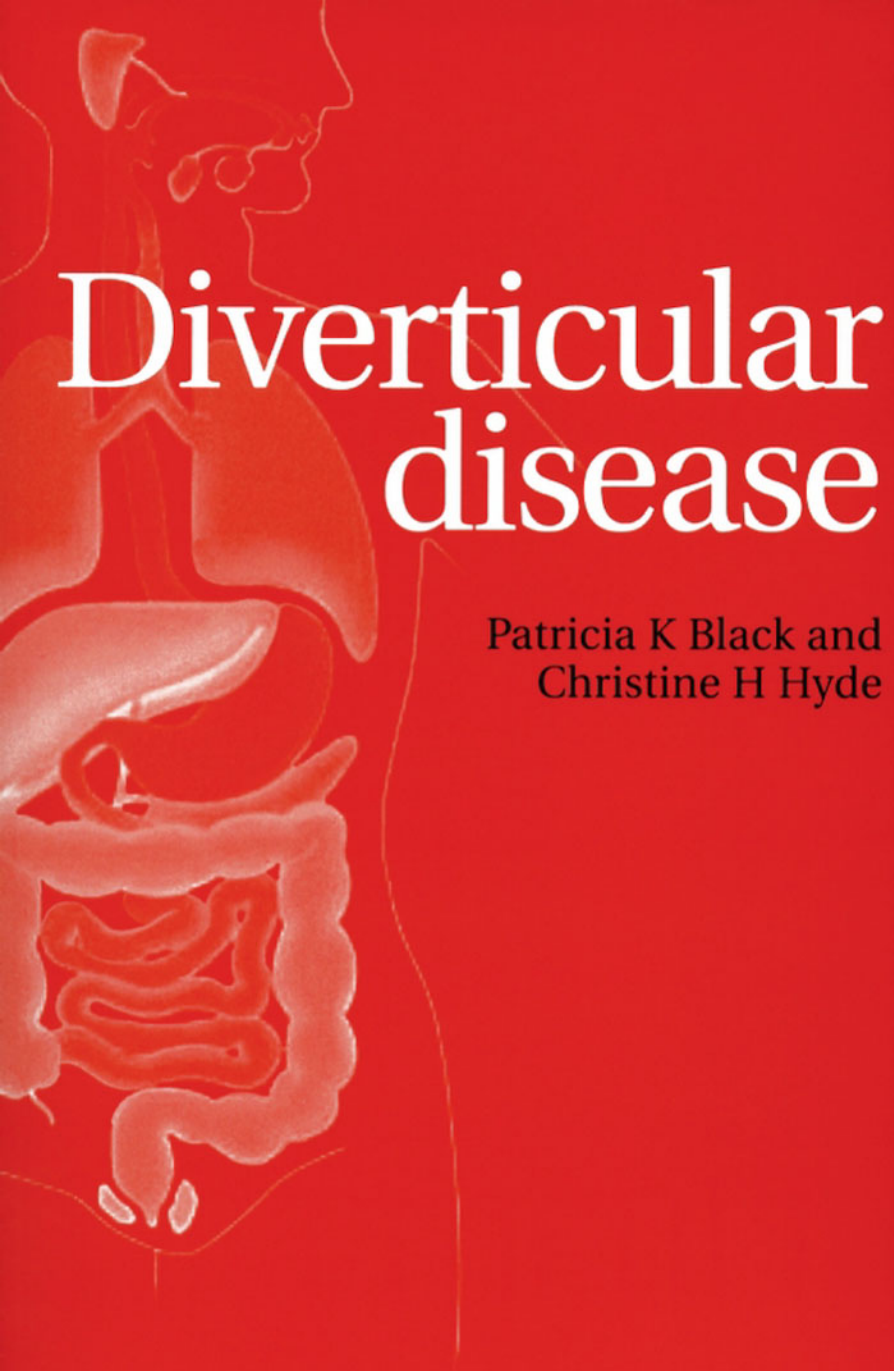

Diverticular disease

Patricia K Black and
Christine H Hyde

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Patricia K Black MSc, SRN, RCNT, FETC, FPA CERT
The Hillingdon Hospital NHS Trust, Middlesex

and

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Dedication

For our families.

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Contents

Foreword

All who know Pat Black and who have read *Holistic Stoma Care* will be delighted to know that its success has encouraged her to undertake another book. Her expertise in stoma and wound care has enabled her to tackle another major pathology – that of diverticular disease.

Having qualified at the Hammersmith Hospital in the late 1960s, Pat Black served her apprenticeship as a staff nurse in teaching and district general hospitals in north-west London. Even as a young nurse her literary ability was recognized, with her publications of case reports and teaching articles.

In the early 1980s I was appointed to the Hillingdon Hospital, where I worked with my colleague John Sales. Having come from the same stable as John – St Bartholomew's and St Mark's – we were able to set up a Gastrointestinal Surgical Unit. We were fortunate to appoint Pat Black as a stoma therapist in 1985, one of the first in a district general hospital. Since then the unit has enlarged and developed, and in 1999 we were fortunate to be able to recruit and appoint Chris Hyde, to support Pat's increasing workload. Chris came to Hillingdon, having followed a varied career in both patient-centred and the commercial side of stoma care.

Over the years our two stoma therapists have also been directly involved with pouch surgery, liver resections and, more recently, rapid access clinics. They have a wealth of experience in the management of diverticular disease. This common condition of the western world can be treated by a simple resection, or can give the colorectal surgeon management problems when resection is complicated, leading to multisystem organ failure. Pat Black and Chris Hyde have been closely involved with the whole spectrum of care, but in particular that relating to the management of wounds and stomas. It is entirely appropriate that they should document their experience on the topic.

Finally, I should like to thank both Pat Black and Chris Hyde for their continued contribution to the working of the Gastrointestinal Surgical Unit at Hillingdon. They have become the focal point for clinic management, audit and the organization of a multidisciplinary team. I wish them success with the book, which I am sure will benefit coloproctologists and nursing staff.

Peter Mitchenere, MS, FRCS
Consultant Surgeon

Preface

Diverticular disease is one of the most common disorders among elderly people in western societies; early in the twentieth century it was believed to be extremely rare and a pathological curiosity. Burkitt and Painter, from as early as 1965, have written much on the subject, in which they called diverticular disease a 'twentieth century problem' and a disease of western societies. In contrast, diverticular disease appears to be rarer in developing countries. The prevalence of diverticular disease appears to increase with age and in western societies the reported frequency of diverticular disease occurs in different age ranges of the population; by the age of 50 years about 33% will exhibit signs of the disease, at 70 years 50% will exhibit signs of the disease and by 80 years 66% can expect to show signs of the disease. Fewer than 20% of patients will go on to develop complications, but these complications may be perforation, fistulae, peritonitis, strictures, bowel obstruction and haemorrhage.

The hypothesis about dietary fibre, or lack of it, has been perpetuated by many writers in human and animal studies (Brodribb and Humphreys, 1976; Findlay et al., 1974; Leakey et al., 1985; Manousos et al., 1985), many believing the disease to be the consequence of a low-fibre diet, as eaten in the western world. In searching the literature, with continuous regularity there are suggestions that fibre in the diet is beneficial in health promotion and disease management. Nurses are ideally placed to help patients understand issues of their disease management and effective promotion of health, but need to be aware of the guidelines and definitions of fibre intake.

We outline the history of diverticular disease and align this with the anatomy and physiology of the bowel, particularly looking at the function of the colon. The disease is then viewed from the uncomplicated cases and how best to look after the patient, to complicated disease, which includes three major complications: diverticulitis, haemorrhage and bowel obstruction. Surgery and its potential outcomes are discussed and a complete chapter on stoma care is included. We are both experienced stoma care nurse specialists

in our own right, and Chapter 8 on stoma care can be used as a stand-alone section for the care of the patient with a colostomy, the rehabilitation process, the correct type of appliance to use and care in the community.

For many, having a colostomy, often undertaken as an emergency procedure, can be devastating and Chapter 8 endeavours to help the practitioner to think in a wider-ranging way and to consider the patient's culture, socioeconomic situation, beliefs, religion and any practices that may relate to his or her ill-health. Without understanding the culture in which the patient has grown up, the practitioner will have difficulty in understanding the reaction to the disease and illness, particularly when a stoma is formed.

Chapter 9 looks at literature from many countries around the world, both developed and developing, to compare the incidence of diverticular disease. Histologically, right-sided diverticular disease was the predominant pattern in far eastern countries, contrasting sharply with predominantly left-sided disease in the sigmoid colon in western countries.

As practitioners, most of us feel that we know enough about healthy eating and fibre in the diet because we have been advising people about it for many years, but fibre is an unexpectedly complex food fraction and a new fibre-oriented vocabulary is emerging that can confuse and misinform people. Chapter 11 helps to update current thinking, advising the practitioner of the new terminology of non-starch polysaccharides (NSPs) that are part of dietary fibre intake and the introduction of the dietary reference values and the Englyst measurement method (Department of Health, 1991).

Evidence-based medicine and evidence-based health care, aligned with research, can help both the promotion of good nursing practice and the understanding of failures in practice, with the aim of rectifying the situation. Nurses have an obligation to keep up with current literature in their field, read it critically and make balanced judgements about the quality and relevance of the work in relation to their practice.

Chapter 12 looks at alternative therapies and what may help and be of benefit to a patient with uncomplicated diverticular disease from Ayurvedic medicine to yoga. In Chapter 13 we look at consensus development in the diagnosis and development of diverticular disease. Importantly, in this chapter the surgical management is reviewed with regard to lowering morbidity, mortality and stoma formation, and who should be operating on patients to enable the patient to have the best possible outcome.

A glossary is included and a list of agencies that can help and support the patient who has a diagnosis of diverticular disease and his or her family.

Patricia K Black
Christine H Hyde
2004

The Authors

Patricia K. Black, MSc, SRN, RCNT, FETC, FPA CERT

Pat Black has been a Clinical Nurse Specialist in stoma care for 19 years at the Hillingdon Hospital NHS Trust. She undertook her Masters degree in Medical Anthropology at Brunel University in 1990. She has travelled extensively in eastern Europe teaching stoma care and setting up courses. She has lectured across the world on all continents at stoma care and colorectal conferences. She publishes widely in the nursing and medical press and in the national media. Her particular interest in stoma care is the patient who comes from an ethnic minority and the politics of sponsorship in stoma care. She is currently course leader for 'Foundations in stoma care' for nurses from non-specialist settings in association with Buckinghamshire Chilterns University College.

Christine H. Hyde, SRN

Christine Hyde came to the Hillingdon Hospital NHS Trust in 1999 as Clinical Nurse Specialist in colorectal nursing. She has worked with the multidisciplinary team and four years ago set up rapid access clinics for rectal bleeding and colorectal cancer. She has wide experience in the private and commercial sector in stoma care and has previously worked as a sister in a busy accident and emergency department; she then went to H.J. Heinz Company as an occupational health nurse. Currently she co-facilitates the 'Foundations in stoma care' course for nurses in non-specialist settings in association with Buckinghamshire Chilterns University College.

Acknowledgements

Writing a book is never as easy as it seems when the commission is accepted. Time has this strange way of suddenly running out as stress levels escalate. To the people who have supported us and stoically put up with us while we completed this project, we give our grateful thanks and apologize again for the days when we were cross and tired. There is no particular reason for the order of thanks, but only as they came to mind: Sue Rigg, Colorectal Co-ordinator; Robin Kantor, Consultant Radiologist; Barbara Stuchfield, Clinical Nurse Specialist Stoma Care; Sonya Francis, secretary to Mr Mitchenere; Juliette Fulham, Colorectal Practice Development Nurse/Stoma Care Nurse; Peter Mitchenere, Consultant Colorectal Surgeon and Clinical Director; Geraldine Gaffney, Head Nurse of Surgery; Bob Nye, Nancy Jackson, Paul Newman at Dansac, and many other friends who always enquired how the book was going. We are also grateful to Dansac Ltd for their support and encouragement.

Chapter 1
The History of Diverticular Disease

The written history of bowel-associated problems can be traced as far back as the Book of Judges in the Bible showing that the pre-Christian Israelites were well aware of abdominal injuries and problems (Black, 2000). But, even before the Bible recordings, the Egyptians in 2000 BC recognized disorders of the bowel, although writings often recorded on papyrus were not specific as to what these may have been. The Greeks and Romans were not to be left out in their writings on bowel problems; however, it was the Greeks with Hippocrates and Herodotus who made specific mention of bowel disease. Although Hippocrates is known as the 'father' of medicine (the Hippocratic oath) and is the most celebrated physician in history, little is known about him, other than that he lived on the island of Kos and taught medicine for money. He tried to dispel the idea of alternative medicine to lay the early foundations of biomedicine. Herodotus, although not a medical man, was a historian, who on his travels collected historical, geographical, ethnological, mythological and archaeological information recording wars and their causes.

After the Romans the period of time until AD 1100 was to be known as the 'Dark Ages' because it has been judged as a time in the western world of unenlightenment and obscurity with political fragmentation and a lack of centres of learning. Yet, although the history of stomas can be traced as far back as Celsus in 55 BC to AD 7, quoted by Dinnick in 1934, diverticular disease was first described by Littre (1732) when he dissected a neonate and described what he saw in the bowel as a diverticular hernia.

In 1783, Matthew Baillie a Scottish physician who studied with William Hunter, succeeded to Hunter's famous anatomy school in London and in 1793 wrote the first treatise in English on morbid anatomy. It was within this treatise that Baillie mentioned diverticular disease (Oschner and Bargen, 1935). In the twentieth century Painter and Burkitt (1975) suggest that the history of diverticular disease can be divided into five phases:

1. The disease as a curiosity
2. The recognition of diverticular disease as a clinical problem
3. The recognition of diverticular disease as a growing medical problem
4. The surgical approach to diverticular disease
5. The role of the colonic muscle in the pathogenesis of the disease.

In 1927, Spriggs and Marxer suggested that the term 'diverticulum' originated from the word 'divertikel' which was said to have been used by Fleischman in 1815 in describing this anomaly in the colon. Between 1815 and 1869 many writers of medical articles were stating that they all believed that these 'divertikel' were not nascent but acquired later in life – thought to be caused by constipation. Even at this early stage in medical history, it was recognized that a fistula could be one of the associated complications of diverticular disease (Jones, 1859).

Although rarely seen in the nineteenth century, the recognition of diverticular disease as a clinical problem was emerging at the beginning of the twentieth century and was described as having complications such as fistulas, adhesions, peritonitis and stenosis (Beer, 1904). In the UK it was not until 1917 that the first 'classic' description of diverticular disease was published by Telling and Grunner (1917) before any medical textbooks.

The recognition of the size of the problem of diverticular disease in medicine was revealed once radiology advanced and could show that diverticula were not unusual; postmortem and barium studies were undertaken to demonstrate this. On the other side of the Atlantic, Mayo (1930) estimated that 5% of patients over the age of 40 years would demonstrate diverticula in their colons. This figure concurs with current postmortem studies undertaken in both Europe and America. Up until World War II, resection of the colon carried a high mortality rate of up to 10%. As a result of this high mortality rate, doctors felt that there should be preventive ways to stop diverticula of the bowel along with their complications and surgery. Believing that roughage could irritate the colon, Spriggs and Marxer (1925) believed that the bowel should be cleansed and there should be plenty of vegetables and fruit in the diet, but that any irritants from fruit and vegetables such as pips, stalks, pith and tough skins should be left out of the diet. As a result of the removal of these irritants in the diet and no other roughage, the low residue diet was born and recommended for diverticular disease with no proof that it would be of any value.

In the 1940s, when antibiotics were on the horizon, Smithwick (1942) advocated that resection of the offending colon with minimal mortality could be carried out, provided that the patient was fully assessed and prepared. Resection of the diseased colon then became a standard surgical

procedure. In 1923 Hartmann (Black, 2000) had perfected the end-colostomy and this procedure was used and is still often used in many hospitals as a two-stage procedure for the resection of diverticular disease. However, in the twenty-first century a new consensus of opinion is evolving towards a single-stage procedure, although selection for a single or staged resection remains the most controversial issue.

The physiology of the colon related to the pathology of diverticular disease, which covers the fifth phase of Painter and Burkitt's (1975) discussion when they and Arfwidsson (1964) investigated colonic pressure in relation to the pathogenesis of the disease, and in 1964 Painter had suggested that the pain, often termed colic, that patients experience in diverticular disease may be caused by 'excessive segmentation leading to an intermittent functional colonic obstruction'.

Aetiology

The historical perspective on the aetiology of diverticular disease can be recognized as far back as 1853 when Virchow (Rankin and Brown, 1930) described inflammatory areas, particularly in the sigmoid colon flexures, as 'isolated circumscribed adhesive peritonitis' and in 1869 when Klebs investigated the relationship of diverticula and their associated blood vessels in the intestinal wall.

In 1930, Rankin and Brown were describing diverticula and their aetiology as a controversial subject, whereas Erdmann (1932) postulated that the presence of diverticula in the intestine was of no more importance than diverticula in other organs. Bell (1929) considered that multiple diverticula, i.e. diverticulosis, was mainly of academic interest. Mayo (1930) suggested that muscular weakness of the colon and not constipation or obesity was the underlying cause of diverticular disease.

Lockhart-Mummery and Hodgson (1931) suggested that after a certain age (45 years) the muscle sheath of the colon could lose its tone and diverticula result from the muscle weakness. Even at the beginning of the twentieth century there was wide divergence of opinion as to whether diverticula occur more frequently at the area of the intestinal wall where the bowel is weak, as previously described, or whether they are associated with the openings between blood vessels coming in from the mesenteric side, because, in the colon, diverticula present at any point along the circumference of the colon wall have added confusion to the understanding of the aetiology of diverticular disease (Rankin and Brown, 1930).

It was initially supposed that a weakened colonic muscle wall in obese people resulted in fat being deposited around the blood vessels, so making a

potential defect in the muscle coat, although it was also observed that people who were thin and people who were wasted had colonic diverticula (Klebs, 1869; Edel, 1894). As no hard evidence could be found to support this hypothesis, obesity has been rejected as an aetiological factor in diverticular disease.

There have been many reasons postulated for weakening of the intestinal wall, among which are old age, muscular atrophy, fatty atrophy and even bacterial damage of the intestine (Henderson, 1994). Thinness of the colon muscle coat, enabling the mucosa and serosa to be in close approximation, is thought to be caused by excessive segmentation which is an acquired defect and not nascent.

In observing the history of dietary change and diverticular disease, Painter and Burkitt (1975) suggest that the British diet started to change from 1870. White flour with little fibre in it was available as early as 1800 and was taken daily mixed with rye and oatmeal, making a fibre intake of 600 g. As the development of transport and the migration of the work force spread around the country, and refrigeration became available, refined sugars within the diet became more freely available for all classes together with meat as a regular meal. Between the years 1860 and 1890 the intake of bread in the diet decreased and refined sugar intake doubled. Other than the periods of the two world wars, when there was food rationing, this trend has continued. If diverticulosis is caused by the move from a high-residue to a low-residue diet, it would follow that, about 40 years after 1880 and these dietary changes, diverticular disease would be noted to become more widespread, as in fact it did in the UK.

In looking at the cultural prevalence of diverticular disease, it has been found that there is a relationship between a population and its economic development (see Chapter 9) and industrialization. Painter and Burkitt (1975) suggest that it takes about 40 years for diverticular disease to develop within a community, after that community departs from its traditional eating habits, and that 'consequently the disease will not be found in a population until its diet and hence the quality of the faecal stream that its colons have to propel have been altered for about forty years'.

Painter et al. (1972), in researching historical and epidemiological studies on diverticular disease, found that they contained much circumstantial evidence to suggest that economically developed countries with altered dietary intake at the turn of the nineteenth century were more prone to diverticular disease. If diverticular disease is considered to be a deficiency disease, a deficiency of high fibre in the population's food intake, the answer must be to retrace our dietary footsteps.

The word 'diverticula' is the plural of the Latin word diverticulum, meaning a wayside house of ill repute. It was in 1916 that diverticular disease was first mentioned in textbooks in the UK and diverticulosis was first mentioned in 1914 (Painter and Burkitt, 1975). Although diverticular disease was little documented or seen in the nineteenth century, the few cases that were documented were accurately described by today's knowledge of the disease. In addition radiographic diagnosis did not become available until a century later. Painter and Burkitt (1975), in their historical overview of diverticular disease, suggest that the term 'divertikel' was used as early as 1815 by Fleischman (in Spriggs and Marxer, 1925). In 1859 in the *Transactions of the Pathological Society of London*, Sidney Jones describes a colovesicular fistula that was caused by diverticulitis and in 1870 Loomis (cited in Hartwell and Cecil, 1910) noted a case of peritonitis as the outcome of diverticulitis.

Chapter 2
Anatomy and Physiology

The digestive system starts with the mouth and ends at the anus. The tract would measure approximately 10 metres if it were laid out in a straight line.

The purpose of the digestive system (Figure 2.1) is to produce a chemical and mechanical breakdown of food:

- Ingestion: the act of eating
- Digestion: mechanical and chemical
- Absorption
- Defecation or elimination.

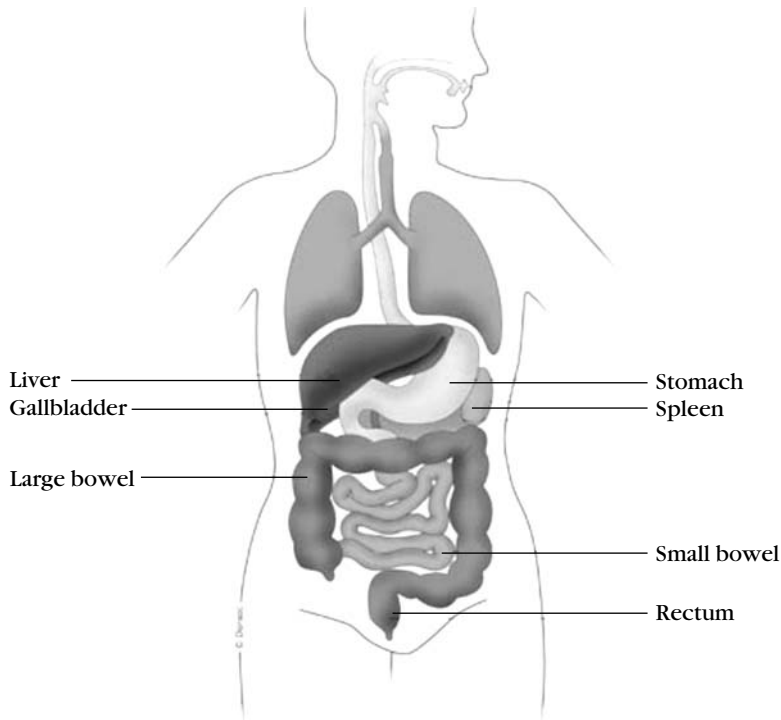

Figure 2.1 The digestive system. (Courtesy of Dansac Ltd.)

The digestive tract or alimentary canal is an epithelial lined muscular tube along which food passes by a movement called peristalsis. Peristalsis is a muscular action of dilatation followed by contractions as muscle fibres relax and contract (Jackson, 1979). This movement is not continuous throughout the tract because some organs store the food bolus whereas others churn and mix it with secreted enzymes. The food is then ingested and digested by various digestive juices and enzymes. Food is required to produce heat and energy for daily living and the food undergoes a complex process called digestion.

The enzymes that are secreted throughout the gut digest food. They convert the protein, carbohydrates and fats within food into their simplistic form, so that they can pass directly into the blood supply. The inorganic salts, water and vitamins are absorbed without chemical changes and waste products are eliminated

The digestive process changes as it passes along the alimentary tract. In the upper part of the tract it is predominantly under nervous control whereas further along hormones control the tract. The control reverts back to the nervous system in the rectum and anal canal. The food in the alimentary tract, along with a local nerve network, regulates the process of digestion.

Defecation or the elimination of waste products from the digestive process is triggered by the ingestion of food and the mass movement process of the bowel that occurs twice a day; both of these processes give us the desire to defecate. Control can be exercised to delay defecation. It is a process called toilet training, which begins at an early age and allows the control of elimination.

The Mouth

The food enters the mouth, the opening of which is protected by the lips. Food is bitten by the front teeth, incisors, passed to the back teeth, premolars and molars, to be masticated and chewed, and mixed with the saliva to form a bolus of food that will slide into the oesophagus through the action of swallowing.

The saliva is an alkaline substance secreted by the thought, sight, smell and taste of food through the salivary glands (experiment – think of citrus fruit and see if your mouth waters). With the aid of peristalsis (an involuntary muscular movement) the bolus of masticated food is propelled into the oesophagus. This peristaltic movement allows food and liquid to be swallowed together without choking.

The salivary glands are three pairs of glands comprising:

- The parotid gland: located in front and under the ears between the skin and muscle.

- The submandibular gland: located under the tongue.
- The sublingual gland: located anteriorly to the submandibular glands.

The salivary glands produce approximately 1 to 1.5 litres of saliva each day. The saliva mixes into the food with the action of chewing.

The parotid salivary gland produces salivary amylase, also known as ptyalin, which converts starch to simple sugar; the submandibular salivary gland produces salivary amylase and some mucus whereas the sublingual salivary glands produce a thicker version of mucus. The digestive process that happens when the saliva mixes with the food is instigated by the breakdown of carbohydrates. The saliva also has a cleansing function in the mouth, which allows taste buds to distinguish between sweet and savoury flavours. Saliva is rich in calcium and this is how teeth are protected from decalcification. The saliva is a lubricant that enables the bolus to be swallowed by making it moist; it also acts as an antiseptic.

Chewing continues until the food is manageable and moist; this is now called a bolus which is manipulated by the tongue and rolled into the pharynx. The soft palate closes the nasopharynx and the epiglottis moves to allow the food passage into the oesophagus (Green, 1978; Jackson, 1979; Tortora and Anagnostakos, 1981).

The Oesophagus

The oesophagus is a muscular tube approximately 25 cm long; it is designed to transport the bolus of food from the mouth to the stomach with the assistance of the mucus that it produces. The oesophagus passes in front of the vertebral column in the chest and through the diaphragm. It is positioned behind the left lobe of the liver and enters the stomach at the cardia. The oesophagus is made up of four layers, as is the rest of the digestive tract:

1. Mucosa: epithelium lined with squamous cells
2. Submucosa: contains blood vessels and nerves
3. Muscular: contains an inner ring of smooth muscle
4. Fibrous: dense connective tissue of longitudinal folds that contain the stratified squamous epithelium.

The food bolus passes into the oesophagus by the action of swallowing and then by peristalsis. It is possible to eat and drink at the same time and in a variety of positions from sitting, standing, lying down and huddled up in a crouched sitting position, all without choking as a result of the vagus nerve stimulating peristalsis. The vagus nerve is the tenth cranial nerve and is responsible for gut stimulation. The bolus of food has to pass over the larynx

where it shares a common opening with the oesophagus. This is achieved by the tongue closing the back of the mouth, the nasal passage being blocked by the soft palate and the epiglottis covering the larynx as the bolus is swallowed. When this process fails we say that 'food has gone down the wrong way' and a cough reflex brings the food back. The swallowing reflex may be lost in anyone who has had a cardiovascular accident or is unconscious.

The food bolus takes between 4 and 8 seconds to travel into the stomach; liquid is much faster. The bolus continues along the oesophagus until it reaches the stomach via the cardiac sphincter.

The Stomach

The stomach is a J-shaped pouch with greater and lesser curves and ends at the pyloric sphincter. It is made up of the following structures:

- The cardia joins the oesophagus to the stomach
- The fundus is the dome-shaped part of the stomach and extends to the left above the cardia
- The body acts as the main reservoir
- The pylorus, is the last section before the pyloric sphincter

The stomach is made of three layers of muscle: the inner oblique muscle, middle circular muscle and outer longitudinal muscle. The stomach is a storage organ and is situated just below the diaphragm between the oesophagus and the duodenum; it is approximately 25–30 cm long and 10–15 cm wide at its widest part. The pyloric sphincter protects the outlet into the duodenum until the food is ready to pass as chyme. The bolus is mixed with the gastric juices which are controlled by the vagus nerve and the hormone gastrin. The motion of the movement of the bolus is to mix it with the gastric juices to change the bolus into chyme.

The gastric secretions consist of:

- water, mineral salts and mucus
- hydrochloric acid
- pepsinogen.

The vagus nerve and the hormone gastrin control the production of the gastric juices. The pyloric region secretes the hormone gastrin into the blood supply and it circulates in the body before returning to the cells in the stomach.

The vagus nerve

The vagus nerve is the tenth cranial nerve and the longest of the cranial nerves; its Latin meaning is 'wandering'. The nerve leaves the brain through the neck, into the thorax and abdomen. It supplies most of the muscles of the pharynx and soft palate. The vagus nerve enters the thorax and branches go to the lungs for bronchodilatation, to the oesophagus for peristalsis and to the heart to slow down the heart rate. In the abdomen, branches enter the stomach, pancreas, small intestines, large intestines and the colon for secretion and constriction of smooth muscle. Nerves in the abdomen and thorax join the left and right vagus nerves to ascend beside the left and right common carotid arteries.

The food bolus is churned and mixed with the pepsin and hydrochloric acid to form a thick paste that is called chyme. The mucus-containing food mixture is composed of partially digested foods mixed with the gastric juices. Every few minutes the pyloric sphincter passes a small amount of chyme into the duodenum. It can take between 2 and 4 hours for the stomach to empty.

Digestive enzymes

The role of enzymes is to stimulate chemical reactions in the food; to achieve this they require a specific temperature and pH.

Rennin

Rennin is required in infants. Its function is to curdle the milk ingested by the young child, which prevents milk from leaving the stomach too quickly, allowing time for the absorption process.

Pepsin

Pepsinogen is secreted into the gastric juice from both mucus cells and chief cells. When it is secreted, stomach acid instigates the conversion into pepsin, which breaks down protein into its simplest form of peptones. Pepsin requires a pH of 2 to work.

Hydrochloric acid

Hydrochloric acid is secreted into the lumen, which turns the stomach into an acid environment. The hydrochloric acid is required to activate the pepsin, so it too is required to digest protein. A hormone called gastrin is important in the control of acid secretion. Hydrochloric acid has quite a few functions:

- It provides an acid reaction needed by the gastric enzymes
- It is a solution for killing some bacteria
- It controls the pylorus
- It inhibits the action of amylase/ptyalin
- It changes pepsinogen into pepsin.

The production is controlled by the vagus nerve and the hormone gastrin. Gastrin is a peptide that contains 17 amino acids. The total amount of gastric juice produced is approximately 3 litres in 24 hours. Mucus covers the entire surface of the stomach to coat and lubricate the stomach wall, so preventing digestion of the stomach itself.

The intrinsic factor

The mucosal lining of the stomach secretes intrinsic factor which is required for the absorption of vitamin B_{12} in the ileum. Vitamin B_{12} is released from the ingested proteins through the action of pepsin and is stored in the liver. The failure to produce intrinsic factor causes pernicious anaemia.

The Liver

The liver is the largest gland in the human body and it is vital to life; death is inevitable without a liver. The liver is situated on the right side, opposite the stomach.

The liver weighs between 1.3 and 1.5 kg depending on age and sex. The liver is a very vascular organ which is made up of left and right lobes. It is found under the diaphragm, filling most of the right hypochondrium. If the liver becomes damaged by trauma it bleeds as a result of its profuse blood supply. A double supply of blood goes to the liver from the hepatic artery. From the portal vein, the liver is supplied with deoxygenated blood and the following nutrients:

- glycogen
- copper
- iron
- vitamins A, D, E and K.

In addition:

- The liver stores fat until it is required, then breaks it down to provide energy
- Urea is manufactured from excess amino acids
- Detoxification of poisons and drugs takes place

- Carotene is synthesized from vitamin A
- Antibodies and antitoxins are manufactured
- The manufacture of heparin takes place
- The liver is the main heat-producing organ of the body
- Synthesis of plasma proteins occurs
- Uric acid and urea are broken down from worn-out cells
- Prothrombin and fibrinogen are synthesized from amino acids (Jackson, 1979).

The liver has the ability to regenerate itself if needed. The liver supplies various chemical substances to the body via the blood supply through the hepatic vein. Waste is secreted via the hepatic ducts that form part of the common bile duct. The liver acts as a storage area for vitamins A, D and B_{12}. Harmful and poisonous substances absorbed by the intestines are stopped from circulating in the blood supply by the liver.

The liver produces about 1 litre of bile every day; it is stored in the gallbladder and enters the duodenum via the bile duct.

Bile properties

- Emulsifies fats
- Stimulates peristalsis
- Route for the excretion of toxic substances, poisons, alcohol, drugs and byproducts of red blood cells.

The Gallbladder

The gallbladder lives in the right lobe of the liver. The liver produces up to 1 litre of bile each day which is stored in the gallbladder. Bile is not absorbed into the blood supply The bile changes the chyme into its brown colour and the bile salts, one of which is amylase, are important for the digestion of fats.

The Pancreas

The pancreas is a lobulated gland 12–15 cm in length**.** This pinky-grey gland lies between the posterior abdominal wall of the duodenum and the spleen. It has a head, body and tail. The pancreatic duct lies within the whole length of the pancreas, terminating at the common bile duct where it meets the pancreatic duct at the hepatopancreatic duct. The pancreas produces up to one and a half litres of pancreatic juices each day; these assist further digestion of foods.

The pancreas has a twofold function: digestion and an endocrine function. The latter is the production of insulin and glucagon, which are

required to regulate the levels of sugar in the body. Glucagon stimulates the conversion of glycogen to glucose.

Ileum (small bowel) (Figure 2.2)

Duodenum

The duodenum is the first part of the small intestine. It starts at the pylorus and ends at the jejunum; it is shaped like the letter C and is about 25 cm long and curves around the head of the pancreas until it meets the jejunum. The hepatopancreatic ampulla drains enzymes into the duodenum via a duct.

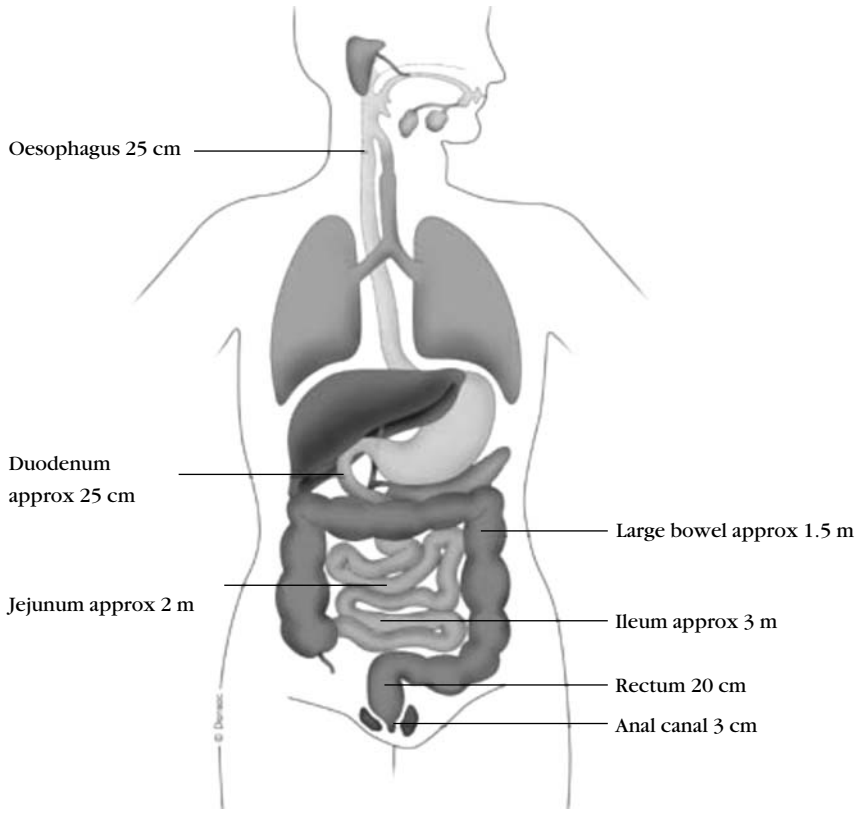

Figure 2.2 Measurements of the digestive system.

Jejunum

The jejunum is 2 m long; it is thicker and wider than the ileum and the folds are covered by finger-like villi. These villi contain a rich blood supply – they are the absorbers of the digestive process.

The Ileum (small bowel)

The ileum is a continuation of the jejunum. It is about 3 m long and has permanent circular folds (3–10 mm), which promote the mixing of the chyme. This enters the villi – the finger-like projections of the mucosal layer into the intestinal lumen.

Absorption of chyme is via the villi to the capillary and venous drainage of the gut. The remaining chyme, which is rich in bacteria and unwanted undigested food, enters the large bowel at the caecal valve.

The whole digestive process is aimed at processing food into the most simplistic forms that can be absorbed easily into the blood and lymphatic systems. About 90% of this absorption takes place in the small intestine. The other 10% is absorbed in the stomach or large bowel.

The Colon (large bowel)

The large bowl is about 1.5 m long, starting with the caecum and ending at the rectum. The caecum starts at the ileocaecal valve, which acts as a one-way valve to stop the reflux of bacteria from the colon into the small bowel.

The colon proceeds into the ascending colon, which passes up the right-hand side of the abdomen to the lower edge of the liver at the hepatic flexure. The transverse colon crosses from right to left across the abdomen, and extends from the hepatic to the splenic flexure. The endoscopist recognizes this area of the colon by its triangular shape. The descending colon extends from the splenic flexure to the sigmoid juncture – about 30 cm. It goes down the left side of the abdomen from the spleen to the left iliac crest. The sigmoid colon is S shaped and 40 cm long (Jackson, 1979). The rectum is a curved pouch, 9 cm long in young people, extending to 20 cm in adults. The rectum extends into the anal canal and is controlled by internal and external sphincters, which control defecation.

Function of the large bowel

The main function of the large bowel is absorption, automatic movement, and the formation and excretion of faeces.

The chyme is very liquid when it passes into the large bowel; in the region of 500 ml passes into the colon each day. It is here in the colon that absorption takes place: mineral salts, vitamins and some drugs are absorbed, along with 90% of the water content of the chyme. The large bowel propels the residue towards the rectum where it is stored. The large bowel motility comprises mass movement and peristalsis.

There are usually three or four mass movements during the day, which prompt defecation. A strong force occurs in the transverse colon pushing

the chyme towards the rectum. This movement is triggered by eating and entry of food into the stomach, which is why the urge to produce a stool frequently happens either following a meal or during a meal.

Defecation is a voluntary act involving the brain, brain stem and spinal cord. The usually empty rectum fills; nerve endings are stretched by the contents of the sigmoid colon, causing involuntary contractions of the muscle of the rectum and relaxation of the internal anal sphincter; the voluntary external sphincter completes the process. In babies the act is an autonomic response until they are 12–18 months of age when the external anal sphincter comes under voluntary control.

The large bowel is heavily colonized by certain types of bacteria:

- *Escherichia coli*
- *Enterobacter aerogenes*
- *Streptococcus faecalis*
- *Clostridium perfringens*.

Bacterial colonization happens within the first week of life from swallowed bacteria and maternal contaminations. The colon is sterile for the first week of life (Society of Gastroenterology Nurses and Associates, 1998). Bacteria in the bowel are either harmful or helpful. If the bacteria are harmful they may cause diarrhoea; however, the helpful bacteria in the gut provide vitamins, mainly in the B group. A number of antibiotics, if taken frequently or in large doses, kill the helpful bacteria, again giving rise to diarrhoea (Green, 1978). The bowel maintains a flora of useful bacteria that are needed in digestion.

The Rectum

The rectum is the final 20 cm of the digestive tract and the final 3 cm of this is known as the anal canal. The rectum can accommodate about 400 ml of stool.

The anal canal ends with the anus, which has two sphincters: the internal sphincter of smooth muscle and the external sphincter, which is made up of skeletal muscle. The anal canal is lined by mucosal epithelium above the anal verge and squamous epithelium below. The anus is kept closed, except for the action of defecation, by the tone of the external sphincter. In some people, however, for a variety of reasons the sphincter loses its tone and can become weak resulting in incontinence.

Anatomy and physiology of diverticular disease

Diverticular disease is a twentieth century disease that results in part from changes in diet, age and lifestyle although there is little research to support

this (Bassotti et al., 2003). It is one of the most frequent diseases seen in gastroenterology departments.

Diverticula are small herniations in the bowel wall; they can occur anywhere in the bowel. The diverticular pouches usually appear in the descending and sigmoid colon (Stollman and Rashkin, 1999), frequently manifesting at the weakest point in the colonic wall where the blood vessels supply the mucosa in the circular muscle layer. A diverticulum is an outpouching of the mucosa of the lining of the bowel. Diverticulosis is the name give to this manifestation and most patients with these diverticula will not have any symptoms. The symptoms of diverticular disease in westernised countries usually relate to the sigmoid colon. Right-sided diverticular disease is more prevalent in the eastern countries of the world. In countries where the diet is high in fibre and very low in refined carbohydrates, diverticular disease is virtually unknown (Hyde, 2003).

The outpouches are blind ends within the bowel wall where undigested food particles, faecal matter and debris can collect and become trapped; this can lead to inflammation and then to diverticulitis. The diverticular pouches usually appear in the descending and sigmoid colon; they can be a single diverticulum or in abundance. Sigmoid diverticular disease is common in the western world and thought to be caused by lack of fibre (Painter and Burkitt, 1975) and over-refined carbohydrates and flour (Keighley and Williams, 1997) in the diet.

Changes in lifestyle and eating patterns in the latter half of the twentieth century are thought to have contributed to the increase in diverticular disease. Another cause of diverticular disease is a consequence of high intraluminal pressure in the bowel, together with slow transit times of the stool. Stollman and Rashkin (1999) say that:

> High intraluminal pressure is caused by segmental contractions of the circular muscles and by contractions of the colonic wall between these segments.

Slow transit time of the stool is the time that the faecal matter takes to navigate the colon. This can assist the formation of the diverticula, which under pressure bulge at their weakest points. Fibre helps to speed this process but, as already stated, the diet is now lacking adequate amounts of fibre to accomplish this. The faecal matter stays in the colon for longer periods of time and becomes more constipated as a result, causing more pressure and straining on evacuation of the bowels.

All of these activities can result in the formation of a diverticulum at the weakest point in the circular muscle layer, where the blood vessels supply the mucosa (Bassotti et al., 2003). The lumen of the colon is at its narrowest

in the sigmoid colon and therefore comes under vast intraluminal pressure; together with slow transit time of the stool through the colon (Mimura et al., 2002), this causes pressure that is exerted on the bowel wall, causing herniations at the weakest point.