.1 Acute pancreatitis (AP)	193
15.2 The pancreas in kwashiorkor	195
15.3 Chronic calcific pancreatitis (CCP)	195
15.4 Other pancreatic infections in the tropics	199
15.5 Pancreatic carcinoma	200
15.6 The Zollinger–Ellison syndrome.	200

Section 4 : The spleen

16. The tropical splenomegaly syndrome (TSS) and other splenic diseases in the tropics	205
16.1 The spleen in the tropics	205
16.2 The tropical splenomegaly syndrome (TSS)	205
16.3 Other causes of splenomegaly in the tropics	215
16.4 Splenic abscess	217
16.5 Splenic tumours	217
16.6 Rupture of the spleen	218

Section 5 : The small intestine

17. Acute bacterial and viral infections of the small intestine (excluding cholera and salmonellosis)	225
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17.1	Pathological basis for acute diarrhoea	226
17.2	Travellers' diarrhoea	228
17.3	'Pig-bel' disease (enteritis necroticans)	229
17.4	Other bacterial causes of 'food poisoning'	233
17.5	Viral causes of acute enteritis	237
17.6	Secondary hypolactasia	239
17.7	Oral fluid-replacement in non-cholera diarrhoea	239
18.	Cholera	244
18.1	Distribution of cholera	244
18.2	Bacteriology and pathophysiology	245
18.3	Presentation of cholera	247
18.4	Investigation of cholera	248
18.5	Treatment	248
18.6	<i>Vibrio cholerae</i> food-poisoning	250
19.	Small-intestinal parasites, excluding those which are associated with malabsorption	254
19.1	Ancylostomiasis (hookworm disease) (HW)	254
19.2	Ascariasis (roundworm infection) (RW)	259
19.3	Other nematode infections	263
19.4	Trematode infections	264
19.5	Cestode (tapeworm) infections (TW)	265
19.6	Protozoal infections	268
20.	Malabsorption in the tropics	271
20.1	Small-intestinal structure and function in the tropics	271
20.2	Causes of severe malabsorption in the tropics	278
20.3	Postinfective tropical malabsorption (TM)	282
20.4	Giardiasis and tropical malabsorption	304
20.5	Strongyloidiasis and tropical malabsorption	309
20.6	Other small-intestinal parasites associated with malabsorption	314
20.7	<i>Pneumatoxis (cystoides) intestinalis</i> and malabsorption	315
21.	Primary and secondary hypolactasia (lactase-deficiency)	325
21.1	Specific (primary) adult hypolactasia	325
21.2	Secondary hypolactasia	334
21.3	Cows' milk allergy (Cows' milk-sensitive enteropathy)	336
21.4	Sucrase-isomaltase deficiency	336
22.	Salmonellosis (typhoid fever)	340
22.1	Bacteriology and pathology of salmonellosis	340
22.2	Presentation of salmonellosis	342
22.3	Complications of salmonellosis	343
22.4	Investigation of salmonellosis	345
22.5	Treatment of salmonellosis	347
22.6	Food-poisoning caused by <i>Salmonella</i> species	349

23. Chronic infections, tumours, and trauma involving the small intestine and peritoneum	353
23.1 Abdominal tuberculosis	353
23.2 Leprosy involving the small intestine	360
23.3 Other causes of chronic peritonitis	360
23.4 Small-intestinal tumours	361
23.5 Small-intestinal obstruction	364
23.6 Meckel's diverticulum	367
23.7 Trauma to the small intestine	368

Section 6 : The large intestine

24. Shigellosis (bacillary dysentery)	373
24.1 Distribution of shigellosis	373
24.2 Presentation of shigellosis	374
24.3 Severe complications of shigellosis	374
24.4 Bacteriology and pathology of shigellosis	375
24.5 Investigation of shigellosis	376
24.6 Treatment of shigellosis	377
24.7 Food-poisoning by <i>Shigella</i> species	378
25. Intestinal amoebiasis	381
25.1 Distribution of amoebic colitis	382
25.2 Presentation of amoebic colitis	382
25.3 Parasitology and pathology of amoebic colitis	383
25.4 Investigation of amoebic colitis	385
25.5 Treatment of amoebic colitis	386
25.6 Complications of intestinal amoebiasis	389
26. Intestinal schistosomiasis	394
26.1 Parasitology and pathology of intestinal schistosomiasis	395
26.2 <i>Schistosoma mansoni</i> and <i>S. intercalatum</i> infections	396
26.3 <i>Schistosoma japonicum</i> infections	398
26.4 Investigation of intestinal schistosomiasis	399
26.5 Treatment of intestinal schistosomiasis	400
27. Other conditions involving the large intestine in the tropics	406
27.1 Colonic infections	406
27.2 Acute appendicitis	411
27.3 Volvulus of the large intestine	412
27.4 Intussusception of the large intestine	415
27.5 Miscellaneous colonic lesions	418
27.6 Lymphoma of the colon	420

28. Antibiotic-associated colitis	423
28.1 Staphylococcal enterocolitis	423
28.2 Pseudomembranous colitis (PMC)	423
28.3 Allergy and transient ischaemic colitis	426
29. Large-intestinal diseases which are uncommon in the third world	429
29.1 Colonic physiology	429
29.2 Colonic cancer	432
29.3 Non-specific ulcerative colitis	434
29.4 Crohn's disease	434
29.5 Diverticular disease	435
29.6 Haemorrhoids	436
29.7 Maintenance of nutrition in chronic large-intestinal disease	436
30. Diseases involving the anal region	439
30.1 Viral and bacterial infections of the anal region	439
30.2 Mycotic infections of the anal region	442
30.3 Parasitic infections of the anal region	443
30.4 Anorectal suppuration (perianal abscess and fistula-in-ano)	444
30.5 Rectal prolapse	444
30.6 Other conditions involving the anal region	444
INDEX	447

Section 1 : Upper gastrointestinal tract

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 complement 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 complement-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