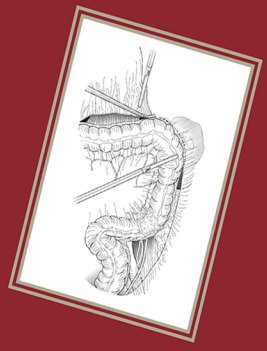
FOURTH EDITION

Keighley & Williams' Surgery of the Anus, Rectum and Colon

Volume 1



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Volume 2

Preface

Colorectal surgery has made so many major strides since the third edition of *Surgery of the Anus, Rectum and Colon* was published in 2008 that it is no longer possible for two individuals ably supported by a team of four others to produce an authoritative account of the ever-expanding speciality, even with the help of guest contributors. Hence, we decided to make this an international resource written by the world's leading experts in the field.

We also recognise that colorectal surgery has changed beyond recognition since the first edition. With the advent of laparoscopic and endoscopic surgery and with colorectal cancer screening, new technologies have emerged, such as robotic surgery and TaTME, where there is a need for national and even international training and mentoring programmes. The ethics of workshop training with mentoring and audit is now without dispute. The public expect that they are no longer victims of the learning curve. Likewise in many areas of practice, multidisciplinary team working is an essential component of responsible practice, not only in colorectal cancer, but in functional disease and inflammatory disease as well. There is thus a far greater need to discuss the risks and benefits of potential therapeutic options openly with the patient and their partner with regard not only to possible complications but also to functional outcome. The impact of specialist high-volume practice in outcome for particular needs, such as advanced rectal cancer, endometriosis, pouch surgery and recurrent Crohn's disease, where there is potential avoidable bowel loss is an active debate that is likely to change clinical practice.

In this fourth edition we have 88 chapters written by at least one senior author, but in many cases by a team of up to four experts representing the specific fields of focussed medical practice. This new venture has relied heavily on our eight section editors, who have coordinated the key areas of clinical practice: general principles – Peter Sagar, Leeds, UK; proctology – Andrew Hill, Auckland, New Zealand; functional disorders – Charles Knowles, Royal London Hospital, UK; neoplastic disease – Stefan Post, Mannheim, Germany; diverticular disease – Patricia Roberts, Lahey Clinic, Boston, USA; inflammatory bowel disease – Willem Bemelman, Amsterdam, Netherlands; acute colorectal disease – Susan Galandiuk, Louisville, Kentucky, USA and miscellaneous topics – John Monson, Florida Hospital System, USA. We are enormously grateful for their expertise and hard work in ensuring their sections represent state-of-the-art developments in their fields.

We have been rigorous in avoiding duplication and providing cross referencing throughout the text. The text is also available in an e-version with access to links that have enhanced the accessibility of this particular edition, and which have been unavailable in the past. We have ensured that, unlike many textbooks in this field, the particular management issues of acutely presenting patients who are critically ill with sepsis or obstruction have been described.

We are fortunate to have the expertise of a new publisher, CRC Press of the Taylor & Francis Group, who have taken over from Elsevier Saunders, and we particularly wish to thank Cherry Allen as editorial assistant for her tireless support throughout the collection and reformatting of material, as well as the overarching managerial supervision of Miranda Bromage.

We wish to thank our long-suffering wives for hours spent ensuring that the product provides the most comprehensive account of our speciality, but we will not be doing this again; the future lies in the next generation of innovators.

Michael R.B. Keighley
Norman S. Williams

Acknowledgements

I would like to make special mention of Mike Keighley's significant contribution to the 4th edition of this book. Although I have had an input in the planning of this edition, the lion's share of the oversight has been provided by Mike. Without his immense drive in reading the whole text to eliminate repetition, assisting the section editors in chasing up tardy contributions, ensuring accurate cross referencing and finding some replacement authors at short notice, the book would never have been completed to the high standard to which we both aspire.

Norman S. Williams

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SECTION

1

Preface: General Principles of Colorectal Surgery

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This section describes the basic physiological principles of the management of colorectal disease and issues that are generic to colorectal practice. Consequently, to avoid unnecessary duplication our contributors have covered important common issues such as bowel preparation, thromboembolic prophylaxis, audit, antibiotic policies, techniques of bowel anastomosis, enhanced recovery programmes, and the challenges of wound management. Risk factors are comprehensively reviewed as are issue surrounding Duty of Candour and obtaining informed consent in the post-Montgomery era. The role of nutritional support is also addressed even though the specific topic of intestinal failure management is covered elsewhere. Stoma management is also addressed in this section even though issues relating to stoma complications and construction in the emergency setting are discussed later. Since perineal wound healing is often compromised in cancer and inflammatory disease, this is covered in this section to avoid duplication in later sections. Throughout this section the risks and benefits of therapeutic measures are highlighted.

CHAPTER

1

Anatomy

Reza Mirnezami and Alex H. Mirnezami

INTRODUCTION

The famed eighteenth-century surgeon and anatomist John Hunter, a central figure in the history of surgery and anatomy, built his considerable surgical proficiency on an unrivalled knowledge of human anatomy. 'When Hunter cut, probed, sliced and sawed, he knew better than anyone else the exact whereabouts, the precise functions and the particular habits of every organ, muscle, blood vessel and tissue, healthy or diseased, that he was likely to encounter'.[1] To this day, surgeons are only capable of practicing the science and art of surgery, and extending their field, when appropriately armed with a thorough knowledge of anatomy, and the pre-eminence of anatomy, as immortalised by Lord Lister 'to intrude an untrained hand into such a divine mechanism as the human body is indeed a fearful responsibility', remains as relevant as ever.[2] Consequently, this chapter aims to provide a synopsis of the pertinent embryology and anatomy of the colon, rectum and anus.

EMBRYOLOGY OF THE COLON, RECTUM AND ANUS, AND DEVELOPMENTAL ANOMALIES

A detailed description of the precise and complex embryological derivations of the colon, rectum and anus is beyond the scope of the present text and can be found elsewhere in the literature.[3] However, a fundamental understanding of the embryology of the gastrointestinal (GI) tract and development with respect to the midgut and hindgut is helpful in the understanding of common developmental abnormalities and their anatomical consequences, and is therefore described in brief.

The Primitive Gastrointestinal Tract

The primitive gastrointestinal tract commences as an endodermal tube, from which all epithelial and glandular elements of the adult GI tract subsequently originate, whilst the intervening connective tissue, muscle, peritoneum and mesenteries derive from the mesoderm. Neural structures within the bowel originate from the embryological neural crest.

The endoderm tube is divided into three distinct areas: the foregut (with blood supply from the coeliac axis), the midgut (supplied by the superior mesenteric artery) and the hindgut (supplied by the inferior mesenteric artery). During an early stage of development of the gut tube, rapid proliferation of the endodermal cells leads to occlusion of the bowel lumen, which subsequently recanalises at a later stage of development by apoptosis of unessential cells in a delicate balancing act.

The Midgut

The caecum, appendix, ascending colon and the proximal two-thirds of the transverse colon are midgut derivatives. As the midgut increases in length during development, it forms a loop that is attached to the vitelline duct. This duct passes through the widely patent umbilicus and, in so doing, takes with itself a series of vitelline arteries from the aorta, which fuse to eventually form the superior mesenteric artery. The bowel loop at its caudal end develops a diverticulum; the upper part of this diverticulum expands to form the caecum, whilst the lower part remains rudimentary and matures into the vermiform appendix. Whilst in the umbilical cord, the midgut rotates around an axis formed by the superior mesenteric artery and the vitelline duct. Viewed from the anterior aspect, a counterclockwise rotation of 90° initially occurs, and later, as the gut returns to the abdominal cavity, a further

180° of midgut rotation follows, meaning a total counter-clockwise rotation of approximately 270°, which locates the normally positioned caecum and appendix in the right lower quadrant and the transverse colon in front of the superior mesenteric artery.

The Hindgut

The hindgut gives rise to the distal-most third of the transverse colon, the descending colon, sigmoid colon, rectum and the upper half of the anal canal. All of these hindgut derivatives receive their arterial blood supply via the inferior mesenteric artery. The distal-most portion of the hindgut terminates as a blind-ending endoderm-lined cavity called the cloaca. This lies in contact with a shallow ectodermal depression called the proctodeum. These apposing layers of endoderm and ectoderm are separated from one another by the cloacal membrane, which separates the cavity of the hindgut from the surface. The cloaca is further partitioned into ventral and dorsal compartments by the urorectal septum, the dorsal cloacal compartment forming the anorectal canal. The lining of the upper half of the anal canal is endodermally derived and that of the lower half is developed from the ectoderm of the proctodeum. The outer layers of the wall of the anal canal and the anal sphincters are developed from the surrounding splanchnic mesenchyme. The posterior aspect of the cloacal membrane breaks down, allowing the gut to open onto the surface of the embryo.

Denonvilliers reported his finding of a 'prostate-peritoneal membranous layer between the rectum and the seminal vesicles'.[4] Later, Cuneo and Veau suggested that this membrane was developed from the fusion of the embryonic peritoneum of the rectovesical cul-de-sac.[5] However, Wesson, in the 1920s, proposed that Denonvilliers' fascia was a condensation of the layers of the embryonic mesenchyme lying over the rectum and bladder.[6] The observation of Tobin and Benjamin[7] that Wesson's rectal mesenchymal layer developed into the fascia propria of the rectum supported Denonvilliers' theory that this layer was separate. This tissue is now considered part of Denonvilliers' fascia – an important distinction with Tobin and Benjamin labelling it as the posterior layer of Denonvilliers' fascia despite its different embryological origin. Finally, electron micrographs of the rectogenital septum show a dense double layer of elastin in the septum.[8]

Clinically, it is important to note that, whilst histologically Denonvilliers' fascia has two distinguishable layers that reflect its development, it is not possible to discern two separate layers during pelvic dissection and there is no 'posterior layer'. The term 'posterior layer' persists in the literature causing misconceptions and it is, in fact, the fascia propria of the rectum.[9] Any reports of dissection 'between the two layers of Denonvilliers' fascia' are, in reality, a dissection between the fascia propria of the rectum and the true Denonvilliers' fascia that lies over the prostate and seminal vesicles.

Developmental Anomalies

Defective midgut embryological development accounts for a variety of abnormalities of location and fixation of the colon (from aberrant rotation); intestinal atresias and stenoses (from imbalances in the relative amounts of cell proliferation and apoptosis in the endoderm tube) and persistence of vestigial structures (e.g. Meckel's diverticulum). With the exception of rotational abnormalities (which cover a wide spectrum, ranging from complete non-rotation or reversed rotation to varying degrees of malrotation), midgut anomalies tend to primarily interfere with small intestinal development and are therefore not covered in detail here. The vast majority of hindgut developmental anomalies are located in the anorectal region and result typically from failure of normal development of the urorectal septum.

THE COLON AND APPENDIX

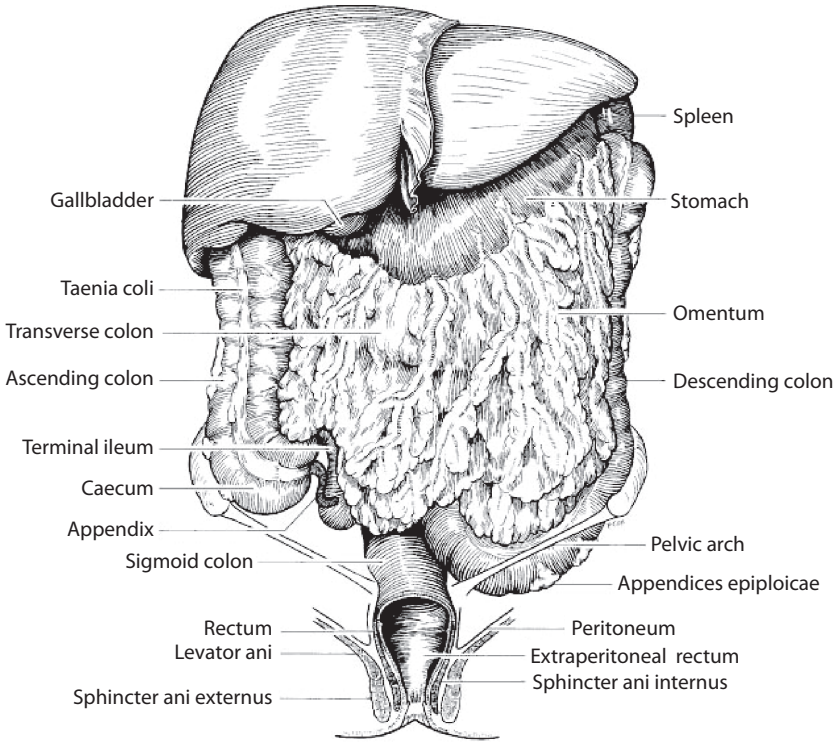
General Description

The large intestine extends from the terminal ileum to the anus and is subdivided for descriptive purposes into: the caecum and appendix, the ascending colon, hepatic flexure, transverse colon, splenic flexure, descending and sigmoid colon and the rectum and anal canal (Figure 1.1). The rectum and anal canal are addressed elsewhere in this chapter.

The large intestine varies considerably in length in different subjects and the caecum and sigmoid colon are particularly variable in size. On average, the colorectal tract is approximately 1.5 m in length. The colon and caecum, but not the appendix or rectum, are marked by the taeniae coli. These are three condensations of the outer, longitudinal, muscle coat of the bowel. Because the taeniae are shorter than the bowel to which they are attached, the colon adopts its typical sacculated shape, as may be noted in a plain X-ray of the abdomen as haustrations when the large bowel is distended. This is in contrast to the radiological appearance of a distended small bowel, which demonstrates complete transverse lines due to the transverse mucosal folds of the small intestinal known as the valvulae conniventes. The colon, but not the appendix, caecum or rectum, bears characteristic fatty peritoneal-covered tags referred to as appendices epiploicae.

Caecum

The caecum is the widest part of the large bowel and lies in the right iliac fossa just above the lateral half of the inguinal ligament on the iliacus muscle. It represents an outpouching of the large bowel below the level of the ileocaecal junction, where the terminal ileum enters the large intestine via the ileocaecal valve. The caecum is completely covered with visceral peritoneum and can be considerably mobile though it does not possess a mesentery. Its mobility puts it at risk of becoming involved by



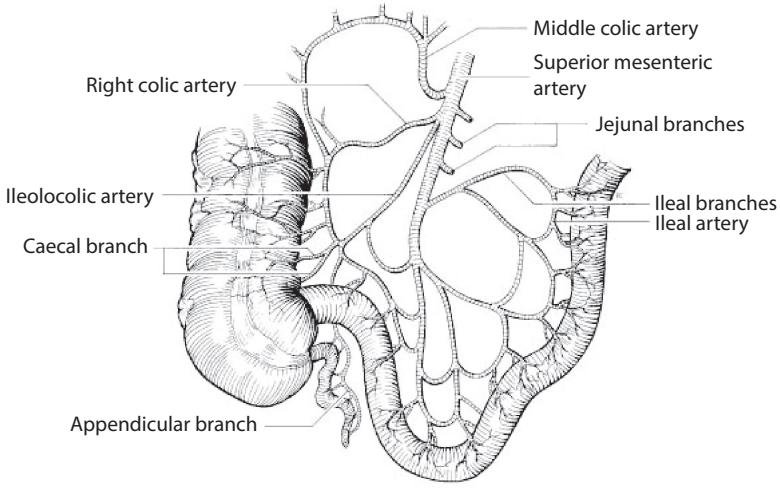
1.1 Schematic of the large intestine.

the volvulus. Attached to the postero-medial surface of the caecum is the appendix. The presence of peritoneal folds in the caecal vicinity creates three recesses that are of surgical importance – the retrocaecal, inferior ileocaecal and superior ileocaecal recesses. The longitudinal muscle of the caecal wall is restricted to three taeniae coli, which converge on the base of the appendix, providing for the latter a complete longitudinal muscle coat.

The ileocaecal valve is a rudimentary structure consisting of two horizontal folds of mucous membrane that project around the orifice of the terminal ileum. The caecum receives its arterial blood supply from the ileocolic artery via anterior and posterior caecal branches (Figure 1.2). Veins drain into corresponding branches of the superior mesenteric vein. Lymphatic drainage is to local paracolic nodes and ultimately to the superior mesenteric lymph node basin. Autonomic nerve supply is provided by the superior mesenteric plexus via sympathetic and parasympathetic nerve fibres.

Appendix

The appendix is a narrow muscular tube containing a large amount of lymphoid tissue that can vary markedly



1.2 The caecum and its arterial supply.

in length and position. The appendiceal base is attached to the posteromedial aspect of the caecum, but the tip is free and is subject to considerable positional variation as a result. In broad terms, the following positions are found at surgery: retro-caecal (~65%–70%), pelvic (25%–30%) and pre- or retro-ileal (~5%). This variability in anatomical location can lead to a variable clinical picture as a consequence of acute appendicitis. Consequently, patients with pelvic appendicitis or retrocaecal appendicitis may not develop right iliac fossa peritoneal irritation.

The appendix is completely covered with visceral peritoneum that is attached to the mesentery of the distal ileum by a short meso-appendix, within which resides the appendicular artery, a branch of the posterior caecal artery. A corresponding appendicular vein drains into the posterior caecal vein. Lymph drainage is via meso-appendix nodes and eventually into the superior mesenteric lymph node chain. The appendix is autonomically innervated via sympathetic and parasympathetic nerves from the superior mesenteric plexus. Visceral pain in appendicitis is triggered by the distension of the appendiceal lumen or a longitudinal muscle spasm. The afferent nerve fibres enter the spinal cord at the level of the tenth thoracic vertebra leading to a dull para-umbilical pain. Once the inflamed appendix irritates the parietal peritoneum, the pain migrates to the right iliac fossa, where it is felt as a precise localised pain.

As described earlier, the outer longitudinal layer of bowel muscle is completely formed around the appendix, but then diverges to form the taeniae coli. Tracing the taeniae on the caecum downward is thus a helpful manoeuvre in trying to identify the base of the appendix in complicated cases of acute appendicitis.

Ascending Colon

The ascending colon extends from the caecum to the inferior surface of the right hepatic lobe, where it turns sharply to the left at the hepatic flexure, becoming continuous with the transverse colon. The anterior aspect and sides of the ascending colon are covered with peritoneum. The lower part of the ascending colon lies on the iliopsoas muscle with the genital branch of the genitofemoral nerve. The upper part of the right colon lies on the quadratus lumborum muscle and the origin of the transversus abdominis. The hepatic flexure lies over the lower pole of the right kidney, medial to which are the second and third parts of the duodenum. The second or third part of the duodenum may be damaged during mobilisation of the hepatic flexure, particularly when resecting colonic Crohn's disease with an associated abscess or a bulky, locally advanced carcinoma.

The arterial blood supply is derived from the ileo-colic artery and right colic artery (which is present as a distinct artery arising from the superior mesenteric artery in approximately 20% of individuals). At the hepatic flexure, numerous veins lie immediately underneath the peritoneum, and these may have to be cauterised when dividing the peritoneum during the mobilisation of the hepatic flexure. These veins enlarge considerably in portal hypertension. Lymphatic drainage is via the superior mesenteric lymphatic drainage basin, and the sympathetic and parasympathetic nerve supply arises via the superior mesenteric plexus.

Transverse Colon

The transverse colon is completely peritonealised. It begins at the hepatic flexure and hangs downward suspended by the transverse mesocolon from the antero-inferior surface of the pancreas. It terminates at the splenic flexure in the left upper quadrant, becoming continuous with the descending colon. The splenic flexure lies higher than the hepatic flexure and is suspended from the diaphragm by the phrenico-colic ligament. The greater omentum, arising from the greater curvature of the stomach, covers the transverse colon. The inferior peritoneal coat of the omentum is adherent to the anterior surface of the transverse colon and the transverse mesocolon containing the middle colic vessels and lymphatics. These peritoneal layers can be divided so that the transverse colon and mesocolon can be freed from the omentum. The proximal two-thirds of the transverse colon are supplied by the superior mesenteric artery via the middle colic arterial arcades. The distal third is supplied by the left ascending colic branch of the inferior mesenteric artery. Veins drain correspondingly into the superior and inferior mesenteric veins (the latter draining into the splenic vein). Nerve supply to the proximal two-thirds of the transverse colon is via sympathetic and vagal nerves via the superior mesenteric plexus. The distal third is innervated by sympathetic and parasympathetic pelvic splanchnic nerves via the inferior mesenteric plexus.

Descending Colon

The descending colon commences at the splenic flexure and down towards the pelvic brim where it becomes continuous with the sigmoid colon. The descending colon is attached to the posterior abdominal wall. It lies on the transverse abdominis, quadratus lumborum and the iliopsoas muscle. The anterior aspect and sides of the descending colon are peritonealised, thereby making it retroperitoneal. The left colic and sigmoid branches of the inferior mesenteric artery provide blood supply, with corresponding venous drainage into the interior mesenteric vein.

Sigmoid Colon

The sigmoid colon commences as a direct continuation of the descending colon in front of the pelvic brim. It is completely peritonealised, and below it becomes continuous with the rectum near the pelvic brim. The sigmoid colon is relatively mobile and is attached by the sigmoid mesocolon to the posterior abdomino-pelvic wall. The sigmoid mesocolon is V-shaped, running upward and medially over the psoas muscle, the genital vessels and the ureter to the aortic bifurcation. The sigmoid is subject to considerable variation in length, and its mobility predisposes it to the risk of rotation about its mesentery,

known as the sigmoid volvulus. Although this can autocorrect, the rotation can continue leading to a compromise to the arterial inflow and venous drainage and large bowel obstruction. Sigmoidal branches of the inferior mesenteric artery provide arterial blood supply with corresponding venous drainage.

Specific Vascular Anatomical Notes

The marginal artery (also known as the marginal artery of Drummond) provides an anastomotic channel between the superior and inferior mesenteric arterial arcades (Figures 1.3 and 1.4). The marginal artery is almost always present and runs in the colonic mesentery close to the bowel wall. Riolan's arcade, also known as the meandering mesenteric artery, is another vascular arcade that connects the proximal middle colic artery and left ascending colic arterial channels. This runs more inferiorly close to the mesenteric root.

The inferior mesenteric vein (IMV) is a continuation of the superior rectal vein. This does not follow the artery, however, instead passing to the left of the duodeno-jejunal flexure (where it may be located during surgery), before passing beneath the pancreas to drain into the splenic vein. In a total mesorectal resection with ultra-low anastomosis, high vascular ligation of the IMV above its last tributary and close to the inferior border of the pancreas is an important step for ensuring full mobilisation of the neo-rectum to reach the low pelvis.

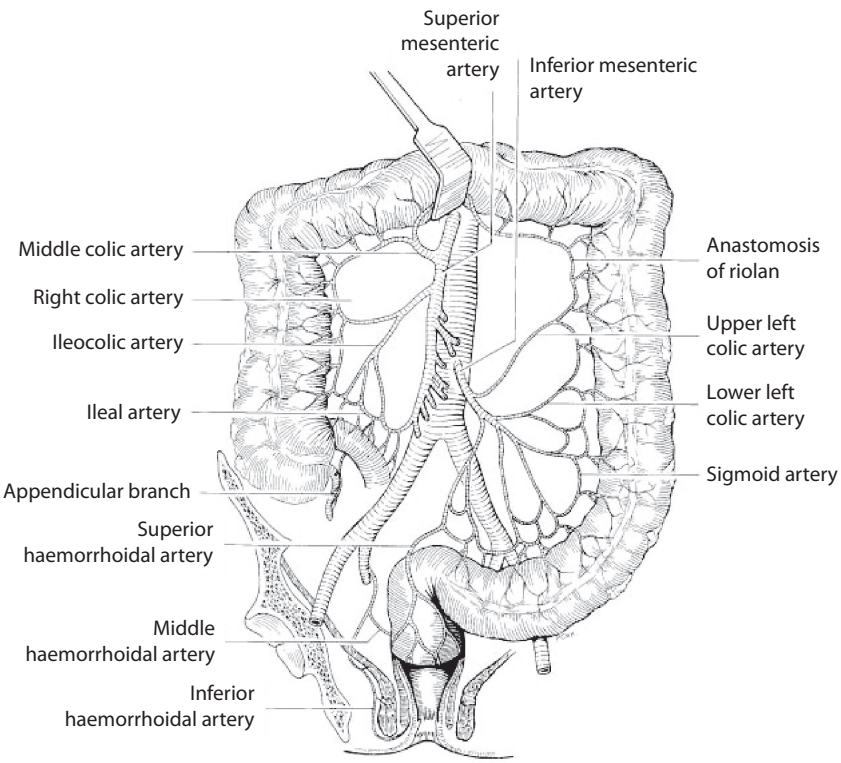
Specific Innervation Notes

The sympathetic supply to the right side of the colon is derived from connector cells in the lower six thoracic segments of the spinal cord, from which preganglionic white fibres pass out to the paravertebral sympathetic chain (Figure 1.5). This chain lies behind the inferior vena cava on the right and to the side of the aorta on the left. The parasympathetic nerve supply to the right colon is probably from the posterior vagus, the fibres of which join the superior mesenteric plexus. The outflow for the left colon takes its origin from the first three lumbar segments of the cord. Fibres leave the ganglionated sympathetic trunks to form a plexus around the superior and inferior mesenteric arteries. It is only in the perivascular ganglia that synapses form, from which nerves follow the arteries to supply the gut.

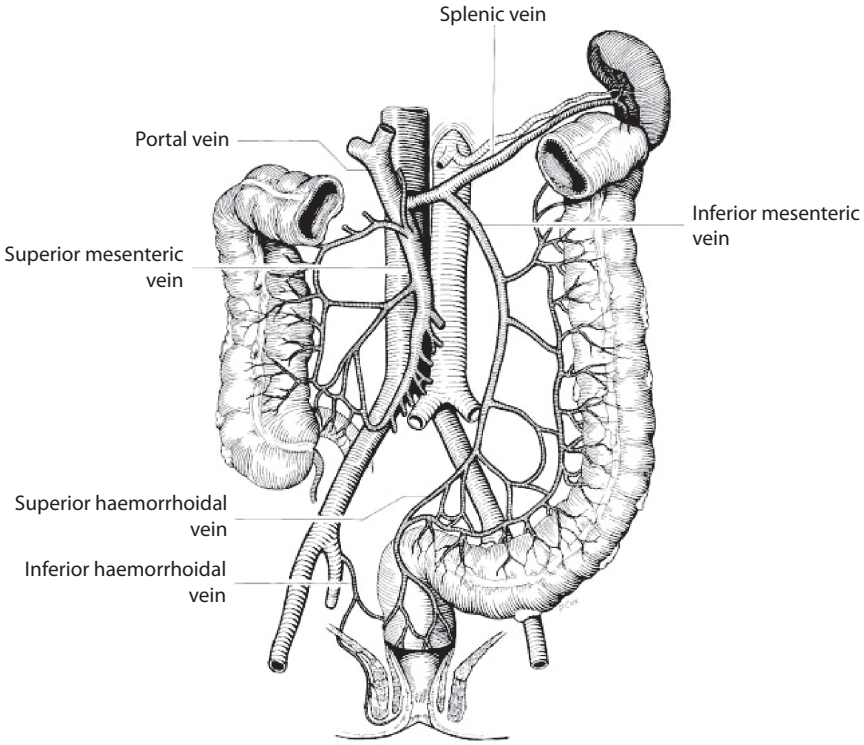
THE RECTUM

General Description

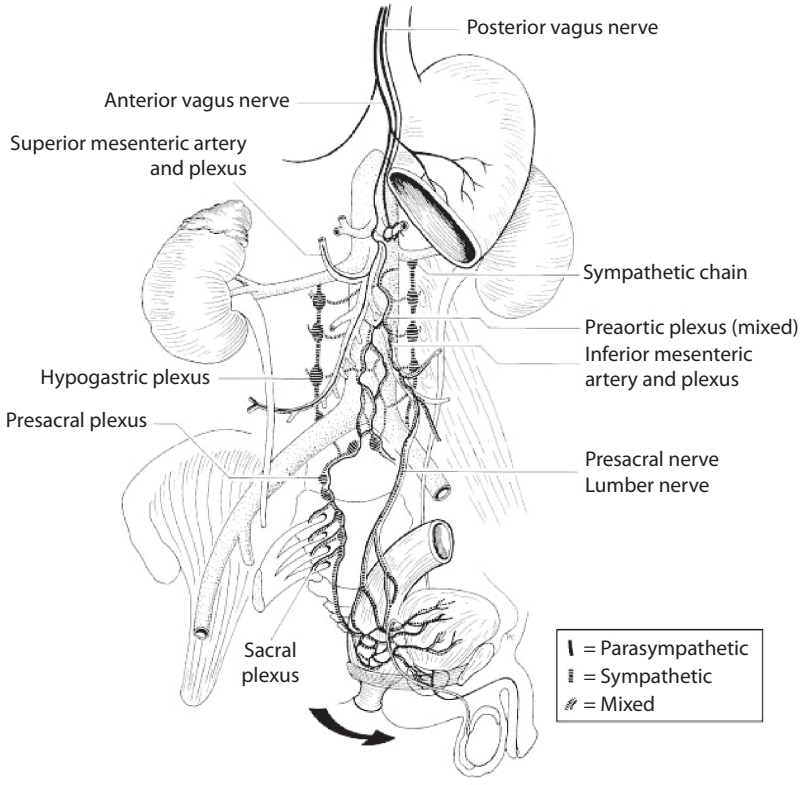
The rectum commences as a direct continuation of the sigmoid colon, and it has been variably defined in the literature. It is entirely intra-pelvic and expressed at (Figures 1.6 and 1.7) times based on its length as being either 12 cm,[10,11] 15 cm[12,13] or 16 cm[14] from the anal verge. In the UK, the rectum is typically described as being 15 cm in length from the anal verge based on endoscopic examinations, and radiologically defined as commencing at the level of the



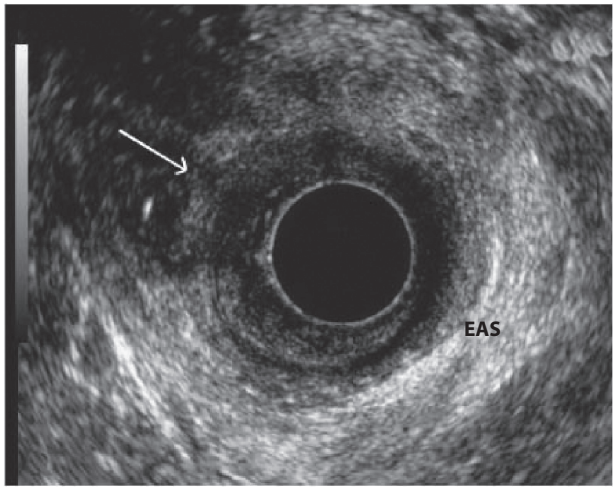
1.3 Arterial supply of the colorectum.



1.4 Venous drainage of the colorectum.



1.5 Autonomic nerve supply of the colorectum.



1.6 Endoanal ultrasound of the anal sphincter complex (axial plane). The arrow points to a lateral sphincter defect. EAS: external anal sphincter.

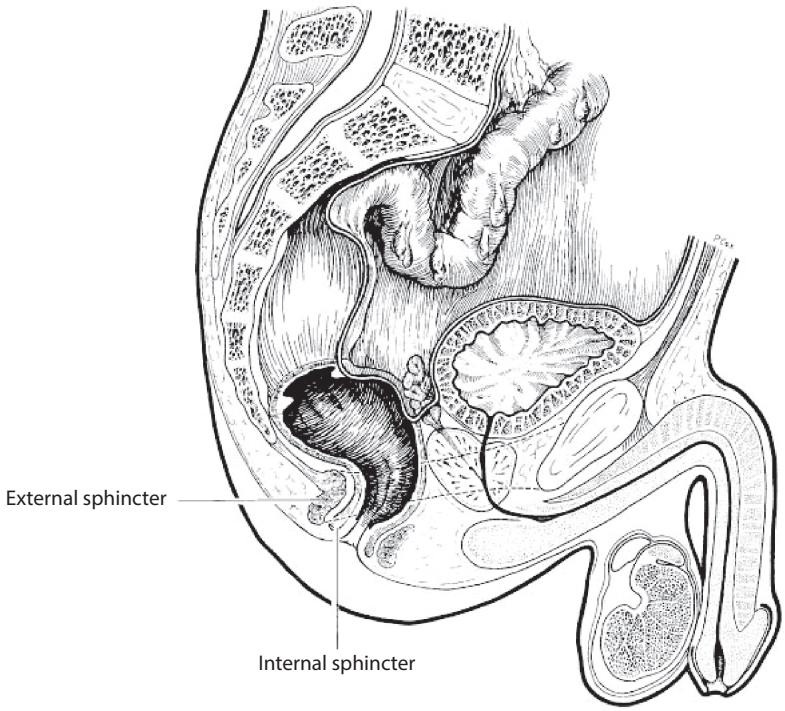
third sacral segment and operatively defined by the confluence of the taeniae in the pelvis with a corresponding absence of appendices epiploicae or a true mesentery.

The longitudinal orientation of the rectum conforms to the ventral concavity of the sacrum and coccyx. Thus, the rectum passes downward and backward initially, and then downward and forward to reach the pelvic diaphragm. This natural ventral bend in the rectum is termed the sacral flexure. At the pelvic diaphragm, the rectum becomes continuous with the anal canal. In addition to ventral curvature, the rectum possesses a series of three laterally orientated curves. The upper and lower curves are directed to the right and the middle curve to the left. Corresponding to each curve there is, on the luminal aspect of the rectal tube, a transverse, sickle-shaped fold. These folds are referred to as rectal shelves, or the valves of Houston, and are produced by the condensed muscle of the rectal wall projecting inwards covered with overlying mucosa. The middle rectal shelf is the most prominent and is readily visualised during rigid and flexible sigmoidoscopy.

The rectum is further divided from a surgical perspective into three portions: upper third, middle third and lower third. With respect to peritoneal coverings, the upper third of the rectum is covered by peritoneum on its anterior and lateral surfaces, and the middle third is covered by peritoneum only on its anterior surface. The lower third of the rectum lies below the peritoneal reflexion and thus possesses no peritoneal covering on any aspect.

Arterial Supply and Venous Drainage

The principal artery supplying the rectum is the superior rectal artery (the name given to the inferior mesenteric artery at the point where the latter crosses the pelvic brim to enter the pelvic cavity). The superior rectal artery runs with the pelvic attachment of the sigmoid mesocolon to enter the perirectal fat behind the rectum. Here it breaks into two, sometimes three longitudinal vessels that travel on either side of the rectum before sinking into the rectal wall. Supplementary arteries that make a contribution to the blood supply of the rectum are the middle rectal arteries and the inferior rectal artery.



1.7 Schematic of the rectum.

The right and left middle rectal arteries arise from the corresponding internal iliac artery and run inferomedially just above the pelvic floor to reach the rectum (Figure 1.3). The middle rectal arteries are inconstant in size both between different individuals and from side to side in the same individual. They are usually not prominent vessels but can be large enough to require separate ligation at times. They may be absent on one or both sides. Each inferior rectal artery (also known as the inferior haemorrhoidal artery) is a branch of the internal pudendal artery and is given off as soon as the latter enters the perineum. The inferior rectal artery crosses the ischioanal fossa (ischio-rectal) from lateral to medial to enter the anal wall. It is the principal artery of the anal canal. However, through the anal wall it is capable of supplying the distal third of the rectum.

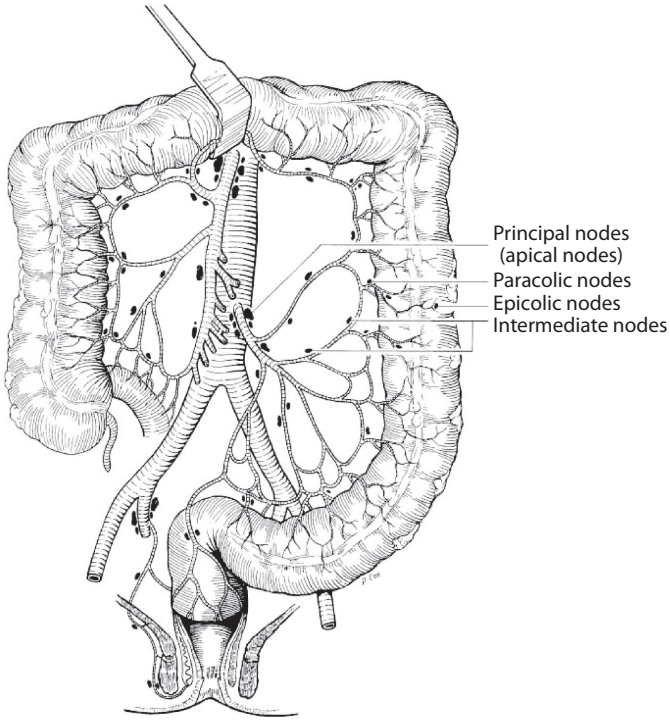
The venous drainage of the rectum mirrors the arterial supply. From a rich and valveless intramural venous plexus, blood enters the perirectal venous plexus, from where rectal blood is carried mainly in the superior rectal vein. The superior rectal vein, running alongside the artery, crosses the pelvic brim from below upward to become the inferior mesenteric vein. Thereafter, the inferior mesenteric vein drains the sigmoid, descending colon and splenic flexure before emptying into the splenic vein and thereby into the portal vein. Some venous blood from the intramural and perirectal venous plexuses travels bilaterally in the middle rectal veins and drains into the internal iliac veins. These veins are usually multiple and small, but can occasionally be large and require ligation. Venous blood from these rectal plexuses also finds its way through the anal wall into the inferior rectal veins which drain into the internal iliac veins via the internal pudendal veins. The anal mucosa and submucosa thus represent sites of natural porta-systemic venous anastomoses. To a limited extent, these anastomoses are also present in the rectal wall. Rarely they can give rise to life-threatening rectal bleeding in patients with portal hypertension.

Lymphatic Drainage

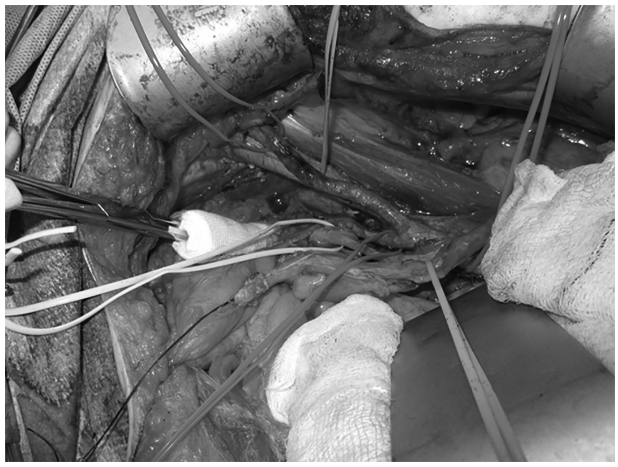
As with the lymphatic drainage of the rest of the colon, the rectal lymph is initially received by the lymphoid follicles in the mucosa (Figure 1.8). Thereafter, the lymph passes successively through three tiers of mesorectal lymph nodes (equivalent to epicolic, paracolic and intermediate nodes) before reaching the so-called principal nodes. The principal lymph nodes that receive most of the lymph from the upper two-thirds of the rectum are the mesorectal nodes and inferior mesenteric lymph nodes that are situated along the path of the inferior mesenteric artery.

Although a point of controversy in the past, there is an increasing recognition that lymph from the lower third of the rectum may also drain into two additional sets of lymph nodes: the internal iliac lymph nodes bilaterally (also called the pelvic side wall nodes) and inguinal nodes (similar to drainage of the anal canal).

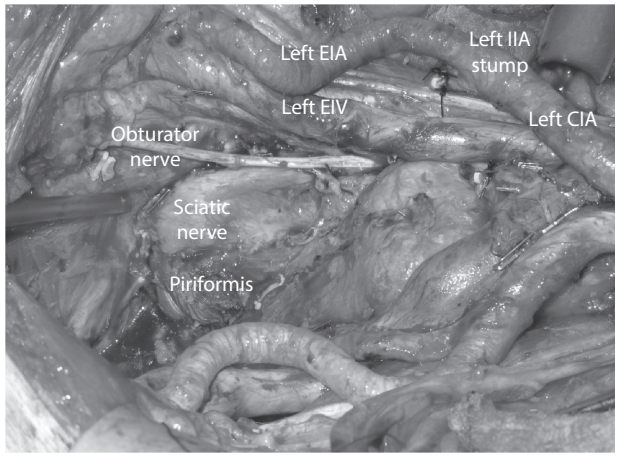
For the purposes of the TNM (tumour, node, metastasis) cancer classification, involved *mesorectal* lymph nodes are expressed as N1 when between one and three peri-colic or peri-rectal nodes contain cancer cells, or N2, when four or more nodes are involved. At present, the involved pelvic sidewall and inguinal nodes are classified (perhaps erroneously) as not representing regional nodal spread, and hence involvement of these is recorded as representing M1 disease.[12] The precise management of



1.8 Lymphatic drainage of the colorectum.



1.9 Pelvic sidewall lymphadenectomy without resection of the internal iliac system.

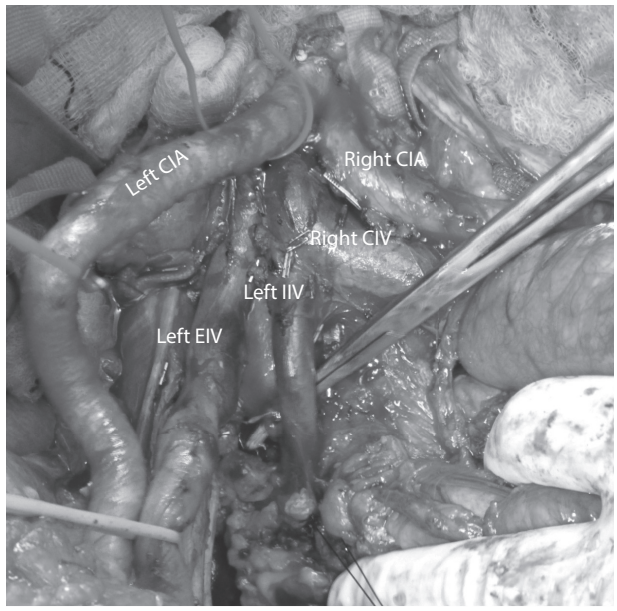


1.10 Pelvic sidewall lymphadenectomy with resection of the internal iliac system.

predicted or confirmed inguinal and pelvic sidewall nodes in rectal cancer is, at present, the subject of significant debate (Figures 1.9 and 1.10). If resection is performed in this area, one needs to be wary of aberrant venous anatomy. Careful dissection and clarification of the relevant anatomy before ligation of major vessels is important in these circumstances (Figure 1.11).

Nerve Supply

Preganglionic fibres from the sympathetic trunk converge over the sacral promontory at the bifurcation of the aorta to form the hypogastric plexus. Fibres leave the hypogastric plexus together with the pelvic parasympathetics to form the presacral nerves. The presacral nerves are easily visible during rectal dissection: they lie behind and below the pelvic peritoneum running from below the aortic bifurcation laterally to the side wall of the pelvis. They therefore lie behind the mesorectum and the superior haemorrhoidal veins. The presacral nerves are closely associated with the middle haemorrhoidal arteries at their



1.11 Aberrant venous anatomy. The left internal iliac vein (left IIV) originates from the right common iliac vein (right CIV). Right and left CIA (right and left common iliac artery), left EIV (left external iliac vein).

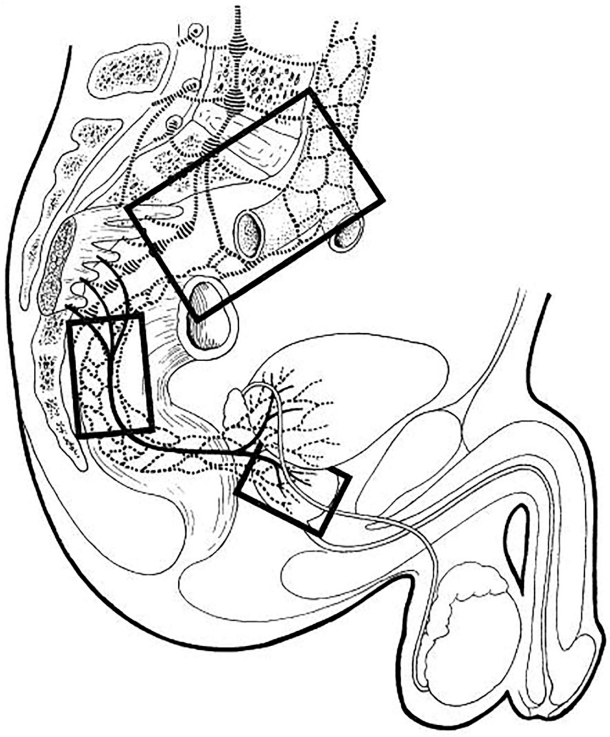
origin from the internal iliac artery. Fibres from the hypogastric plexus travel with the arteries to supply the urethra, prostate, seminal vesicles, penis, vagina and base of the bladder as well as the rectum and anal canal.

The parasympathetic supply to the left colon, rectum and anal canal, as well as the pelvic viscera, is from the second, third and fourth sacral nerve roots as they emerge on the piriformis from the sacral foramina. The parasympathetic fibres continue laterally as the nervi erigentes to join the presacral nerves, which, with the sympathetics, supply the genital organs, bladder and anorectum. A few fibres join the hypogastric plexus and run over the aortic bifurcation to accompany the inferior mesenteric plexus supplying the sigmoid and descending colon.

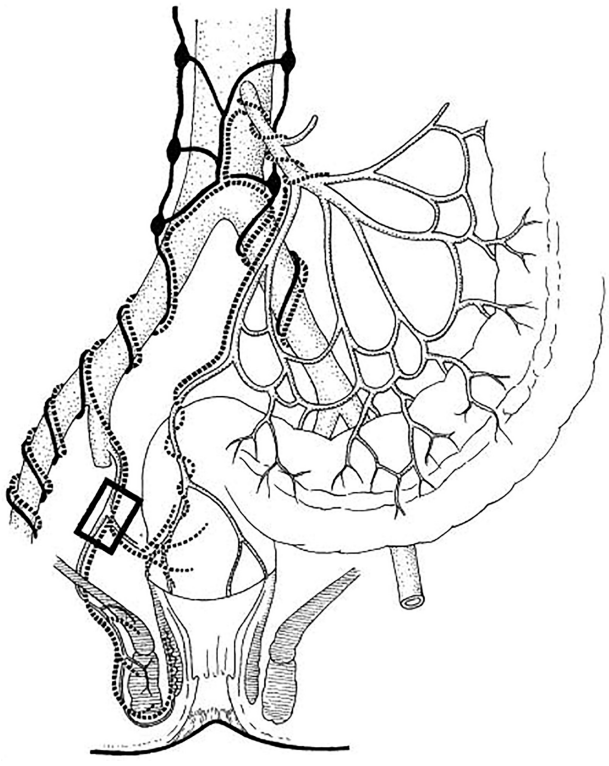
The nervi erigentes lie behind Waldeyer's fascia before they join the presacral nerves and may be damaged by stripping the fascia off the sacrum. Damage to the presacral nerves may also occur during lateral dissection of the rectum or during clearance of the internal iliac artery for malignant disease. Some fibres may also be damaged in front of the aortic bifurcation (Figures 1.12 and 1.13).

The Mesorectum, the TME Concept and Surgical Anatomy

The rectum is surrounded by a layer of perirectal fat, which is generally more abundant posteriorly and has a characteristic bi-lobed appearance when resected in the correct plane. These two 'lobes' are a result of the paired concavities crated by the levator muscles. Within this perirectal fat lie the perirectal lymph nodes and the superior rectal vessels. This perirectal fat is in turn surrounded by a distinct circumferential fascial layer called the fascia propria of the rectum. Collectively this envelope



1.12 Autonomic nerve supply to the pelvis and rectum with sites of common nerve injury at surgery. Sagittal view.



1.13 Autonomic nerve supply to the pelvis and rectum with sites of common nerve injury at surgery. Coronal view.

of tissue is known as the mesorectum. The technique of total mesorectal excision (TME), popularised by Heald, refers to the precise dissection of the rectum and accompanying lympho-vasculature to deliver an oncologically superior specimen, within an intact mesorectal envelope.

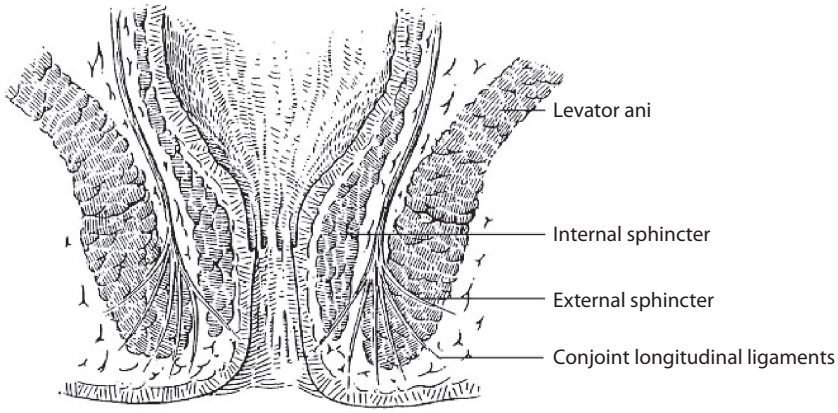
Posteriorly, the TME plane (also known as the 'holy plane')[15] lies between the visceral fascia surrounding the mesorectum and the parietal (presacral) fascia. In TME surgery, gentle positioning of laparoscopic or open retractors at the level of the pelvic brim will expose the layer of loose areolar tissue, or angel hair, which marks the holy plane and serves as a useful guide to surgical dissection. In dissecting in this plane, and developing the posterior mesorectal dissection, one must be mindful of the autonomic hypogastric nerves and plexus which lie just on top of the aortic bifurcation and sacral promontory. Careful dissection in the correct plane will avoid these, and the wishbone formed by the bilateral nerves diverging from the plexus is usually identifiable. Compromise of these nerves would impair sexual function.[15]

Anteriorly, the fascia separating the rectum from the male or female urological/gynaecological organs becomes condensed to form Denonvilliers' fascia, whilst posteriorly the condensed fascia covering the sacrum is eponymously named as Waldeyer's fascia. Heald and Moran[16] have described a fusion of these two fascial layers below the level of the fourth sacral vertebra, where they condense to form the rectosacral ligament.

THE ANAL CANAL AND PELVIC FLOOR

The anal canal is the most terminal part of the lower gastrointestinal tract and is completely extra-peritoneal. It is approximately 4 cm long, commencing above at the level where the rectum passes through the pelvic diaphragm and terminating below at the anal verge. The pelvic diaphragm is comprised of the levator ani muscles and the small coccygeus muscles, together with their investing fascia. Anteriorly, the diaphragm is incomplete to permit passage of the urethra and the urethra and vagina, in males and females, respectively. The muscular junction between the rectum and the anal canal is palpable on digital rectal examination as the anorectal ring which is formed by the union of puborectalis fibres from the two levator ani muscles, the deep portion of the external anal sphincter and the highest fibres of the internal anal sphincter (Figure 1.14).

The mucous membrane of the upper half of the anal canal is derived from hindgut endoderm and is lined by columnar epithelium which is innervated by autonomic hypogastric plexus derived nerve fibres (as with the rectal mucosa) and is thus sensitive to stretch only. The anal mucous membrane of the upper anal canal is arranged into a series of 8–12 longitudinal folds referred to as the anal columns (or the columns of Morgagni). These columns are linked at their lower ends by a series of

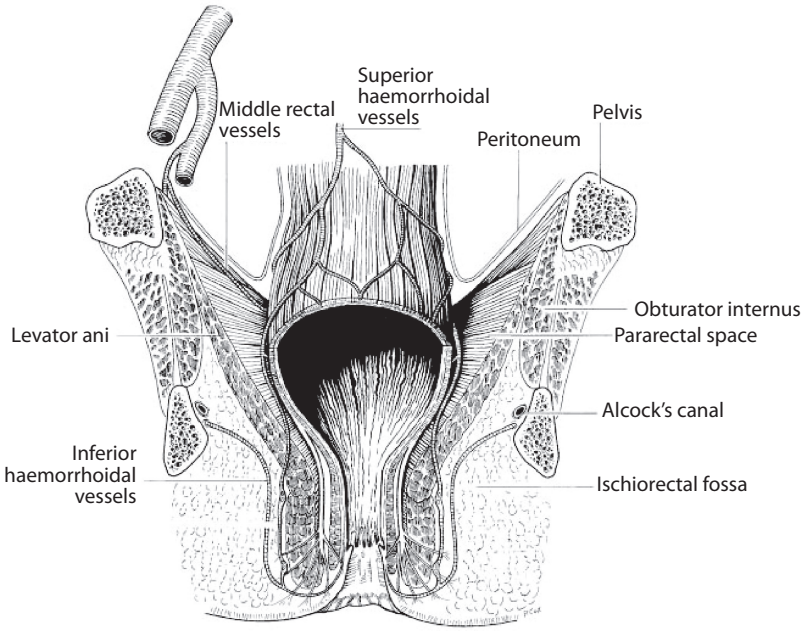


1.14 Anal sphincter complex.

semilunar folds called anal valves (or the anal valves of Ball). These valves lie along and indeed constitute the waviness of the dentate (or pectinate) line that represents the junction between the upper and lower halves of the anal canal. Immediately above the level of the dentate line, the mucous membrane has a plum-coloured appearance, whilst immediately below there is a transition to parchment-coloured stratified squamous epithelium which is innervated by the somatic inferior rectal nerve and is accordingly sensitive to pain, temperature, touch and pressure. At the lower limits of the anal columns are situated the anal sinuses, or crypts, into several of which (mostly those located posteriorly) open the anal glands. There are thought to be 4–8 of these glands in the normal anal canal. Each has a direct opening into the apex of an anal crypt and occasionally two glands open into the same crypt. Infection of the anal glands is believed to be the initiating event in the development of perianal abscess and fistula-in-ano.

Arterial Supply and Venous Drainage

The superior rectal artery descends into the pelvis as a direct continuation of the inferior mesenteric artery within the sigmoid colon mesentery and divides into right and left branches typically at the level of the third sacral vertebra (Figure 1.15). These in turn give off branches that pierce the muscular coat of the bowel down to the level of the internal anal sphincter, where they communicate with

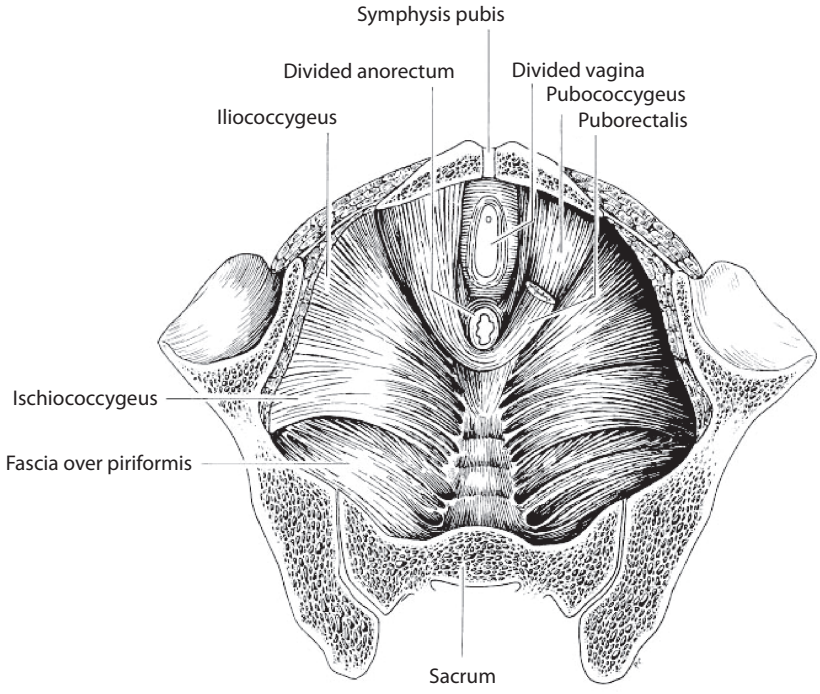


1.15 Anatomy of the anal canal and its vascular supply.

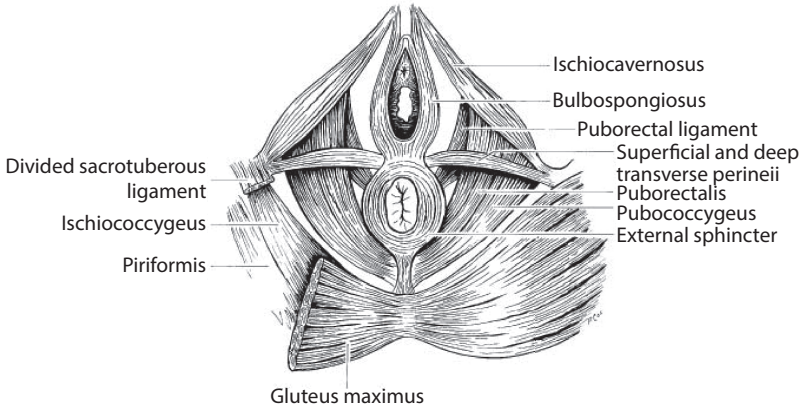
branches of the middle (arising from the internal iliac artery) and inferior (arising from the internal pudendal artery) rectal arteries. Because of its hindgut origin, the upper half of the anal canal is supplied primarily by branches of the superior rectal artery. The arterial supply of the internal and external anal sphincter complexes, as well as the lower half of the anal canal, is derived from the inferior rectal artery. The middle rectal artery is generally believed to make a more modest contribution to anorectal blood supply. The venous drainage of the anal canal corresponds to the main arterial tributaries and originates in the venous plexus situated in the anal wall. Proximal to the dentate line, venous drainage occurs mainly into the superior rectal vein and thereby eventually into the portal venous system. Distal to the dentate line, venous drainage is mainly to the internal iliac veins either directly via the middle rectal veins or indirectly via the inferior rectal veins and internal pudendal veins. In addition to the intramural venous plexuses in the anal wall, there are mucosal cushions situated in the upper half of the anal canal. These cushions are thought to aid the internal and external anal sphincters in effecting tight closure of the anal canal and make a modest contribution to resting anal tone. Haemorrhoids represent varicose dilatations of these cushions and of the venous plexuses within them. Initially residing within the anal canal (1st degree), they may enlarge and prolapse temporarily (2nd degree) or more permanently (3rd degree).

Lymphatic Drainage of the Rectum and Anus

The dentate line marks a watershed between two different lymphatic drainage basins for the upper and lower anal



1.16 Anatomy of the muscles of the pelvic floor.



1.17 Schematic depicting the muscles of the anterior and posterior perineum.

canal. Proximal to the dentate line, anal canal lymph drains principally into the internal iliac lymph nodes bilaterally, and to a more limited extent to the para-aortic and inferior mesenteric lymph node basins. Distal to the dentate line, anal canal lymph drains primarily into the superficial inguinal lymph nodes chains bilaterally. Thus, lymphatic dissemination of squamous cell carcinoma of the anus usually results in inguinal, and not abdominal, lymphadenopathy.

Anal Canal Musculature

Anal continence is essentially dependent upon four structures: the internal anal sphincter, the external anal sphincter, the puborectalis sling (which is derived from the levator ani muscles on either side) and the anal cushions. The internal anal sphincter is responsible for approximately 70% of resting anal tone, whilst the external anal sphincter and puborectalis collectively contribute to about 20%. The remainder is thought to be provided by the arteriovenous anal mucosal cushions (Figure 1.15).

The internal anal sphincter is of variable length in health and disease and between men and women, but typically may be 3–4 cm. It constitutes the distal extension of the inner circular smooth muscle layer of the rectal wall and is composed of smooth (involuntary) muscle and has an intrinsic nerve supply via the myenteric plexus as well as additional supply from the autonomic nervous system. Sympathetic nervous activity and parasympathetic activity are believed to enhance and reduce internal anal sphincter contractility, respectively. The distal edge of the internal sphincter is well-demarcated and in the normal individual is usually easily palpable. In cases of fissure-in-ano, after failure of more conservative measures, one option is to conduct a partial, right lateral, internal anal sphincterotomy.

The external anal sphincter is conventionally described as a tripartite structure with a subcutaneous part, a superficial part and a deep part. The external sphincter is longer and wider than the internal sphincter, and its lower-most edge is normally seen to lie distal to that of the internal sphincter by at least 1 cm. Between these two edges, the intersphincteric groove is palpable, and in proctectomy for benign pathology can be a plane of dissection. The external anal sphincter is composed of striated (voluntary) muscle and is somatically innervated by the right and left inferior rectal nerves, each derived directly from the corresponding pudendal nerve (S2–S4). The puborectalis sling comprises those fibres of each levator ani muscle that arise from the periosteum on the posterior surface of the pubic bone a centimetre, or more, lateral to the pubic symphysis. These fibres run posteriorly and swing medially behind the recto-anal junction to meet their counterparts from the other side. Together these fibres form a sling behind the recto-anal junction which accounts for the recto-anal angle (Figures 1.16 and 1.17). Voluntary relaxation of the puborectalis sling allows straightening of the recto-anal tube, a prerequisite to defaecation. The puborectalis muscle is innervated like the rest of the levator ani by the ipsilateral perineal branch of S4, a branch of the lumbo-sacral plexus. The deepest part of the external anal sphincter blends with the puborectalis sling behind the recto-anal junction. This area of fusion is palpable as the anorectal ring on digital rectal examination.

IMPORTANT ANATOMICAL CONSIDERATIONS

Surgical Planning and Imaging Investigations

Careful planning and anatomical understanding, especially in surgical oncology, through the use of detailed imaging and accurate staging are a cornerstone of modern surgical practice. In this way the surgeon may better appreciate the extent of any disease process, and plan their surgical approach and the treatment required more accurately.

In the abdomen, imaging through high-resolution, thin-section, contrast-enhanced computed tomography (CT) gives good detail of most intra-abdominal structures, and may be supplemented with Magnetic Resonance Imaging (MRI) scanning for retroperitoneal structures.

In the pelvis, MRI is superior to CT for assessment of the pelvic anatomy and relationships of viscera and fascial planes (imaging is covered in detail in Chapter 29).

An appreciation of the anatomy allows for an understanding as to the sites of risk of damage to the pelvic nerves during rectal excision. There are four key zones (see Figures 1.12 and 1.13):

1. *Origin of the inferior mesenteric artery*
   The purely sympathetic hypogastric nerves are at risk during ligation of the inferior mesenteric artery when taken flush with the aorta.
2. *Posterior dissection*
   The nerves lie just outside the plane of dissection which should be carried out in the loose areolar tissue just outside the fascia propria of the rectum. The damage is purely sympathetic at this level.
3. *Lateral dissection*
   The pelvic plexus may be damaged if the rectal dissection strays too far laterally away from the mesorectal plane, especially if excessive traction is placed on the rectum, thereby pulling the nerves both superiorly and medially. The resulting damage is both sympathetic and parasympathetic.
4. *Anterior dissection*
   The space between the rectum and the prostate/seminal vesicles is very narrow. The cavernous nerves are at risk at this level, and this is likely where most damage to the parasympathetic nerves occurs and why most cases of post-operative impotence occur after deep pelvic dissection.

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CHAPTER

2

Physiology

Anwen Williams and Martyn D. Evans

COLONIC FUNCTION – ABSORPTION, STORAGE AND MOTILITY

Absorption

Absorption and digestion of nutrients primarily take place in the small intestine. However, the colon still plays a major role in the absorption of some nutrients and in the regulation of water and electrolyte balance. In healthy individuals, the colon receives 1.5–2 L of isotonic chyme daily. This contains water, electrolytes and organic residues from diet and salivary secretions, yet only 200 g of solid faeces per day is produced.[1,2]

The proximal colon processes some complex carbohydrates and proteins resistant to digestion and absorption in the small intestine. Nutrients are salvaged from these products by fermentation by over 400 species of bacteria, predominately obligate anaerobes. Fermentation primarily occurs in the ascending and proximal transverse colon and results in the production of short chain fatty acids; acetate (60%), propionate (25%) and butyrate (15%).[3] More than 95% of short chain fatty acids (SCFAs) are created and absorbed in the colon, providing 5%–15% of human calorific daily needs. They are incorporated as a basic element for lipogenesis, gluconeogenesis, mucin and protein production. The SCFA butyrate is important for colonic homeostasis, being the primary energy source of colonocytes, supplying 70%–90% of its energy requirements. It also promotes absorption of water, sodium and chloride from the colon. The bacterial fermentation of undigested proteins also generates ammonia, phenols, indoles and sulphurs, with some of these proteolytic metabolites becoming a nitrogen source for bacterial growth. These remain dissociated in the colonic lumen until absorbed in exchange for bicarbonate or by diffusion in their lipid soluble form.

The colon regulates water and electrolyte transport and preserves intestinal homeostasis by absorption and secretion via surface epithelial cells and crypt cells. The approximate mucosal surface area of the colon is 2000 square cm.[2,4] Water is passively absorbed along an osmotic gradient determined by a luminal sodium concentration which is lower than that of epithelial cells. The absorption of water therefore relies upon sodium conservation. Over 90% of water is reabsorbed in the colon primarily by paracellular absorption and to a lesser extent a transcellular route. The right colon has the greatest absorptive capability where chyme resides for a longer period, thus maximising mucosal contact. As a result, patients who undergo right hemicolectomy have more symptoms of diarrhoea than those who have a left-sided resection. Fluid absorption in the colon is promoted by antidiuretic hormone. Fluid secretion in the colon occurs only in the presence of laxatives, endotoxins, bile acids and hormones, e.g. VIP.

The colon absorbs sodium and chloride via several mechanisms: Na^+/H^+ exchange channels in the proximal colon and Na^+-specific channels in distal colon and rectum, both of which are coupled to $Cl^-HCO_3^-$ exchange channels. These transport channels allow the passive diffusion of sodium into epithelial cells along an electrochemical gradient (low intracellular sodium) and a negative intracellular electrical potential difference, resulting in absorption of Na^+ via Na^+/K^+ ATPase pump on basolateral membrane of epithelial cells. Aldosterone, somatostatin, $\alpha 2$ adrenergic agents and SCFAs also enhance fluid and sodium absorption. In the proximal colon, Cl– is exchanged for HCO_3^- linked to Na^+/K^+ exchange. Potassium transport is passive, following Na^+ across cell membranes although the H^+/K^+ ATPase channel actively absorbs K^+ into epithelial cells of distal colon and rectum. Colonic microbiota metabolise urea into ammonia which is then absorbed by epithelial cells into the enterohepatic circulation to the liver where it is broken down to urea.

Storage and Motility

The colon relies on contractions to propel luminal content distally from the caecum to the rectum, promoting absorption and the mixing of colonic content.

Phasic single non-propagating contractions occur frequently in both shorter and longer segments of the colon, whereas phasic mass contractions are high amplitude propagating contractions that occur infrequently during the daytime or after meals (gastrocolic reflex). They originate in the caecum, propelling luminal content distally to the rectum. Tonic contractions are less well defined and are longer lasting over several minutes. They are not associated with a change in luminal pressure but rather a change in colonic tone, and are described as tetanic (fused phasic contractions) or specific (chemical contractions).

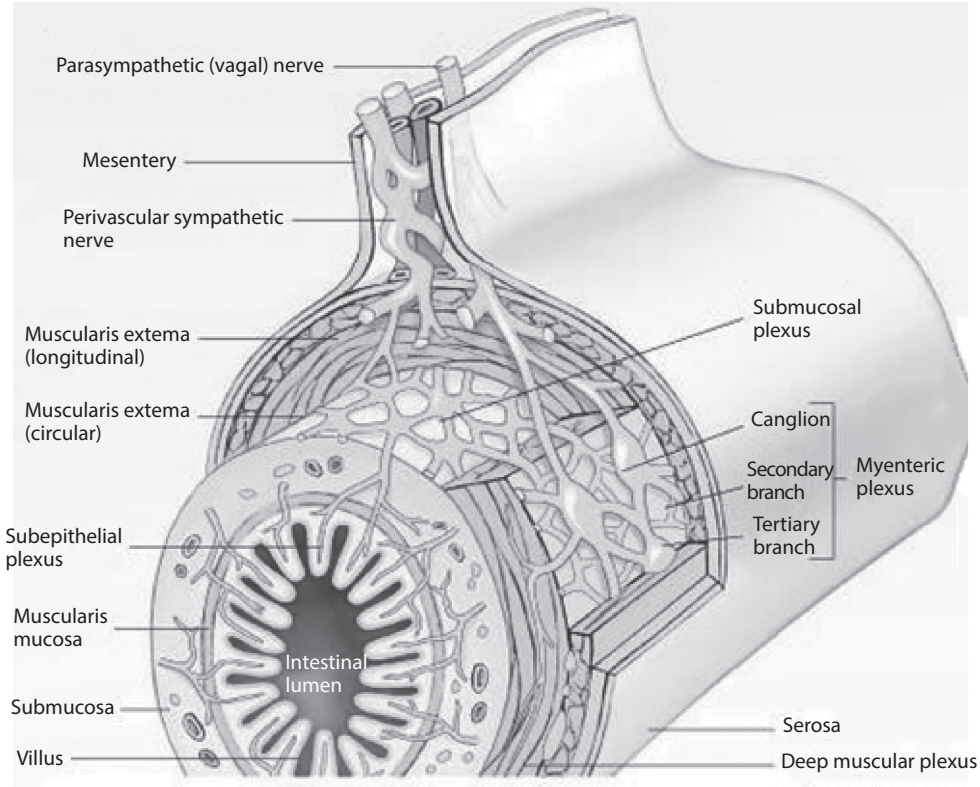
Colorectal motility is controlled by nervous, immune and hormonal systems modulating motility at four levels: enteric nervous system (ENS), prevertebral sympathetic ganglia, parasympathetic and sympathetic systems and higher brain centres. Colonic innervation emanates from two sources: the extrinsic and the intrinsic nerves. Extrinsic innervation involves the sympathetic and parasympathetic nerves of the autonomic nervous system, which are responsible for colonic motility and sensation. Intrinsic innervation arises from the ENS.

ENS neurons are separated into two types of ganglia: myenteric (Auerbach's), located between the inner and outer layers of the muscularis externa and submucosal (Meissner's) plexuses, located in the submucosa (see Figure 2.1).

The ENS is a division of the autonomic nervous system with which it has extensive connections. It contains complete reflex circuits that detect the physiological condition of the gastrointestinal tract, integrate information about the state of the gastrointestinal tract and provide outputs to control gut movement, fluid exchange between the gut and its lumen and local blood flow. Prevertebral sympathetic ganglia are mediators of the gastrocolic response-mediating tonic and phasic activity after a meal. Sympathetic stimulation causes inhibition of gastrointestinal secretion and motor activity, and contraction of gastrointestinal sphincters and blood vessels. Parasympathetic stimuli typically stimulate these digestive activities. Higher brain centres supply information integrated in the ENS thought to be inhibitory in function.

ASSESSMENT OF COLONIC FUNCTION

Colonic dysmotility can be evaluated by colonic transit time. Progression time traditionally has been assessed with radio-opaque markers and whole-gut transit with colonic scintigraphy. More recently, wireless pH motility capsules have been used to validate colonic transit time.



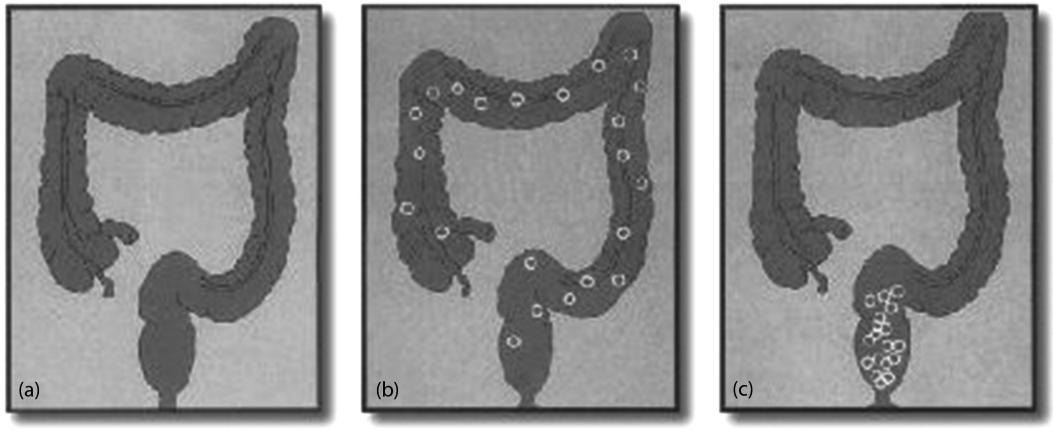
2.1 Cross section diagram of the colon showing enteric nervous system.

Courtesy of Michael Gershon Columbia University (taken from The New York Times *2005).*

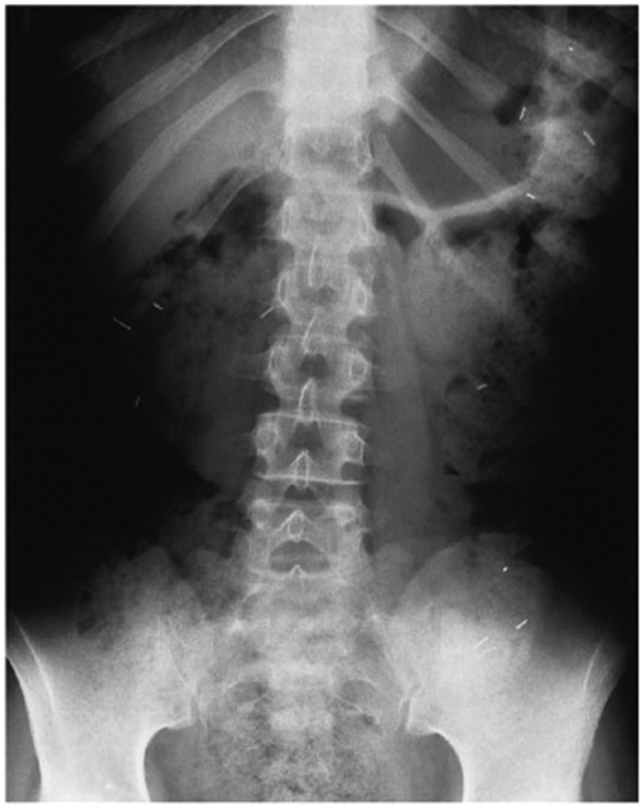
Radio-Opaque Markers

The first method devised to assess colonic transit time was ingestion of radio-opaque markers. All laxatives are ceased for 48 hours and 20 markers swallowed. A single radiograph is taken on day 5; if colonic transit is normal, at least 80% (14 markers) should have been passed. The retention of more than 20% suggests slow transit constipation (Figures 2.2 and 2.3).

Further information is obtained by assessing segmental colonic transit. Taking daily x-rays can do this. The colon is divided into three segments: right colon, left colon and rectosigmoid. Segmental transit and total transit times are assessed. Ingesting 20 markers of different shapes daily on three consecutive days and taking a single radiograph on day 4 minimise radiation exposure. Normal mean total colonic transit time in hours is 30.7 hours (SD ± 3.0) for men and 38.3 hours (SD ± 2.9) for females.[5]



2.2 Colonic transit time assessment using SITZMARKS diagnostic radio-opaque markers. (a) If 5 or fewer markers remain, patient has grossly normal colonic transit. **(b)** Most rings are scattered about the colon. Patient most likely has hypomotility or colonic inertia. **(c)** Most rings are gathered in the rectosigmoid. Patient has functional outlet obstruction.



2.3 Radio-opaque marker study, showing retention of markers on day 5 in a patient with constipation.

Colonic Scintigraphy

Colonic scintigraphy can be used as an alternative to radio-opaque markers. This technique is more complex and costly but provides clear quantitative information about segmental transit, and is particularly helpful when colectomy is being considered for constipation. Patients refrain from laxatives or opiates 24 hours before the test, and a normal diet is maintained throughout the study. The isotope is coated with pH sensitive polymer methacrylate, comprised of charcoal or polystyrene pellets and labelled with indium-111-diethylenetriaminepentaacetic acid (DTPA) or 99mTc (nuclear isomer of technetium-99). The coating dissolves at a pH of 7.2–7.4 in the distal ileum, after which the radioactive material is delivered into the colon.[6,7] Images are taken with a gamma camera at 4, 6, 24, 48 and 72 hours after consumption of the isotope (see Figure 2.4).

Segmental transit is expressed in one of two ways: percentage of isotope retained in each segment and total percentage retained or as the midpoint of the isotope column (mean activity position). The results are expressed as the geometric centre of the isotope mass at any given point, with a low count indicating the isotope is close to caecum and a higher count has progressed more distally.[8] Segmental and total percentage retention in normal subjects are shown in Table 2.1.

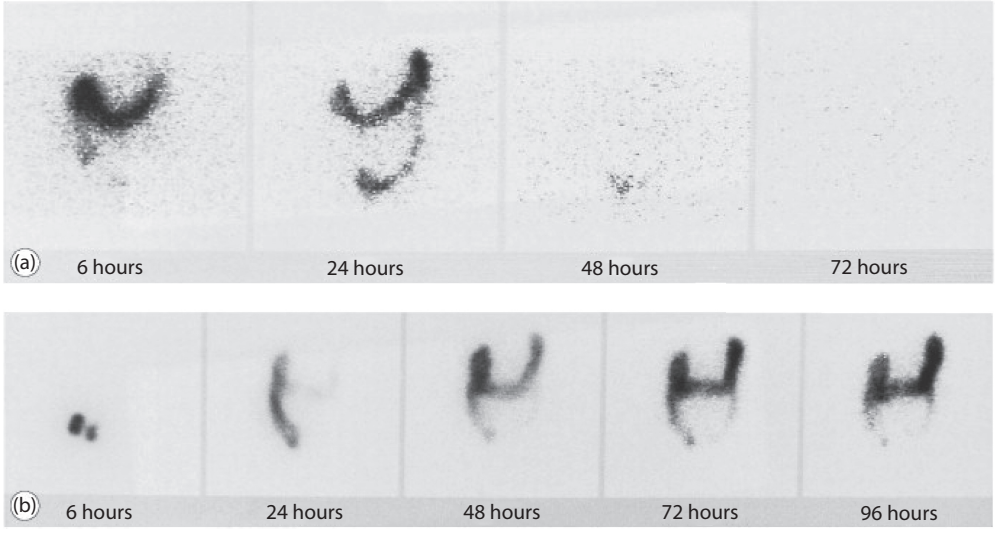
Wireless Motility Capsule

The use of a wireless motility capsule is an alternative method to determine colonic transit time. It uses a capsule containing miniature pressure, temperature and pH measurement devices. The capsule is ingested, after which continuous recordings are obtained over 5 days via a wireless device (Figure 2.5).[9]

Results from the wireless capsule have correlated well with radio-opaque markers, with a similar sensitivity and specificity in detecting abnormal transit in those with constipation.[9,10]

IMMUNOLOGY OF THE GUT

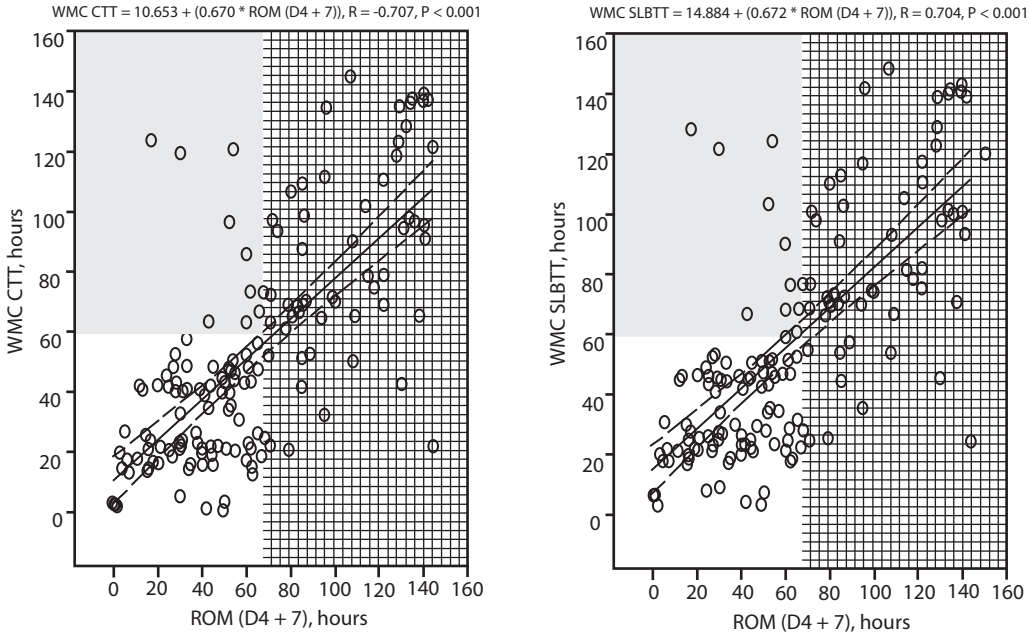
The human gut harbours >100 trillion microbes – the gut microbiota. Most reside in the colon. Gut microbiota are essential for the development of gut mucosal immunity. Microbiota-driven immune response can prevent the development of inappropriate inflammation, which allows the microbiota to survive in the inflammatory free environment. Host-microbial symbiosis is essential for gut homeostasis. If a pathogen penetrates physical barriers, then the innate immune system provides immediate non-specific response mediated by T lymphocytes and the



2.4 Radioisotope colon transit study. (a) Normal subject. At 6 hours after ingestion isotope has reached the right colon. There is normal passage of isotope through the colon over 48 hours. **(b)** Subject with severe constipation. There is prolonged retention of isotope in the colon to 96 hours.

TABLE 2.1 Mean segmental and total percentage retention of isotope in the colon in normal subjects (SD)

	Right colon	Left colon	Rectosigmoid	Total colon
24 hours	16 (7)	26 (17)	15 (9)	48 (28)
48 hours	3 (3)	5 (5)	6 (7)	11 (11)
72 hours	0.8 (1)	1 (2)	0.5 (0.5)	2 (4)
96 hours	0.2 (0.6)	0.1 (0.3)	0	0.3 (0.9)



2.5 Relationship between colonic transit time (CTT) and small and large bowel transit time (SLBTT) by wireless motility capsule (WMC) and colonic transit time by radiopaque markers (ROM) (at day 4 plus 7) in the entire patient cohort with evaluable data. Note the significant correlations between WMC estimates and ROM transit time. Interrupted line shows the 95% CI around the regression line. The shaded areas show the values at and above the 95th percentiles for the different methods: 67 hours for ROM transit, 59 hours for CTT and 65 hours for SLBTT.

From Kamada N et al., Nat Rev Immunol., 13(5), 321–35, 2013.

humoral immune system provides a slower, antigen specific response mediated by antibodies produced by B lymphocytes. The function of T cells and B cells is to recognise specific 'non-self' antigens, during a process known as antigen presentation. The cells generate specific responses that are tailored to eliminate specific pathogens or pathogen-infected cells. B cells respond to pathogens by producing large quantities of antibodies which neutralise foreign objects, i.e. bacteria and viruses. In response to pathogens, some T cells, called T helper cells, produce cytokines that direct the immune response, whilst other T cells, called cytotoxic T cells, produce toxic granules which induce the death of pathogen-infected cells. Following activation, B cells and T cells leave a lasting legacy of the antigens they have encountered in the form of memory cells.

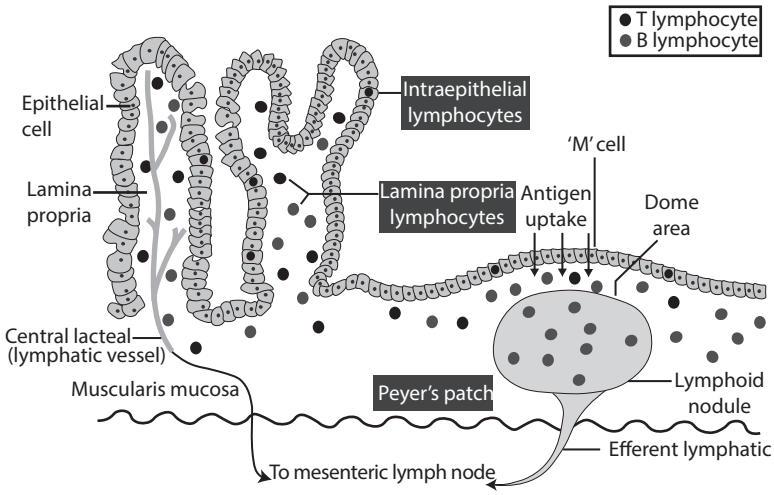
Epithelial cells form a physical barrier via intercellular tight junctions, and any injury results in migration of adjacent cells to cover the denuded area. Lymphocytes and macrophages migrate out the basement membrane and provide temporary host protection. Any change in normal microflora of the intestine allows pathogenic bacteria to flourish. The mucosa-associated lymphoid tissue (MALT) is a diffuse system of small concentrations of lymphoid tissue found in the gut. It is populated by lymphocytes such as T cells and B cells, as well as plasma cells and macrophages, each of which is well situated to encounter antigens passing through the mucosal epithelium.

The mucosa-associated lymphoid tissue (MALT) of the intestine also has M cells present, which sample antigen from the lumen and deliver it to the lymphoid tissue. Gut-associated lymphoid tissue (GALT) is a component of MALT which works to protect the body from invasion in the gut, i.e. Peyer's patches (PPs), tonsils, isolated lymphoid follicles (ILFs) and mesenteric lymph nodes (MLNs) (see Figure 2.6).[11]

Peyer's patches appear as clusters of over three lymphoid aggregates: with an overlying follicle associated epithelium, a T-cell zone and a subepithelial dome containing dendric cells (DC).[12,13] The follicle-associated epithelium contains M cells that facilitate the uptake of antigen and microbes from the gut lumen and its delivery to underlying lymphoid tissue, where priming of both T- and B-lymphocytes occur.[14,15] Within PPs are specialised T cells that induce immature IgM B-lymphocytes to switch to isotope to IgA, promoted by DCs. ILFs are similar to PPs without a T-cell zone and smaller but are present in the colon.

Primed B lymphoblasts migrate from PPs to MLNs via the lymphatics and onwards to the thoracic duct and circulation. They are recycled to the lamina propria and once in the gut mature into IgA plasma. DCs induce the activation and differentiation of naïve B cells to plasma cells that produce commensal specific IgA. Secreted IgA binds to commensal bacteria and soluble antigens, inhibiting their binding to host epithelium and penetrating the epithelial barrier.

T lymphocytes have a similar migration pathway where T blasts from MLNs 'home' to epithelium and the lamina propria. Intraepithelial lymphocytes (IELs) are



2.6 Diagram of gut associated lymphoid tissue.

functionally and phenotypically distinct from peripheral blood lymphocytes. They reside within the epithelium of the intestine and are mainly composed of CD8+ T cells. IELs delivers epithelial cell growth factor, preserving the integrity of damaged epithelial surfaces but are also capable of producing interferon γ production, resulting in inflammation.

Microbiota are crucial in the development of GALT and promote mucosal barrier function. Microbiota induce generation of the intestinal lymphoid tissue through an innate detection system. They enhance the innate immunity through regulation of mucous secretion and antimicrobial peptide production. Mucosal barrier function is enhanced by microbiota through production of by-products, short chain fatty acids. Microbiota prevent inappropriate inflammation, allowing them to survive in the absence of unnecessary inflammation. Dysbiosis of the gut microbiota could be related to several intestinal and extraintestinal diseases such as inflammatory bowel disease, irritable bowel syndrome, coeliac disease and allergy, asthma, cardiovascular disease, metabolic syndrome and obesity respectively.

Bacterial Translocation

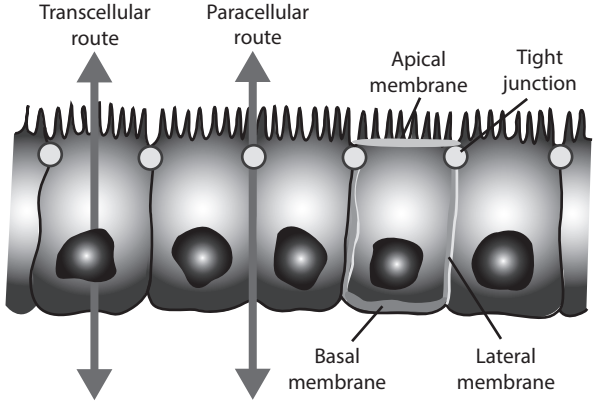
The human intestinal microflora contains 300–500 different types of bacteria with very few species in the upper GI tract due to the luminal composition and propulsive activity. The contrary is true for the colon, where over 60% of faecal matter consists of bacteria approximately to at least 10^{12} organisms per gram of luminal contents.

Microflora have several functions which include: fermentation of non-digestible dietary residue and endogenous mucus, production of short chain fatty acids by the anaerobic metabolism of peptides and proteins, potassium synthesis and the absorption of calcium, magnesium and iron.[16]

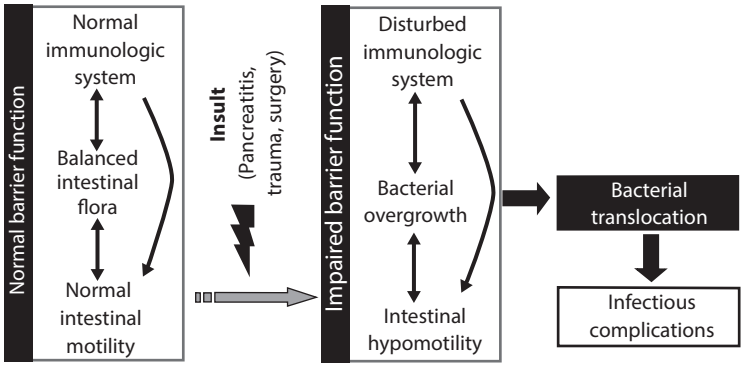
The structural organisation of the intestinal mucosal barrier and mechanism of permeable substrates through it is essential to understand bacterial translocation. The epithelial barrier contains an internal water lining, epithelial surface of phospholipids and mucous gel coat, epithelial cells, subepithelial connective tissue and capillary endothelium. Tight junctions exist between the epithelial cells which allow for selective paracellular permeability excluding the passive movement of large hydrophilic non-charged compounds, i.e. bacteria and macromolecules. The intercellular tight junctions turn over within 24–96 hours. Any injury to the epithelial barrier results in rapid migration of adjacent cells whilst lymphocytes and macrophages migrate out through the basement membrane to provide temporary host protection.

There are two major pathways of gastrointestinal permeability that cause translocation: transcellular through the enterocytes and paracellular through the tight junctions (see Figure 2.7).

Tight junction translocation is affected by luminal osmolality and enterocyte cytoskeleton. Damage results in hyperpermeability resulting in macromolecules (endotoxins) reaching the subepithelial mucosal layer and subsequently the bloodstream. More commonly, translocation is transcellular. Bacteria reach the systemic circulation



2.7 Routes of permeation through the intestinal mucosal barrier. The arrows depict the routes of permeation through the epithelial cells with transcellular and paracellular routes controlling migration between the bowel lumen and the blood.



2.8 Bacterial translocation overview.

through lymphatic enteric drainage and through the portal venous system to the portal vein.

Bacterial translocation may be a normal physiological phenomenon in healthy individuals and in very small amounts probably constitutes a physiologically important boost to the reticuloendothelial system. Excess bacterial translocation occurs due to damage of the gut barrier (direct injury, intestinal microvascular alterations in shock, SIRS), alterations in microbiota small bowel (alterations in motility, absence of intestinal bile salts and antibiotic use) and systemic immunosuppression. Common causes of bacterial translocation are acute pancreatitis, obstructive jaundice, cirrhosis, abdominal surgery, burns, trauma, haemorrhagic shock, AAA repair, CABG and bowel transplant (see Figure 2.8).

INTESTINAL GAS

Flatulence is defined as the expulsion of gastrointestinal gas from the anorectal canal and eructation as the expulsion of gas from the oral cavity. The rumbling noise of gas propagated through the gastrointestinal tract is borborygmus. The volume of gas is dependent on the dynamic balance between input and output. Healthy human individuals produce 500–1500 mL of gas daily, and retain a relatively constant small amount, approximately 200 mL. Gastrointestinal gas is derived from four sources: aerophagia, bacterial fermentation, luminal chemical reactions and diffusion from circulation into the lumen. Excretion involves eructation, bacterial consumption, absorption into blood and anal evacuation.

Ninety-nine percent of intestinal gas is composed of five gases – N_2, O_2, CO_2, H_2 and CH_4. There are high concentrations of nitrogen and low concentrations of oxygen within intestinal gas. Highly variable concentrations of hydrogen sulphide, carbon dioxide and methane exist from products of fermentation.

Aerophagia and Eructation

Air is swallowed during eating and is thought to be the major source of stomach gas. Normally, 10–20 mL of gas is contained within the stomach.[17] Dependent on the position, the gas either forms a bubble in the fundus in an upright position or within the antrum in the supine position and is either expelled or emptied into the duodenum respectively.

Intestinal Gas Transit – Propulsion, Accommodation and Tolerance

A small amount of gas in the small and large bowel can be secondary to aerophagia, but larger amounts are secondary to endogenously formed gas. In the proximal small bowel bicarbonate (HCO^{3-}), originated from biliary, pancreatic and small intestinal secretions react with gastric acid, from gastric secretions or fatty acids from triglyceride digestion, to form CO_2 and H_2O. Each mole of H^+ is neutralised by HCO^{3-} and produces 1 mole of CO_2. Gas transit is independent of solids and liquids within the GI tract although is more effective in the erect position.[17] Gas is then absorbed by diffusion or transported to the colon. Gas transit is normally effective and modulated by a series of reflex mechanisms. Intestinal gas propulsion and clearance is associated with a tonic contraction of the gut wall and reduced gut capacitance.[18] Mechanical stimulation with consumption of solid food and mild rectal distension has a prokinetic effect, whereas lipids and fibre delay gastric transit.[19,20]

Flatulence

Intestinal gas homeostasis is incompletely understood. Partly metabolised substrates are passed into the colon, which harbours large numbers of gas-producing microbiota. These substrates are metabolised by gut flora to release H_2, CO_2, CH_4, sulphur containing gases and trace gases NO. Bacterial production of CO_2, H_2 and CH_4 reduces the partial pressures of N_2 and O_2, resulting in diffusion from blood to lumen. A proportion of the O_2 is consumed by intestinal microorganisms allowing metabolism. The composition of colonic microflora and consequently gas production varies amongst individuals and depends on environmental factors including dietary factors. Gas disposal is therefore by one of three ways: absorption into the bloodstream and subsequent exhalation, metabolised by gas consuming microorganisms and remainder expelled through the anus. The average person who eats a normal diet will evacuate flatus 10 times a day with the upper normal limit of 20 times a day.[21] Approximately 200 mL of N_2 is evacuated from the anus daily.

Intestinal Accommodation and Tolerance to Gas

Intestinal gas dynamics have been investigated with a gas challenge test, whereby exogenous gas is infused into the intestine and gas evacuation, girth and abdominal symptoms recorded. Gas infused at 30 mL/min is well tolerated and evacuated in its entirety in a healthy cohort without discomfort, whereas infusion at a smaller volume of 12 mL/min have reproduced symptoms of abdominal distension and impaired gas transit in patients with irritable bowel syndrome (IBS).[22] Patients with functional bowel disorders, IBS and related syndromes frequently attribute their symptoms to intestinal gas retention.

PHYSIOLOGY OF IRRITABLE BOWEL SYNDROME

Irritable bowel syndrome (IBS) is a common functional bowel disorder, characterised by pain and altered intestinal motility.[23] Traditionally, IBS was thought to be due to visceral hypersensitivity and altered gastric motility.[24] with no known underlying structural or biochemical explanation. It is now accepted that alterations in both sensory and motor function are responsible for the clinical manifestation of symptoms; however, it is not the cause. The exact pathophysiology of IBS remains incompletely understood with multiple physiologic and pathological factors described that may play a role, although the contribution of each in any one individual patient is probably variable (Figure 2.9).[25]

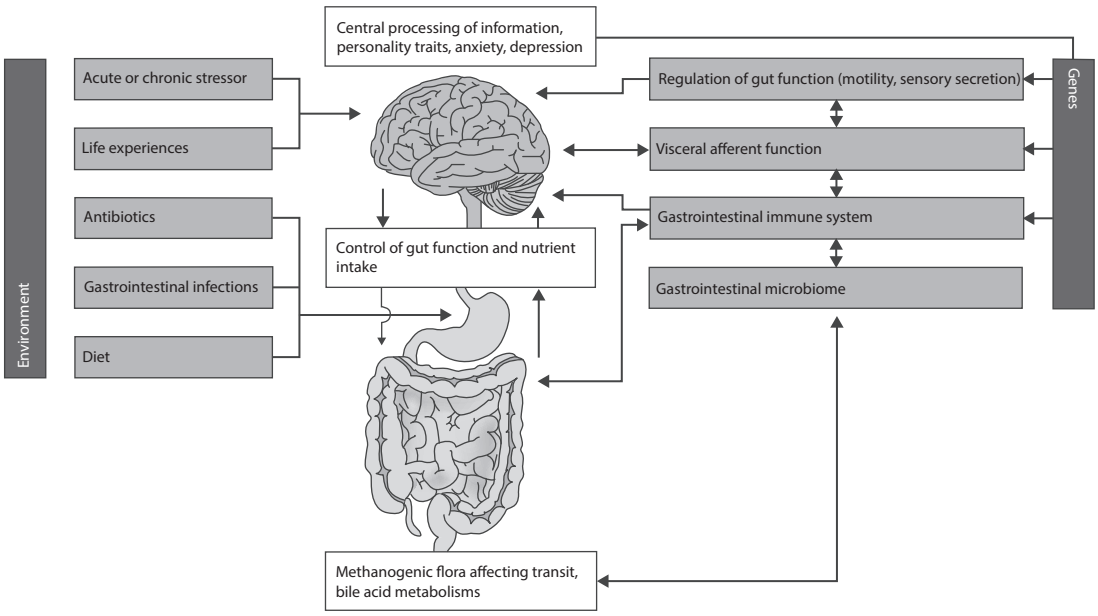
IBS: A Gut–Brain Disorder

Psychological factors may influence the expression of IBS, with anxiety and depression being prevalent in the IBS cohort.[26,27] Such observations have led to the conceptualisation of IBS as a primary disorder of the brain–gut function[28] or primary somatisation[29] with the brain driving the gut manifestation. Overactivity in the brain releases corticotropin releasing factor (CRF), which is considered to be a major mediator of the stress response and contributes to anxiety and depression disorders.[30] It is unknown whether depression causes the GI symptoms or whether GI symptoms predispose to the psychological symptoms.

IBS: A Diet Disorder

Many patients with IBS report worsening of symptoms after ingestion of certain foods. One aetiology suggests a role for fermentable oligosaccharides, monosaccharides and disaccharides and polyols (FODMAPs). FODMAPs are poorly absorbed short-chain carbohydrates that can cause excess fluid and gas accumulation that leads to abdominal distension and pain. It is thought that FODMAPs entering the distal small bowel and colon and being fermented leads to increased intestinal permeability and subsequent osmotic effect and inflammation.[31] Elimination of FODMAPs from the diet has been shown to improve symptoms in 86% of patients.[32]

The theory that diet is contributory to the pathogenesis of IBS is supported by the findings of several studies that have shown an overlap between coeliac disease and IBS.[33,34] A study in patients with diarrhoea-predominant IBS without coeliac disease found that dietary gluten altered small intestine permeability and had a greater effect on bowel movement frequency in patients that are HLA-DQ2/8 positive (associated with coeliac disease) compared with those who were negative.[35] A double blind study in which non-coeliac IBS patients adhered to a gluten-free diet and then re-introduced gluten in a double blind fashion found 68% of patients who took gluten had



2.9 Potential factors that determine the manifestation of irritable bowel syndrome symptoms.

a deterioration in their symptoms, as opposed to 40% who were treated with a placebo.[36]

IBS: Genetic Factors

Familial studies suggest that genetics may have a modest contribution to the development of IBS.[37] Studies that have examined the incidence of IBS in monozygotic and dizygotic twins are conflicting; some studies report higher incidence in monozygotic twins, whilst others have shown no difference. In the largest study of 1870 twin pairs, there was no difference in IBS prevalence between monozygotic and dizygotic twins.[38] A further study has found that having an effected parent was a greater independent predictor than having an effected twin, which implies that the familial nature of IBS could be due to social learning rather than genetic influence.[39]

IBS: Infectious and Disturbances in the Intestinal Microbiotica

Acute enteric infections of bacterial, protozoan and viral origin frequently precede the onset of IBS.[40,41] A meta-analysis of 18 studies concluded that the incidence of IBS after gastroenteritis was ten percent, and the odds of developing IBS after gastroenteritis are increased sixfold.[42] The reasons for the development of post-infectious IBS are poorly understood. Potential theories include the development of idiopathic bile salt malabsorption and increased serotonin-containing enteroendocrine cells and T lymphocytes which cause increased GI motility and hypersensitivity.

Changes in a patient's faecal microbiota have been linked with several gastrointestinal diseases, including IBS. The faecal microbiota of individuals with IBS, post-infectious IBS and healthy controls have been shown to be different. This suggests that alterations in the microbiota may be contributory to the symptoms of IBS sufferers. Dysbiosis in IBS patients results in abnormal levels of intestinal fermentation. Patients with IBS have shown to have lower number of lactate producing, lactate metabolising and hydrogen consuming bacteria and more sulphides and hydrogen, less butyrate within their microbiota.

Low-Grade Mucosal Inflammation and Immune Activation

In some IBS patients, low-grade mucosal inflammation has been identified using immunohistological techniques. In particular, elevated numbers of immune cells (lymphocytes and mast cells) have been observed in the small intestine and colon of some patients with IBS.

In IBS patients, there are increased concentrations of pro-inflammatory cytokines and mast cells within close proximity to enteric nerve fibres in the GI mucosa.[43] High cytokine concentrations have been associated with anxiety and depression. Humoral immunity activation with activation of B-lymphocytes have shown to be more active in patients with diarrhoea-predominant IBS. The cause of the altered immune function is unclear but may be due to the integrity of the GI mucosal epithelial barrier.[25]

Alteration in Intestinal Permeability

Patients with IBS with and without an infective aetiology have abnormal levels of intestinal permeability. In the duodenum of patients with IBS and food intolerance, within 5 minutes of exposure to antigens, intraepithelial lymphocytes increase and epithelial gaps increase, and as a consequence intervillous space widens.[44] Increased intestinal permeability leads to a cascade of microbes upregulating mast cells through T-helper-2 cells. Toll-like receptors (TLRs) on mast cells may interact directly with relevant microbes. Mast cells release histamine, which interacts with histamine 1-4 receptors, proteases (PAR1 receptor) and serotonin (5-HT3 and 5HT4 receptors). Chemical signalling via rectors results in neural excitation and smooth muscle contraction leading to abdominal pain, abnormal intestine-abdominal reflex responses and disturbed intestinal transit.[45]

Bile Salt Metabolism Disorder

Bile acids are produced in the liver and released into duodenum and 95% are then reabsorbed in the terminal ileum via entero-hepatic recirculation. The remainder are passed to the colon and recycled via the portal vein to hepatocytes. Faecal bile acid is higher in patients with diarrhoea-predominant IBS and lower in constipation-predominant IBS than in healthy controls. Approximately 20% of patients with diarrhoea-predominant IBS symptoms may have evidence of idiopathic bile acid diarrhoea shown by 23-seleno 25-homotaurocholic acid retention scanning. However, bile acid diarrhoea could equally be a cause of IBS with rapid transit leading to bile acid depletion. Increased faecal bile acids in patients with diarrhoea-predominant IBS has been associated with dysbiosis, with studies showing increase in *E. coli* and reductions in bifidobacteria and leptum.[46]

Serotonin Metabolism Disorder

Serotonin (5-HT) is an important enteric nervous system and brain neurotransmitter. Ninety percent of the body's 5-HT stores are within the intestinal enterochromaffin cells, which act as sensory transducers of intraluminal stimuli, i.e. pressure. It affects motility and transmission of information to the CNS. Serotonin reuptake by enterocytes is via the serotonin transporter (SERT), where it is broken down to 5HIAA. Studies have reported a reduced 5HIAA (breakdown product of 5HT)/5HT ratio in those with IBS with diarrhoea, suggesting reduced reuptake. A reduced level of SERT is associated with duodenal immune activation with an increased number of intraepithelial lymphocytes and mast cells.[47] Colonic biopsies from patients with IBS show a higher proportion of 5HT-positive enterochromaffin cells than controls. Mucosal serotonin is also higher and related to the mast cell count and severity of abdominal pain.

The definitive cause of IBS in all patients remains unclear. It is unlikely to be a single abnormality that causes the diversity of clinical symptoms experienced by IBS patients. It seems more plausible that there are

multiple causes that share similar pathways that would explain the similarity and variability of the symptoms in IBS patients. It may also be possible that that the development of IBS may not be an all-or-nothing event, rather it may that multiple 'hits' from the various factors may culminate in symptoms that are attributed to IBS.

ANORECTAL

Mechanism of Rectal Evacuation

Whilst the main function of the colon is the transit of faeces, the function of the rectum is to act as a reservoir and to defer emptying until a socially convenient time for the individual. This is a complex process dependent on the continuity of the muscular tube of the colon rectum and anus, a normally functioning puborectalis sling and an intact sensory and motor nerve supply via the pudendal nerves and the pelvic plexuses.

Colonic contractions ensure propulsion of faeces into the rectal reservoir that accommodates by relaxation of its muscular walls and an increase in rectal capacitance. The tone of the puborectalis sling and the internal and external anal sphincters ensures that the rectum can retain its contents. The puborectalis muscle forms the pelvic floor and surrounds the rectum posteriorly and laterally to create an angle between the anus and rectum to help preserve continence. During defaecation, the puborectalis muscle relaxes reducing the sling effect and therefore the anorectal angle. The rectum straightens as a result, facilitating rectal emptying as the anal sphincters relax. This whole process is entirely dependent on the nervous innervation of the rectum and anus. During normal rectal emptying, the following changes are observed: (1) As straining begins, abdominal pressure produces a slight concavity of the anterior rectal wall. (2) The pelvic floor descends. (3) The anorectal angle widens. (4) The anal canal begins to open, shortens and becomes funnel-shaped. (5) Rectal evacuation begins and emptying is completed. (6) A slight degree of rectal wall intussusception may occur.

Rectal distension occurs initially without conscious awareness of the individual until a certain volume is reached (usually 20–60 mL). Sensory pathways to the cerebral cortex alert the individual to this distension and a desire to defaecate may ensue. This sensation may be consciously overridden to defer defaecation if such activity was not convenient. As the rectal distension increases, the desire to defaecate increases and will eventually become a painful urge, usually at a volume more than 200 mL.

When defaecation is convenient and the individual chooses, a conscious process is initiated by which the puborectalis and anal sphincters relax, the rectum straightens and rectal contractions ensure complete evacuation. The tone then returns to the anal sphincters and the puborectalis which restores the anorectal angle. Whilst involving involuntary reflexes and contractions, rectal evacuation remains under voluntary control and can be interrupted if the individual wishes. This is achieved because the puborectalis and external anal sphincter are made of striated muscle under voluntary control and can be contracted at will, interfering with the smooth process of rectal evacuation.

The rectum is sensitive to distension alone, whereas the anal canal is sensitive to temperature, touch and pain. The lining of the anal canal above the dentate line consists of an area of mucosa which is richly innervated and can distinguish between solid, liquid and gas. The internal sphincter relaxes transiently when the rectum distends, and this enables the sensitive mucosa to determine the nature of the bowel content. This rectoanal inhibitory reflex is essential for normal function, as the rectum can evacuate flatus without releasing any solid or liquid material. A loss of the rectoanal inhibitory reflex is pathognomic of Hirschsprung's disease. Passing flatus relieves distension of the rectum and can facilitate deferring rectal evacuation of solid and liquid motion.

The normally functioning anorectum exhibits other nervous reflexes which can be used to determine the presence of nerve injuries. The ano-cutaneous reflex causes the external sphincter to contract in response to touch or pain stimulus of the anal skin; this is absent in cauda equina injuries. Contraction of the external sphincter also occurs in response to coughing, sneezing and laughing (cough-anal reflex) and is absent in sacral nerve or cauda equina injuries. Squeezing the glans penis or clitoris induces contractions in the external sphincter (bulbocavernous reflex) and is absent in S2 to S4 injuries.

Assessment of Disorders of Rectal Evacuation

Digital Rectal Examination (DRE)

DRE is a routine part of colorectal assessment and whilst not a physiological assessment, it can provide important information and direct further investigation. It is usually performed in the lateral decubitus position but may also be undertaken with the patient in the knee elbow position. Verbal consent is obtained, and an explanation of the technique is given. The anus is always inspected first for any external abnormalities, including skin tags, haemorrhoids, dermatological conditions and fissures or fistulae. A gloved and lubricated finger is then inserted after applying pressure to the posterior aspect of the anus to relax it. The finger is inserted to its full length if possible, and the anal and rectal walls are inspected for haemorroidal columns, ulceration and masses. The resting tone of the sphincters can be assessed, followed by the ability to voluntarily squeeze the external sphincter. With experience, it is possible to assess whether the resting tone and squeeze are high, low or normal. With the finger in the rectum, the patient is then asked to bear down, and a note of any paradoxical squeeze is noted. In women, the anterior rectal wall is assessed for the presence of significant rectocoele, bearing in mind that most will have a degree of laxity.

Defaecography

Defaecography is the most widely used assessment of rectal evacuation to date, as it is a dynamic assessment and is the closest approximation to physiological evacuation available. More recently, dynamic MRI scanning has been utilised.

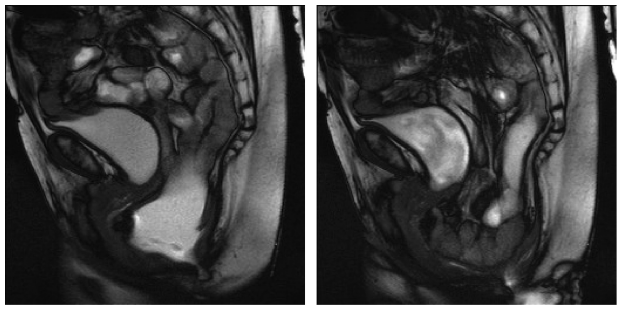
The dynamic procedure involves both static radiological views of the rectum and dynamic views using fluoroscopy. The patient initially lies in the left lateral position and a catheter is placed into the rectum. Up to 100 mL of liquid barium is instilled into the rectum and a still radiograph is taken to confirm rectal filling. Thickened barium paste consisting of barium, water and porridge oats is then instilled via the catheter until it enters the sigmoid colon. The patient then sits on a radiolucent commode, and static views of the rectum are taken when straining and relaxing. The barium paste is then evacuated as the patient would normally evacuate faeces whilst undergoing fluoroscopic screening. The procedure can be completed in about 15 minutes.

Fluoroscopy allows a visual assessment of rectal evacuation as well as the ability to quantify various parameters of anorectal function. These parameters can then be compared to a normal population (see Table 2.2).

Defaecography therefore is useful in identifying abnormalities of rectal evacuation. However, the clinical significance of these abnormalities is often difficult to determine because anatomical abnormalities demonstrated on proctography do not necessarily correlate with patient symptoms, as noted in a study of defaecography in normal volunteers that found that 40% of patients with no symptoms had a rectocele.[48] Taken with the clinical assessment of the patient, the findings may be used to determine which treatment modality should be implemented (see Chapter 16).

TABLE 2.2 Parameters and range of anorectal function

Observation	Normal range
Puborectalis length	Rest 14–16 cm Squeeze 12–15 cm Push 15–18 cm
Anorectal angle	Rest 70–140 degrees Squeeze 75–90 degrees Push 100–180 degrees
Perineal descent	Rest <3 cm Push <3 cm change from rest
Puborectalis notch	Push-blunted
Rectocoele	<2 cm. Complete emptying with push
Prolapse/ Intussusception	Absent
Anal canal opening	Push-complete opening
Rectal emptying	10–12 seconds to completion



2.10 Dynamic MR imaging. Broad necked rectocele on the left and an enterocele on the right.

Dynamic MRI

MRI scanning does not involve exposure to ionising radiation and therefore makes it an attractive alternative to defaecography. It can be undertaken using either an open MRI or closed MRI machine. Prior to the procedure, the patient drinks 600 mL of water to highlight the small intestine. Also, intravenous contrast is administered to highlight the renal system and bladder following excretion. Finally, 240 mL of aqueous sonographic gel is instilled into the rectum via a narrow catheter with the patient lying on their side. In a closed scanner, images are acquired at rest or whilst squeezing or straining with the patient lying still on their back (see Figure 2.10).

Whilst it is possible to investigate the evacuatory phase in the supine patient, this is often omitted, as it is considered less physiological and can present difficulties for the patient. This can be overcome by using an open MRI scanner with the patient sitting upright in a wooden chair to facilitate defaecation. This also negates the need to clean the patient which is necessary following defaecation in a closed scanner.

Although open MRI scanners are a scarce resource and not widely available, there is evidence that the detection rate for abnormalities is greater when an open MRI is used to include the evacuation phase in the investigation.

Comparisons of conventional defaecography (CD) and MR defaecography (MRD) have been made previously.[49–51] These studies suggest that CD may be more accurate than MRD in the diagnosis of enterocoele and rectocele and is less time consuming and cheaper but MRD gives a global view of the anatomy of the pelvic organs and musculature and may be better tolerated by patients (Table 2.3).

Anorectal Manometry and Balloon Expulsion Test

Anorectal manometry studies (ARM) are most frequently used for the assessment of faecal incontinence but can also be used in the investigation of patients with symptoms of obstructed defaecation. A variety of pressure measuring probes are available and will vary between different departments. The probes can measure pressure by either water perfusion based systems or solid state catheters.

TABLE 2.3 Risk benefit analysis of conventional defaecography and MR defaecography

	Conventional defaecography	MR defaecography
Advantages	Physiological position for defaecation Cheaper More accurate diagnosis of enterocoele and rectocele	Detailed global anatomical assessment of pelvis No ionising radiation
Disadvantages	Only visualises the posterior pelvic compartment	Non-physiological position for defaecation* More expensive

*Unless performed in an open MR scanner.

The catheters can be either non-high resolution systems, high resolution systems (HR-ARM) or high definition systems (HD-ARM). HR-ARM and HD-ARM systems are more expensive and more fragile, having a shorter lifespan than the non-high resolution systems. Also, measurements from each system will vary and are not necessarily directly comparable.

Regardless of which system is used, the technique should at the very least assess the following parameters of anorectal function: rectoanal pressure, anal canal length at rest, rectoanal pressures during squeeze, simulated evacuation and coughing, rectal sensation and the balloon expulsion test.

The test is undertaken with the patient in the lateral decubitus position. The rectum should be empty of faeces and so is best performed 30 minutes after an enema, though this is not routinely given if the rectum is empty on digital examination.

The lubricated probe is inserted into the rectum up to between 6–10 cm and is left in place for the duration of the study. A period of up to 5 minutes is used to allow the patient to relax before measurements are initiated and the anal sphincter pressures return to baseline.

Anal resting pressure is generally measured over a 20-second period. The patient is then asked to squeeze the anus for a maximum of 30 seconds followed by a 1-minute rest. This is then performed three times, preferably without an increase in rectal pressure which would indicate that the patient had contracted their abdominal muscles. Asking the patient to cough is useful to assess spinal reflex pathways, since in the normal patient this induces external anal sphincter contraction.

To assess rectal sensation, the rectal balloon is distended with air in increments of 10 mL until the patient reports first sensation, usually after insufflation of around 20 mL. Subsequently, the balloon is inflated in increments of 40 mL up to a maximum volume of 400 mL or until it is not tolerated by the patient. In between first sensation and maximal sensation, the patient is asked to report desire to defaecate and urgency to defaecate.

The rectoanal inhibitory reflex is assessed by rapid instillation of 20 mL of air into the empty balloon. If there is no detection at this level, 20 mL increments of air are instilled until there is sensation of distension. Rapid distension of the rectum causes an initial short contraction of the external anal sphincter, followed by a slower and more sustained relaxation of the internal anal sphincter. This reflex is absent in Hirschsprung's disease.

The balloon expulsion test involves inflating a balloon to a volume of 50 mL and asking the patient to expel the balloon in the sitting position. This should normally occur within 1 minute and is considered pathological if longer than 3 minutes.

Pudendal Nerve Motor Latency

This test is of limited clinical usefulness for evacuatory disorders but can be abnormally long in patients with rectal prolapse. The anal sphincters are innervated bilaterally by the pudendal nerves and injury or stretching of both nerves is necessary for there to be a clinically detectable problem. The test involves a determination of the time elapsed between pudendal nerve stimulation at the ischial spine and contraction of the sphincters, which normally occurs within 2 milliseconds. With the patient in the lateral decubitus position, a fingerstall device with implanted electrodes is placed on the gloved index finger along with some electrode gel. The coccyx is palpated, and the finger rotated towards the lateral wall of the rectum until a maximal response is obtained from the contracting external sphincter muscle. The average of three readings is taken, as the pudendal nerve motor latency and the procedure is then repeated on the opposite side.

Electromyography

EMG is a test of the strength of muscle depolarisation and captures the activity of the puborectalis and external sphincter. It is of limited clinical importance, but abnormalities can be detected in patients with constipation. The electrodes used may be surface electrodes, needle electrodes or an anal plug electrode. Activity can be assessed at rest or on squeezing and pushing. In a normal patient, amplitude of motor unit contractions is a maximum of 2 mV; voluntary contraction potential lasts 5 to 7.5 milliseconds and during defaecation EMG activity should approach zero. Whilst in patients with nerve injury, the amplitude will be decreased, in patients with symptoms of obstructed defaecation and paradoxical puborectalis contraction, the EMG activity will be high during defaecation. EMG is used as part of biofeedback for

muscle retraining in patients with obstructed defaecation (see Chapter 16).

Physiology of Faecal Continence

Faecal incontinence is the unintentional loss of faeces or flatus via the anal canal. Continence is dependent on the pressure within the rectum being lower than that exerted by the closed anal sphincters and should be achievable whatever the rectal contents. The most important muscle for continence is the internal sphincter. This involuntary smooth muscle contributes over 50% of the resting tone of the sphincter complex by means of slow constant waves of contraction. The external sphincter consists of striated muscle which is under voluntary control but nevertheless contributes 30% of the basal resting tone. Voluntary contraction of the external sphincter can be performed to retain rectal content, but fatigue occurs after approximately 1 minute. The remaining tone is provided by the anal cushions which therefore contribute significantly to the continence mechanism. The puborectalis muscle is attached to the pubic bone, and the resulting sling effect causes closure of the distal rectum. This prevents rectal content passing into the distal rectum.

The sensory innervation of the rectum is via the hypogastric nerves and pelvic plexus. These detect distension of the rectum that results in a non-painful urge to defaecate. With increased distension, the rectoanal reflex causes relaxation of the anal sphincters via the pudendal nerves to allow defaecation. Although an intact anorectum demonstrates coordinated relaxation of the anal sphincter because of rectal sensory innervation, it is not essential for continence, as demonstrated by the fact that the majority of patients who undergo colo-anal or pouch-anal anastomoses are continent.

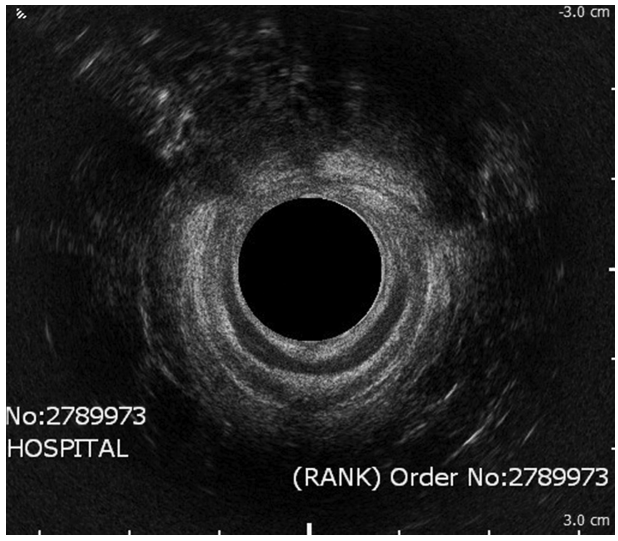
A failure of any of the above components of normal function can result in incontinence to either solid, liquid or flatus. This can occur to varying degrees but can be socially debilitating for many patients.

Investigations of Incontinence

Endoanal Ultrasound

Whilst clinical findings may be useful in the assessment of patients with faecal incontinence, it is not always possible to accurately determine the presence or degree of sphincter defects. In this scenario, endoanal ultrasound is the most useful modality. It is performed with the patient in the left lateral position, and the lubricated ultrasound probe is inserted into the anal canal as far as the distal rectum. Cross-sectional images are obtained from the upper, middle and lower anal canal by withdrawing the probe slowly and can be stored for later review. A 10 MHz probe provides the best pictures which may be either 3 dimensional or 2 dimensional cross sections of the puborectalis as well as the internal and external anal sphincters (see Figure 2.11).

Findings may include the location of specific defects, the degree and extent of defects, presence of thinning of the sphincter muscles and bulk of the perineal body, all of which may be useful in planning the management of the patient.



2.11 Endoanal ultrasound showing disruption of internal and external anal sphincter 3 months after obstetric damage.

Anal Manometry

This technique, along with endoanal ultrasound, forms the mainstay of investigating patients with faecal incontinence. The technique has been described previously. Patients with faecal incontinence may have reduced tone because of weak internal or external sphincter muscles. In addition, weak squeeze pressures may be demonstrated due to a weak external sphincter or puborectalis muscle. Anal manometry studies will therefore provide evidence of impaired muscle function and will also indicate areas of possibly defective muscle which can be represented visually in a 3-dimensional format. Taken together with the information provided by endoanal ultrasound, the tests will indicate whether the patient has neurogenic faecal incontinence or is suitable for repair of a defect in the external anal sphincter. Normal values in men and women for manometry are presented in Tables 2.4a and 2.4b.

Pudendal Nerve Motor Latency

PNML is used to determine a delay in anal sphincter contraction following electrical stimulation of the pudendal nerves. It has been utilised in patients with faecal incontinence who have no evidence of sphincter defects on endoanal ultrasound or anal manometry investigations. Evidence suggests that the vast majority of such patients have normal latency. Furthermore, it has been demonstrated that unilateral increase in PNML is not associated with faecal incontinence. Bilateral increase in PNML which occurs in up to 12% of such patients does correlate with degree of incontinence but has no influence on the choice of therapy.

TABLE 2.4A Normal values of high resolution and high definition manometry in men

Authors	Li et al.[52]	Lee et al.[53]	Carrington et al.[54]	Cross-Adame et al.[55]
Years	2013	2014	2014	2015
Participants	64	27	19	36
Ethnicity	Asian	Asian	Western	Western
Variables	Mean	Median	Mean	Mean
Max resting pressure	69.5			90
Mean resting pressure	61.3	46	73	266
Max squeeze pressure	194.8	178	290	
High pressure zone length	3.6		3.9	4.3
Duration sustained squeeze	12.3		16	30
Residual anal pressure	81.2	26	57	40
Anal relaxation rate (%)	22.5	16	16	
Intrarectal pressure	72.3	69	71	43
First sensation to defaecate (mL)	44.2	10		22
Desire to defaecate (mL)		80		94
Urge to defaecate (mL)	102.5	130		163
Discomfort (mL)	154.5			206
Balloon expulsion time		15		

Defaecography

The parameters measured by Defaecography have been discussed. It is useful for the investigation of rectal evacuation disorders but has limited use in the investigation of faecal incontinence.

Urodynamics and Their Relevance to Colorectal Surgery

The bladder and rectum are closely related, anatomically sharing their innervation from the same pelvic plexuses. It is therefore no surprise that during complex rectal surgery, the nervous innervation of the bladder, urinary sphincter and pelvic floor may be compromised, potentially resulting in impaired function. The colorectal surgeon must have an extremely detailed anatomical knowledge of these related structures to safely perform resectional surgery. However, a detailed knowledge of pelvic neurophysiology is not necessary and is beyond the scope of this chapter. However, colorectal surgeons do require a basic understanding of the function of the pelvic plexuses to assess such patients following pelvic surgery.

The bladder performs a similar function to the rectum in that it acts as a reservoir by maintaining a sphincter pressure which is greater than the intravesical pressure. The smooth muscle of the bladder wall has a unique ability to distend without an increase in pressure. This property is important since unlike the rectum, the contents of the bladder are entirely liquid and any change in the compliance of the bladder can affect its storage capacity. The balance between the compliance of the bladder muscle and the tone provided by the urinary sphincter mechanism is dependent on the sympathetic nerves (via the hypogastric nerves and sympathetic chains) the parasympathetic nerves (S2 to S4 via the lateral pelvic plexuses) and the somatic innervation via the pudendal nerves and its branches. The bladder has two functions, to fill and to empty. During the filling phase, relaxation of the bladder is maintained by sympathetic activity to the detrusor muscle and simultaneous sympathetic drive to the sphincter, causing it to contract. Bladder contractions are driven by parasympathetic activity which is largely absent during the filling phase. Bladder sensation is transmitted to the central nervous system (CNS) via general visceral afferent fibres (GVA). The GVA fibres on the superior surface follow the course of the sympathetic efferent nerves back to the CNS, whilst GVA fibres on the inferior portion of the bladder follow the course of the parasympathetic efferents. When the bladder reaches near full capacity there is a strong urge to urinate; voluntary emptying of the bladder is initiated by bladder outlet relaxation (pudendal innervation) followed by a decrease in sympathetic stimulation then increased parasympathetic stimulation, causing detrusor contraction. Parasympathetic activity continues until complete emptying of the bladder occurs.

TABLE 2.4B Normal values of high resolution and high definition manometry in women

Authors	Noelting et al.[56]	Noelting et al.[56]	Li et al.[52]	Lee et al.[57]	Carrington et al.[54]	Cross-Adame et al.[55]
Years	2012	2012	2013	2014	2014	2015
Participants	30 < 50 years	30 > 50 years	46	27	96	42
Ethnicity	Western	Western	Asian	Asian	Western	Western
Variable	Mean	Mean	Mean	Median	Mean	Mean
Max resting pressure	88	63	68.5			76
Mean resting pressure			60.2	32	65	
Max squeeze pressure	167	162	167.4	75	225	205
High pressure zone length	3.6	3.5	3.5		3.5	4
Duration sustained squeeze	12	14	14.7		11	28
Residual anal pressure	63	32	65.2	19	43	36
Anal relaxation rate (%)	32	25	27.2	30	24	
Intrarectal pressure	20	32	45.8	37	64	39
First sensation to defaecate (mL)	33	32	40	10		24
Desire to defaecate (mL)	56	59		60		88
Urge to defaecate (mL)	86	96	92.6	115		139
Discomfort (mL)			145			193
Balloon expulsion time	31	17		15		

Trauma to any of these nerves either by bruising, stretching or division can result in impairment of either the filling or emptying functions of the bladder. Furthermore, if the bladder is partially resected, for example when there is a colovesical fistula, this can significantly reduce bladder capacity. The nerves to the bladder are most likely to be affected during low anterior resection and abdomino-perineal resection but can also be damaged to a lesser degree during rectopexy for rectal prolapse. Whereas the hypogastric nerves are usually readily identified, it may be much more difficult to identify and preserve the parasympathetic supply via the lateral pelvic plexuses. This is particularly true when undertaking rectal excision in the narrower male pelvis especially if the rectal tumour is a large one. Therefore, the most likely problem encountered by the colorectal surgeon is a non-contractile bladder which either fails to empty or empties poorly, although there may be some spontaneous improvement during the first 6 months following surgery. There is no medication available to stimulate a bladder which has had its parasympathetic innervation impaired during surgery, and so such patients will require self-intermittent catheterisation to ensure complete emptying of the bladder.

Patients who present with urinary symptoms post-operatively may have had pre-existing conditions such as bladder outlet obstruction, bladder instability and urinary incontinence, and for this reason it is often difficult to determine exactly what the patient's problem is on clinical grounds alone. Full urological assessment may be necessary to eliminate bladder and prostate pathologies, but the mainstay of functional assessment are urodynamic studies which will be necessary following colorectal surgery.

Since transient urinary difficulties are not uncommon following pelvic surgery, urodynamic studies should be reserved for patients who have not improved after 2–3 months. These studies are pressure-flow studies to evaluate bladder function during the filling and voiding stages. A pressure sensitive catheter is placed in the bladder via the urethra to measure intravesical pressure, and a similar one is placed in the rectum or stoma in a patient who has undergone abdomino-perineal excision of rectum (APER),

to measure intra-abdominal pressure. The pressure generated by the detrusor muscle is calculated by subtracting the intra-abdominal pressure from the intravesical pressure.

In colorectal patients with hypocontractile bladders, the findings include low pressure, weak intermittent contractions and incomplete bladder emptying. This is in contrast to patients with outlet obstruction who tend to have high pressure contractions with low flow.

Post-surgical patients with low bladder capacity due to partial cystectomy may demonstrate involuntary contractions, possibly resulting in incontinence. Stress incontinence is associated with leakage during a rise in intra-abdominal pressure (coughing or sneezing) in the absence of detrusor contractions. This is due to a weak sphincter complex as a result of pudendal nerve injury.

Detrusor external dyssynergia occurs when there are involuntary detrusor contractions in association with a spastic external sphincter complex. This very rarely occurs in patients who have had radical pelvic surgery.

In summary, surgeons require a detailed knowledge of pelvic anatomy to avoid nerve injury primarily during rectal excision. Most problems are due to parasympathetic nerve injury causing an atonic bladder for which the only treatment is self-intermittent catheterisation. A bladder which is overactive because of sympathetic nerve injury may respond to anticholinergic medication. Incontinence is rare following pelvic surgery. Many patients will have transient urinary disturbances which will improve, and so urodynamic studies are best left for 2–3 months following surgery.

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CHAPTER

3

Process Delivery in Colorectal Surgical Practice

Thomas Pinkney

RUNNING A SERVICE

Collaborative Approach

In the past, surgery and medicine existed in separate camps and there were structural, political and economic barriers separating the medical personnel who would be needed to provide a colorectal surgery service. Now, however, we are witnessing the provision of system-based medical services involving the integration of multiple specialist teams and allied practitioners.

Most modern units now benefit from having a group of committed physicians, surgeons, radiologists, histopathologists, nurses, dietitians and counsellors working together to create a unified colorectal unit, working closely with basic sciences and oncology. Recently created units can also benefit from having a common geographical location for the various necessary aspects involved in patient care, including ward, theatre, outpatient and endoscopy components.

Having said this, it is not the 'bricks and mortar' but the people that work together to create the right environment that make a successful colorectal surgery unit. Industry, compassion, sensitivity, enthusiasm, teamwork and enquiring minds are some of the attributes needed to make this venture succeed. Most clinicians trained in colorectal surgery are endoscopists, physiologists and diagnosticians; some are surgeons with an emphasis on therapy, whilst others are trained as physicians who play a greater role in endoscopy. Within colorectal surgery, we now see focussed, multidisciplinary teams providing specialist oncology care, services for inflammatory bowel disease, counselling and treatment for functional bowel disease and screening in patients at risk of familial colorectal cancer. These teams also include nurse specialists, physiotherapists, dietitians, stoma care nurses, audit clerks, those involved with nutritional therapy, radiologists, specialist histopathologists and counsellors, anaesthetists and pain control experts.

The Doctor–Patient Relationship

There are few other fields of practice where communication between the doctor and the patient is more important. Many patients are terrified that their symptoms are due to bowel or rectal cancer. For these patients, the thought of cancer is bad enough, but the potential in their mind for needing to undergo treatment that results in the formation of a stoma, and the (largely unfounded) negative connotations associated with stomas is also a significant concern for many patients.[1] Patients may also be aware of the potential for colorectal disease and its treatment to impact on sexual behaviour and function, which will add to their stress.[2,3]

Before seeing the colorectal surgeon, a patient may not only have been suffering from pain, diarrhoea or bleeding, but may also have had episodes of incontinence. Clearly it is both unkind and inappropriate to treat a patient with even a minor colorectal disorder in the same manner as, for instance, a patient with a hernia or gallstones. Patients referred with colorectal symptoms must be adequately assessed so that they may be reassured that

they do not have a malignancy. If malignancy is identified, an honest appraisal of the clinical outcome and its natural history should be provided in collaboration with oncologists, specialist nurses and counsellors where necessary. Most patients will require information and dietary advice. It may be necessary to trace members of a family; most patients will need some form of bowel assessment involving bowel preparation, some will be offered outpatient intervention, others day-case surgery and a subset will need a major operation. For all of these reasons, the method, attitude taken and extent of communication between the doctor and the patient is crucial to the success or failure of treating the whole person.[4–6]

Dissemination of Information

Patients should understand why they may have developed their disease, what we know about the condition, the available therapeutic options and their efficacy, including risks and complications of both interventional and conservative treatment pathways. Booklets and information packs should be available on common colorectal disorders and their treatment, particularly on subjects such as haemorrhoids, fissure, fistula, pilonidal sinus, warts, irritable bowel syndrome, Crohn's disease, ulcerative colitis, restorative options after proctectomy, bowel cancer and diverticular disease. Many patients nowadays use the internet as their primary source of information on their condition, and clinicians should be both encouraging this practice whilst being abreast of the most reliable and highest quality online resources for patient-facing material.

Teaching

Teaching of both undergraduate and postgraduate medical and allied medical staff can be conducted in a stimulating and informative manner if the right environment is created and nurtured. Undergraduates may be able to directly assess a patient and be taken through the most direct and effective process of diagnosis, including the use of endoscopic, radiological and histopathological modalities, before deciding on the optimum therapy and follow-up, all within the one clinical environment. Postgraduate education of a variety of specialists can take place in a variety of contexts including operative skills, post-operative patient management on the ward, new patient assessment and recognition and treatment of the acutely unwell patient presenting as an emergency. At an even more senior level, units should aim to also run regular multidisciplinary case presentation meeting and journal clubs to keep all staff abreast with the latest developments and technology.[7]

Assessment

A great deal of treatment can be delivered on an outpatient or day-case basis. However, not all patients are suitable for this, either because of co-existing pathology, unsupported home circumstances or patient choice. As such, a thorough assessment of the patient and their home circumstances and support structure should be incorporated within the patient evaluation. Checklists to assess the suitability for day-case management of a patient exist in most units and these can often be initially completed by the patient at a suitable stage in the pathway, with input thereafter from pre-operative assessment nurses and clinicians. The ultimate decision about the patient's suitability for day-case surgery should be made by the clinician in charge of their care, with input from an experienced anaesthetist where necessary. Similar checklists may also be used for more major inpatient operations during the pre-assessment phase to identify which patients need to undergo a formal pre-operative assessment by an anaesthetist in an effort to establish coordinated plans for medical management of co-morbidities or to optimise a patient's physiological status prior to surgery. Checklist systems may additionally provide a failsafe mechanism to ensure that patients requiring a post-operative higher dependency or intensive care unit bed are identified to allow the necessary arrangements to be made.

Colorectal Surgery and the Law

Increasingly we live in a world dominated by litigation. The principal areas of potential negligence in colorectal surgery seem to be: (a) inadequate counselling leading to unacceptable informed consent; (b) delayed diagnosis of colonic perforation or malignancy leading to complications or reduced life expectancy; (c) iatrogenic bowel perforation at colonoscopy, laparoscopy or laparotomy, particularly delays in the diagnosis of post-operative bowel perforation or anastomotic leak; (d) failure of diagnosis by clinical acumen, endoscopy or radiology; (e) iatrogenic incontinence following inappropriate colorectal excision or sphincter damage during anal surgery and (f) inadequate training or experience of certain procedures such as laparoscopy, pouch surgery or low rectal excision. We are frequently involved, though not directly liable for post-obstetric incontinence or fistulas, and for bowel damage leading to sepsis, fistulas and sometimes death caused by our colleagues in urology or gynaecology.

Few physicians would ascribe to defensive medicine, but all of us should be aware of potential pitfalls which can be minimised or avoided. Adequate supervision of trainees and appropriate availability, especially for emergency cases being operated upon out-of-hours are key areas that both colorectal units and individual surgeons can endeavour to address. Proper accreditation and ongoing monitoring of performance, but within and outside of theatre, are becoming increasingly important to both improve performance and to deflect any criticism or claims of inexperience or incompetence. Attendance at regular meetings for CME accreditation is now essential in all areas of clinical practice.

Thorough counselling to enable truly informed consent is clearly mandatory in all areas of surgery. The principles of informed consent have been more clearly defined over recent years, and the Royal College of Surgeons of England in their recent *Good Surgical Practice*

guidance document[8] outline the key areas that must be covered in the consent discussion:

- The patient's diagnosis and prognosis
- Options for treatment, including non-operative care and no treatment
- The purpose and expected benefit of the treatment
- The likelihood of success
- The clinicians involved in their treatment
- The risks inherent in the procedure, however small the possibility of their occurrence, side effects and complications. The consequences of non-operative alternatives should also be explained.
- Potential follow-up treatment.

The document also outlines how the patient must be given both enough time as well as enough information to make a fully informed decision. The days of taking consent for an operation from a patient who is in the anaesthetic room immediately prior to surgery must be consigned to the history books. There are various adjuncts available to provide more detailed or more accessible information for patients as well as to allow them to reflect on what has been discussed prior to coming to a final decision. These measures range from basic written information in the form of patient leaflets to specific decision aids tools, educational videos and online resources such as websites and forums.

AUDIT

Clinical audit can be defined as 'a quality improvement process that seeks to improve patient care and outcomes through systematic review of care against explicit criteria and the implementation of change'.[9] Audit is essential for monitoring standards and performance, and can help plan future structures, resource management and education. As such, audit generally forms the core of clinical governance.

A proper clinical audit is more than just a data collection exercise; it should involve measuring current patient care and outcomes against explicit criteria, also called 'audit standards', and have the specific intention from the outset that practice will be assessed and improved. Many authors describe an audit cycle or spiral (Figure 3.1), which can be a useful reminder of the key steps and also serves to remind us that the process never really ends; rather, repeated cycles will aspire to a higher level of performance or quality. Audit should also be transparent, non-confrontational and non-judgemental.[10]

Almost every aspect of clinical care can undergo audit. Some examples within colorectal surgery would include:

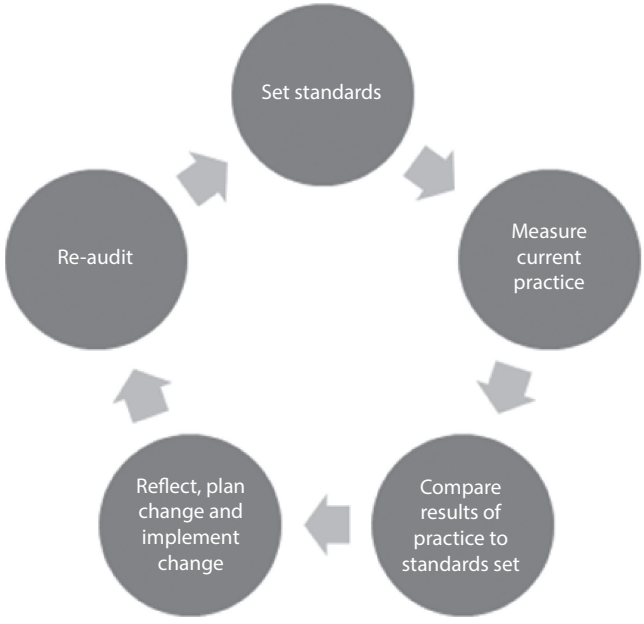
The *structure* of care – e.g. the workload and throughput of a specialist pelvic floor clinic service within a region.

The *process* of care – e.g. patient compliance with self-administered bowel preparation prior to colonoscopy.

The *delivery* of care – e.g. compliance with the tenets of enhanced recovery after surgery (ERAS) after major colorectal resection.

The *outcome* of care – e.g. rates of recurrence after parastomal hernia repair surgery.

Audit can be undertaken at a range of levels, from single centre and regional to national or even global. The breadth and scope of the audit will be largely determined by its intended aims, although logistical and data protection issues can also be important to consider. An increasing number of audits will include the collection of financial data and resource use information as clinicians and health service providers examine the cost effectiveness



3.1 The audit cycle.

of treatments in surgery, which can be particularly relevant in the context of novel surgical approaches or new technologies.

In some countries, central bodies now run large-scale audits which are mandatory for clinicians and units to participate in. Examples include the National Bowel Cancer Audit in the UK and the Dutch Colorectal Surgical Audit in The Netherlands.[11,12] These national programmes can enable high-quality assessments on a large scale, and if they are accurately and reliably completed, may enable comparison of performance and outcomes between units or even individual surgeons. It may be possible to identify outliers whose surgical practice or patient outcomes appear to fall outside of the expected distributions of statistical variability. There is ongoing debate about how such data should be used, including how to undertake risk adjustment to account for the baseline differences at patient and disease level. In England since 2013, these data has also been used to publish the outcomes of have colorectal surgeons undertaking elective bowel cancer resections. It seems likely that other countries may follow suit in due course.

A new type of audit has been gaining popularity over recent years – the multicentre 'snapshot' audit. These studies have the ability to gather large patient numbers in short time periods from many hospitals. They allow exploration of differences in patients, techniques and management across the cohort to identify areas of practice variability that may result in apparent differences in outcome. It must be noted they do not provide true evidence of causality or the impact of a particular variable, because despite complex multivariable regression modelling, they can never fully control for selection bias effects or the hidden confounders and interaction effects inherent in the complex decision-making processes that underpin surgical care. This said, they can certainly be hypothesis-generating and identify areas warranting further study in future controlled prospective research or randomised controlled trials. Good recent examples of such studies include a national audit of appendicectomy in the UK or the ESCP International audit of right hemicolectomy and ileo-caecal resection.[13,14]

CLINICAL RESEARCH

Participation in active clinical research in surgery is thankfully no longer seen as an optional luxury or the final vestige of academic surgical departments. The vast majority of colorectal surgeons now appreciate the central role that clinical research plays in improving outcomes for their patients, and are actively beginning to challenge the historical notions that surgical trials are difficult to conduct and rarely lead to changes in practice.

It has been proven previously in the fields of ovarian cancer and coronary artery disease that patients treated at research-active hospitals demonstrate improved survival rates.[15,16] A similar beneficial effect from research participation has recently been demonstrated for the first time in colorectal cancer.[17] This fascinating study identified a strong independent association between participation in interventional clinical studies and survival for all patients, at hospitals of all sizes and research pedigrees. This positive association exhibited a 'dose-effect', with improvements in patient-level outcomes increasing with both the level of participation (in terms of the numbers of patients in a unit going into clinical trial) and the number or years of sustained participation from that unit.

These findings should serve to further boost the will and determination to create and participate in clinical research in surgery, but there remain major challenges and significant hurdles to overcome. These are a mixture of both genuine methodological challenges peculiar to surgery alongside some historical, environmental and logistical factors.

There is increasing recognition that surgery itself is a complex intervention, made up of multiple components that can act independently or inter-dependently to impact on outcomes.[18,19] These components generally cannot be separated to identify the salient element(s) of interest, in contrast to most pharmacological interventions which can readily be unpicked and defined.[20,21]

In a trial that aims to test whether an in-theatre intervention can improve individual patient outcomes, in addition to the main intervention under study, there will be multiple concomitant interventions and actions undertaken before, during and after the operation that could individually or collectively influence results. Examples relevant here could include pre-operative patient behaviour (such as bathing, skin shaving, nutritional status), anaesthetic management and surgical technique, as well as post-operative ward routines (including dressing removal, staff handwashing, etc.). There is also another group of contextual factors relating to the local setting in which the operation/intervention takes place that can impact upon outcomes, such as throughput rates, operating theatre policies and ward geography.[22] Finally, the complex intervention of surgery is ultimately delivered by individual practitioners and the inherent variabilities in expertise, skill, decision making and experience can all influence outcomes.[21]

Other specific challenges encountered in the design, running and analysis of interventional trials within surgery include technology stabilisation and learning curve/expertise issues.[23,24] These may also be inter-related, and whilst these can sometimes be overcome by performing a well-designed trial at the correct moment in time, this can be difficult to anticipate and accommodate. The balancing act of waiting until the development of a technique or technology has stabilised enough to allow a meaningful and high-fidelity evaluation has been described as 'Buxton's Law' – 'It is always too early [for rigorous evaluation] until it's suddenly too late'.[25] Some argue that a non-randomised cohort study is needed early in the developmental evolution of the intervention to refine the procedure and overcome the learning curve phase. However, the risk of delaying definitive evaluation of the technique is that it becomes widely adopted by surgeons and clinical equipoise is lost.[26] To undertake a trial after this point becomes increasingly challenging.[27]

Historical, Cultural and Logistical Challenges in Clinical Surgical Research

The master-apprenticeship model in surgery has long been widely used as the central method for dissemination of knowledge in surgery. Many surgical procedures and techniques are considered 'standard' without ever having gone through any formal or rigorous evaluation. Development of surgical practice is largely experiential, and this persists in the development of new surgical techniques or procedures.[28] These generally come from a single surgeon or a small group of surgeons, who might employ the new procedure on their patients, observe outcomes (with variable degrees of rigour) and then report their findings. This methodology does not allow comparison of the new procedure against others, or against non-surgical options. There is an inherently judgemental process of comparing the new technique with the old, to assign a perceived difference in outcomes (or speed, or cost) that determines whether or not the new method is accepted.

It must be noted that this ethos of active innovation is not surprising, given that surgeons are specifically trained to undertake constant situational assessment, dynamic decision analysis and improvisation during each and every operation. Riskin described this well: 'most surgeons innovate on a daily basis, tailoring therapies and operations to the intrinsic uniqueness of every patient and their disease'.[29] As such, these skills are important to nurture to improve patient outcomes, but can be potentially deleterious when it comes to the staged and controlled introduction and evaluation of advances in clinical surgery.

Modern surgical practice has moved towards the development of efficient patient pathways. In elective surgery, these include the majority of patients being admitted on the morning of operation, use of minimal access techniques and enhanced recovery after surgery (ERAS) programmes to reduce lengths of stay. Together, these organisational changes decrease the ability of the research team to access patients prior to surgery or enter them into trials and can make follow-up more challenging.

The final issue, and perhaps most importantly, has been the lack of experience and infrastructure in surgical research, reflecting a poor funding history, itself secondary to the poor quality of a large proportion of previous surgical research.[30]

This said, in many countries, there now exists a much healthier atmosphere for the conduct of prospective clinical research in surgery, bought about by a combination of various factors, including enhanced interpersonal communication between researchers via social media and other rapid-access online systems, improved funding opportunities for surgical research, a younger generation of more evidence-driven clinicians and most importantly an emerging environment of national and international collaboration, as witnessed by the large-scale projects outlined above which have all coalesced over the past five to ten years. We are on the verge of a tide-change in clinical research in surgery, and colorectal surgery is best-placed to lead this important charge.

CLINICAL ASSESSMENT

Despite the numerous advances in radiological assessment tools and the continuing development of new biomarkers from both blood and stool samples, the mainstay of accurate diagnosis in colorectal surgery remains a careful and thorough history and examination. The majority of colorectal conditions can be diagnosed at the initial consultation if the clinician is equipped with the ability for judicious clinical evaluation. When coupled with the availability of a myriad of diagnostic tests and a range of interventions that can be administered in an outpatient setting, such as anorectal biopsy, rubber band ligation of haemorrhoids and rigid sigmoidoscopy, it means that a reasonable proportion of patients can actually undergo complete diagnostic evaluation and personalised curative therapy at their first colorectal clinic attendance. Even if this is not possible, a tailored and efficient investigation plan will be created after a careful initial consultation.

HISTORY

Many anorectal disorders present with a very characteristic pattern of symptoms and signs. This means that an individual's diagnosis is often already strongly suspected after the history-taking stage, with examination and/or investigations only serving to confirm the diagnosis or instigate treatment for it.

A comprehensive history, paying particular attention to the patient's own description of symptoms, is essential. In discussion of the primary presenting complaint, details of the key colorectal symptoms – pain, bleeding, altered bowel habit, incontinence, swelling, discharge and irritation – should also be covered. A brief obstetric, gynaecological and urinary tract history should be taken. Exploration of family history is also essential, particularly enquiring about any family members affected by colorectal or lynch-associated cancers or inflammatory bowel disease.

Thorough documentation of previous gynaecological, urological, anal and abdominal operations must be recorded. A list of risk factors for anaesthesia and contraindications for day case surgery should be checked: hypertension, diabetes, ischaemic heart disease, previous cerebrovascular accident, renal dysfunction, valvular heart disease, epilepsy and others. Co-existing medical therapy, particularly use of anticoagulants, diabetic medication, anticonvulsants, antihypertensives and immunosuppressants should be recorded. Social circumstances should also be assessed, including the level of support, if any, needed for the patient to complete their own activities of daily living such as washing, dressing, cooking and

shopping. This gives an estimation of functional status and can be important both in making personalised treatment decisions and in the planning of post-operative discharge arrangements and support requirements.

Some symptoms must be explored in some depth. These are discussed in turn:

Bleeding

Bleeding is one of the commonest symptoms encountered in colorectal clinic, but is always worrying to the patient. The relationship between bleeding, defaecation, straining, scratching, prolapse, constipation and diarrhoea is noted as is the colour of the blood loss and its presence in relation to the stool. The relationship of blood loss to pain or altered bowel habit needs to be sought.

Whether blood is on the surface or mixed with the faeces provides a pointer to the pathology. A bleeding source at the level of the anal canal typically produces bright red blood that is often only seen on the toilet paper, although may drip or even squirt into the toilet pan. Bleeding originating from within the colon or rectum produces a blood of variable colour and consistency, depending on the site of the pathology, the colonic transit times and the rate at which blood is being lost. Colonic bleeding is unlikely to produce the fresh red blood typical of an anal source unless the bleeding is very brisk, which would normally be evident by a concurrent and acute deterioration in the patient's physiological and haemodynamic status. Colonic bleeding may also result in mixing of the blood within the stool and other symptoms such as abdominal pain or altered bowel habit. Bloody stools combined with significant diarrhoea, mucus and abdominal pains may suggest inflammatory bowel disease.

Generally speaking, the darker the blood, the more proximal the source is likely to be. Right colonic or caecal pathology will tend to result in altered and very dark red blood, whereas melaena is the passage of black, tarry stools, which have a characteristic and very offensive smell due to the presence of digested blood; it generally indicates a proximal gastrointestinal bleeding source.

It can be very difficult for a patient to quantify the amount of blood being lost and a small amount of blood can go a long way – a few drops of blood will turn the water in the toilet to red.

Abdominal Pain

Abdominal pain is an important symptom and the clinician will need to know its site, whether it is related to eating, what relieving factors there are, whether it is constant or colicky and whether there is relief from posture, defaecation or medication. Duration of symptoms must be recorded but severity is difficult to quantify in an objective fashion.

When considering abdominal pain, the clinician needs to recall their knowledge of the embryological derivation and neuroanatomy of the gastrointestinal structures and peritoneum. This particularly relates to the poorly localised and midline-based pain, often associated with nausea, that results from irritation or stretch upon the visceral peritoneum overlying the abdominal organs. The height of this vague pain, in the epigastric, umbilical or hypogastric regions, reflects the embryological origin of the affected viscus – being foregut, midgut or hindgut respectively. In addition, pain from the sigmoid colon may radiate to the flank or lower back, and rectal pain may be felt in the sacral region. Continuous abdominal pain, especially of a sharp and well-localised nature, signifies irritation of the parietal peritoneum from any number of sources. Physical examination of the abdomen will allow the clinician to determine presence of peritoneal signs, as well as yield other clues such as masses or organomegaly to help elucidate the underlying diagnosis.

Anorectal Pain

Anorectal pain is a common symptom seen in colorectal clinics and it can be very disabling for the patient. Careful exploration of the nature, timing and characteristics of this pain can often yield a likely diagnosis prior to any attempt at examination. Information should be sought on the relationship between pain and defaecation, posture or sexual activity, as well as any radiation.

Anorectal pain that is exacerbated during and after defaecation is often due to a fissure. This pain is often described as a 'cut-glass' sensation and can lead to the avoidance of defaecation. Unrelenting and throbbing pain, associated with a palpable perianal lump, is generally due to a thrombosed external haemorrhoid. Pain accompanied by local swelling and tenderness of the perianal tissues, especially alongside fever or signs of systemic sepsis, may suggest a perianal or ischiorectal abscess.

It is worth remembering that rectal malignancy rarely causes anorectal pain, unless the lesion invades the anal canal, sphincters or sensate tissues distal to the dentate line.

Change in Bowel Habit

Details of bowel habit are best ascertained by encouraging the patient to provide the history spontaneously. There is a significant variation in the 'normal' bowel pattern, so a change from typical activity is the only relevant metric. When taking a history in this area, factors influencing frequency and some details of consistency, stool characteristics and defaecatory difficulty should be sought. A history of straining, self-digitation, rectal sensation, urgency and assisted defaecation by perineal or vaginal pressure may provide valuable information about the pathophysiological problem.

A change in bowel habits is important to always ascertain as it is one of the commonest symptoms seen with a colonic neoplasm. The combination of a change in bowel habits alongside colonic-type bleeding is a particularly strong indicator of the presence of a serious underlying pathology.

Incontinence

Information on incontinence must be asked as it is rarely volunteered. A distinction must be made between the

patient being truly unaware of passing stool and urgency. Similarly, it is essential to distinguish soiling from true incontinence. Frequency of incontinence and the relationship between it and stool consistency and lifestyle helps to define the severity of the problem. The relationship of symptoms due to obstetric, gynaecological and urinary tract symptoms and their treatment must be included.

Other specific proctological symptoms which will need to be explored include discharge, soiling, irritation and prolapse.

The interview may involve relatives and friends; some questions are extremely personal and should only be discussed on a one-to-one basis. Above all, this conversation must be undertaken in a place where there is privacy, available counselling and a relaxed environment.

EXAMINATION

General Considerations

The way in which the history, and particularly the examination, is conducted often sets a seal on the entire future communication process. The patient must be made to feel at ease. It is often helpful for the clinician to reassure the patient prior to examination that their symptoms are most likely due to a benign condition (if appropriate) and not cancer. This can significantly decrease anxiety and make the entire examination process more likely to be completed successfully.

The room should be clean but not too clinical, well ventilated and warm with adequate lighting. The couch should have height and backrest adjustment, and there should be a stool on which the doctor can sit during examination if wanted. A hand-basin for the patient and doctor to use is necessary, and there should be separate examination and treatment trollies. The patient should be left alone to undress and, if possible, given a light bathrobe to wear. He or she must be covered when lying on the couch.

The first part of the examination should help to further reassure the patient whilst general clinical information is obtained. The clinician should make the patient feel at ease whilst checking for anaemia, cyanosis, clubbing, jaundice and lymphadenopathy and inspecting and palpating the abdomen. During abdominal examination, all four quadrants should be carefully palpated superficially, before systematically examining the nine zones of the abdomen with deeper palpation. Abnormal masses must be evaluated, and their characteristics and anatomical origin ascertained. The liver and spleen must be specifically examined for abnormal enlargement, before moving on to ballot both kidneys. Groins should be examined for the presence of herniae or lymphadenopathy. Auscultation of the abdomen is rarely useful except in the setting of possible acute bowel obstruction when absent or high-pitched 'tinkling' bowel sounds can be a clinical sign of note.

Position for Anorectal Examination

Views differ about the best position for the anorectal assessment. It could be argued that more information can be obtained in the knee–elbow position; however, most patients find this position undignified and will not readily allow the examination to be repeated. By contrast, the left lateral position enables most conditions to be diagnosed with all except the patient's perineum covered.

The patient lies on the left side of the examining table or bed with buttocks protruding over the edge, hips flexed, knees slightly extended and right shoulder rotated anteriorly. The examiner may sit or stand depending on the height of the table or bed. Although this position is the easiest for the patient, it is not as convenient for the examiner as the prone position. It is, however, often the only attainable position in the very obese or immobile patient. There is no evidence to suggest that the examination position influences the ability to successfully pass a sigmoidoscope or undertake useful and complete digital rectal examination.

Inspection

Inspection of the perianal region is critical and may reveal scars, a fistula, a fissure, tags, a patulous anus, vaginal and rectal prolapse or dermatological problems (including pruritic changes). The position of the perineum at rest is noted, as is the movement of the perineum in relationship to the ischial tuberosities during pelvic floor contraction and straining. During straining, a rectocele, haemorrhoids and anal polyps, intra-anal warts or a rectal prolapse may become visible. Parting of the buttocks may reveal an anal fissure; often a sentinel skin tag at the inferior aspect of a fissure can provide a further clue. If the clinician suspects a rectal prolapse, it may be necessary to examine the patient during straining on a toilet.

Rectal Examination

If a satisfactory and reasonable comfort is to be achieved, thereby obtaining the maximum information, it is essential to inform the patient continually of what is to be expected and what is happening. Rectal examination may be a frustratingly unsuccessful experience if proper explanation is not provided, particularly in view of the patient's understandable reluctance to submit to such an unpleasant intrusion. Having applied a water-soluble lubricant to the gloved index finger, the pulp of the finger should be placed gently over the anal orifice and pressure exerted until the sphincter relaxes, allowing the finger to enter the anal canal and rectum. The anal canal and rectum and their surrounding structures should then be examined in an organised manner. This examination should usually be combined with a vaginal examination in women.

First, the resting tone of the anal sphincters is assessed, then the presence of scars, induration, local pain and discharge. The patient is then asked to contract the sphincters and pelvic floor maximally to gauge their activity, degree of movement and position in relation to

the rectal ampulla and vagina. The rectovaginal septum must be carefully palpated from both sides. Deeper palpation is needed to feel for the prostate and most rectal tumours. The clinician should then sweep the examining finger from anterior to posterior, consciously thinking of a possible lesion that might be present. The conscious thought process is emphasised because too often this phase of examination is simply performed as a routine. In the case of a tumour, its position, size and characteristics, especially whether it is polypoidal, sessile or ulcerated, together with its depth of bowel wall involvement, mobility, fixity and relationship to local anatomy, must be recorded, preferably on a chart. Finally, as the finger is withdrawn, the presence of additional anal pathology is noted (e.g. hypertrophied papilla, thrombosed haemorrhoid, stenosis, scarring, etc.).

PROCTOSIGMOIDOSCOPY

The current author almost always passes a rigid sigmoidoscope at the completion of the digital examination in the unprepared patient provided there is no painful anal lesion. In previous years, a reusable metal sigmoidoscope would have been used. but these have now been consigned to the history books in favour of single-use plastic sigmoidoscopes. These not only afford a better view as the light can be conducted down the clear plastic walls of the scope to the tip, but they also avoid any risk of infection or cross-contamination, negate the need for any washing or cleansing of instruments and have the added benefit of not needing to be warmed prior to insertion. Some newer products are now available which further expand the model by incorporating a single-use battery-powered light source within the handle and disposable bellows, which means the entire kit is disposed after use, at only a marginal increase in cost which may be justified by the reliability and increased flexibility offered.

Method

The limit of a 25 cm instrument can usually be reached in 40% of examinations, and in over half of these the presence of stool does not prevent adequate inspection of the anorectum. Bowel preparation is not normally necessary, although a digital rectal examination should always precede instrumentation.

The well-lubricated sigmoidoscope is inserted and passed to the maximum height under vision as quickly as possible without causing discomfort. Air insufflation may be of value in demonstrating the lumen and is of even greater benefit in visualising the mucosa, but it should be kept to a minimum because it tends to cause pain. Most information is obtained as the sigmoidoscope is withdrawn, when the entire circumference of the bowel wall can be inspected.

The sigmoidoscope is one of our most valuable diagnostic tools and the rigid sigmoidoscope is the best instrument available for evaluation of the rectum. The purpose of the examination is to identify polyps, benign strictures, vascular abnormalities, malignancy and colitis. Any visible lesion or abnormality should be biopsied, and a palpable lesion can be scraped for cytopathology and biopsied. In patients with diarrhoea, the stool can be sent for microbiological assessment and culture from the clinic.

Biopsy

Various biopsy forceps are available. The best ones are probably the Lloyd-Davies biopsy forceps which have very strong blades. The lesion is grasped with the forceps, which is then rotated to prevent bleeding when shearing the mucosa. Cytology smears may be prepared from potentially malignant lesions to gain an immediate diagnosis. Random biopsies for inflammatory bowel disease should always be performed on the posterior rectal wall and from a valve of Houston where possible.

PROCTOSCOPY, VAGINAL SPECULUM EXAMINATION AND OUTPATIENT THERAPY

Proctoscopy

Proctoscopy allows thorough inspection of the anal canal at rest and during straining to exclude an internal opening of a fistula, a discharging intersphincteric abscess, haemorrhoids, condylomata acuminata and a chronic fissure.

There are a number of proctoscopes, most of which have fittings for a fibreoptic light source. As with sigmoidoscopes, the wide variety of metal proctoscopes of varying sizes and roles have generally been replaced with single-use plastic counterparts, although a good clinic should have a variety of different diameter scopes available to suit all patients and conditions. Similar single-use proctoscopes with an incorporated battery-powered light source that are fully disposable also exist and are being used by some. Proctoscopes with a slot removed from one side of the instrument to allow a side view of the anal canal are available. These instruments are perhaps more commonly used in the operating theatre to aid in the undertaking of interventional procedures on haemorrhoids and are now rarely used in the diagnostic setting. When rotating the anoscope around the circumference of the anal cavity, it is helpful to reinsert the obturator. The site of any pathology should be recorded.

Vaginal Speculum Examination

A speculum examination of the vagina is sometimes necessary and carried out to exclude a fistula, to assess uterine descent, to evaluate a cystocele or rectocele and to swab a chronic discharge to exclude specific causes of vaginitis.

Outpatient Therapy

After a complete clinical assessment, certain disorders can be treated at the same time as the initial consultation, provided the patient has been informed and is agreeable. Thus, rapid outpatient therapy is eminently feasible at the first consultation in many cases. Outpatient or office procedures include: polypectomy, cryotherapy, biopsy, rubber-band ligation of haemorrhoids, application of podophyllin for condylomata and curettage of a pilonidal sinus.

Different organisations have their own specific facilities. In some countries, the culture is geared to day-case surgical procedures usually not undertaken at the time of the first consultation but booked on a minor or day-case list. With the provision of a minor operating theatre for colorectal surgery, the range of outpatient therapeutic options increases considerably.

PHYSIOLOGY

This is covered in Chapters 2 and 3.

ENDOSCOPY

This is covered in Chapter 26 in Screening, Chapter 22 in *Endoscopy and Polyp Management* and Chapter 55 in *The Diagnosis of Inflammatory Bowel Disease*.

RADIOLOGY

This is covered in Chapter 28 in *Ultrasound in Neoplasia*, Chapter 29 in *MRI and CT in Neoplasia*, Chapter 30 in *PET and Nuclear Medicine in Neoplasia*, Chapter 47 in *Diagnosis in Diverticular Disease*, Chapter 55 in *The Diagnosis of Inflammatory Bowel Disease* and Chapter 56 in *Imaging in Inflammatory Bowel Disease*.

MULTIDISCIPLINARY APPROACH

The concept of a multidisciplinary team (MDT) that drives patient care and joint clinical decision making has been increasingly adopted in the management of complex diseases over the past decade. The basic premise of MDT care is to involve all key professional groups in the management and discussion of diagnostic dilemmas or conditions where more than one viable treatment pathway exists. These experts are bought together in a common forum where the individual cases are discussed and coordinated decisions or recommendations made.

The model originated within the cancer setting; it was further consolidated and strengthened after the publication of the Calman–Hine report in 1995 which extolled the virtues of a specialist rather than generalist model for improving outcomes in the care of cancer patients.[31,32] Over the subsequent years, further efforts have been made to standardise the running and organisation of cancer MDT meetings, including mandating a set of core members, to ensure both the smooth running and the reliable and reproducible ability to make high-quality decisions for patients.

The concept has percolated into non-cancer specialist settings within colorectal surgery, most notably in inflammatory bowel disease (IBD), although they also are increasingly utilised for functional bowel disease, endometriosis surgery involving the rectum and urinary tract, complex fistula and pelvic floor, as well as those with co-existent urogynaecological conditions. A UK national audit showed that around three-quarters of units treating patients with IBD undertake weekly dedicated MDT meetings for these patients,[33] although a recent national quality standard has been published which mandates that all units treating IBD patients should be running a regular MDT programme,[34] so this proportion should rise up to 100% in the near future.

FACILITIES

Ideally, there would be a single self-contained colorectal unit comprising an outpatient facility, counselling rooms, follow-up and screening areas, adjacent to an endoscopy suite, radiology, oncology and anorectal physiology rooms. There would be purpose-built recovery and waiting areas, a dedicated day-case unit and theatre offices, an operating theatre suite and the ward for inpatient care. The entire network should be linked by telephones and computers. The colorectal surgery unit should incorporate changing areas, toilets and teaching and seminar rooms. The plan should provide offices for physicians, surgeons, nursing staff, stoma care nurses, dietitians and, if possible, dedicated radiologists, histopathologists and a psychologist. This vision is rarely achievable in all but the most modern and purpose-built facilities, but we can strive to create functioning sub-units of this integrated vision, as outlined below.

Outpatient Area

There should be sufficient waiting room space with both educational and entertainment options for patients waiting to be seen. Separate rooms will be needed to undertake pre-consultation observations on patients, such as their height and weight, and there should be dedicated areas to lay up trolleys and store equipment and dressings. There will need to be plenty of multipurpose clinic rooms that can be used interchangeably for patient assessment and consultation, examination, counselling, specialist nurse and stoma therapist use and wound assessment or dressing changes. There should be a robust

system for patient administration including check-in and repeat booking arrangements. Ideally, there would be a seminar room which is fully equipped for teaching. Booklets should be available, preferably in a reading room with video and information technology facilities.

Diagnostic and therapeutic trolleys in clinic should contain a reliable light source with proctoscopes, sigmoidoscopies, rubber band ligation devices and biopsy forceps. Other items such as local anaesthetics, dressings, silver nitrate sticks and similar sundries can be useful to keep in each clinic room. Clearly, sheets, wipes, tissues, lubricant jelly and gloves must be in good supply and regularly replenished. Many clinics nowadays have a separate area and dedicated staff to undertake phlebotomy services.

Endoscopy

There must be a large waiting area; several parallel endoscopy suites; good changing, washing and lavatory facilities; a sterilisation area; a room for bowel preparation; a sluice; linen cupboards; a patient trolley store and a recovery area. Video teaching bays should be part of the facility since explanatory video programmes are useful for those patients who have never had an endoscopy before. All modern units will use electronic reporting systems now which are invaluable both to compare against for repeat surveillance or assessment endoscopies, and to access in multidisciplinary meetings or the outpatient suite during patient assessment or consultation. Many endoscopy units now also have live video linkage to a teaching or seminar room which enable high-quality assessment and training of those professionals undertaking the endoscopy examinations.

Ward

The ward area should be bright, light and attractively decorated. Ideally, this zone should include the data manager's office, the admissions unit and the secretarial and academic offices with a library and a small lecture theatre or seminar room. There should be office space for stoma care nurses, the nursing staff and other paramedical staff. There should be a room in which the staff can relax. Hard copies of patient notes, where utilised, should also be easily available, and storage facilities for appliances, stationery, linen and toilet requisites should be supplied. The patients will need a waiting area and a reading room. There should be an area for preadmission registration and clerking.

It may be wise to incorporate some flexibility over the use of beds. Shared beds with other general surgical services, particularly the upper gastrointestinal team, are often utilised, which can offer a degree of elasticity in bed occupancy, although this must be offset against the need for a more generically trained nursing staff who must be able to deal with patients who are suffering from a wider range of conditions or who have undergone a greater variety of increasingly specialist operations. Some units also aim to close part of the ward over the weekend, and run a section of their service on a 5-day basis from Monday to Friday. This can then provide a useful buffer for emergency admissions and allows intermediate-level operations to be performed on patients who would not be suitable candidates for day-case surgery.

The main ward area will need a central nursing station or stations and plenty of lavatories, showers, baths and washing facilities. Most beds will be in single- or at most four-bedded cubicles. Most units will need a small high-dependency unit in case there are patients who require a higher level of monitoring or increased nursing care and observation. There is also an argument for placing all patients needing parenteral nutrition in a specific area. There should be close access to an intensive care unit to accommodate those patients needing ventilation or cardiovascular support. We prefer to admit all emergencies to a surgical assessment and triage unit for resuscitation, investigation and observation; many can be discharged the following day, whilst those needing operation or admission are transferred to the colorectal unit.

Operating Theatres

There should be separate day theatre, emergency theatre and elective theatre suites. A dedicated colorectal elective theatre will be able to stock specialised instruments, stapling devices, trays and a purpose-built operating table. Furthermore, the staff can be trained specifically in the disciplines and nuances of colorectal procedures. Separate anaesthetic and recovery bays, stores and offices are incorporated into the theatre suite. Many national bodies now mandate the use of an internally linked electronic system for writing, storing and accessing operation notes after surgery.

Day-Case Unit

There should be a dedicated day-case unit, which must include operating theatres, anaesthetic rooms and a recovery area. Patients will generally come to a separate and dedicated reception and admissions area; they will then be situated on a devoted ward area both before and after their operation. There is considerable teaching potential in a day-case unit, for both more junior surgeons in generic and simple surgical procedures and basic skills acquisition, as well as for more senior trainees who may be able to attend specialist theatre lists to gain exposure to advanced specialist techniques. We often run dedicated day-case lists for minor operations to assess and treat perianal Crohn's sepsis for this reason. A robust system must be incorporated to provide primary care physicians and nurses with information about the procedure that has been undertaken on their patient.

Patients should only be booked into the day unit after they have been carefully screened by the medical and nursing staff to ensure they are fit for day-case surgery and that their home facilities are adequate for recovery purposes. A drug history is crucial, since diabetics, those on anticoagulants and patients receiving antihypertensives and cardiotropic agents may not be suitable. Patients with unstable epilepsy or those suffering from asthma will need to

be carefully screened. Thus, there must be a preadmission assessment service for day-case operations.

Emergency Admission

At least one-fifth of colorectal cancers still present as emergencies with obstructive symptoms, pain, advanced disease or perforation. The outlook in such patients is poor; a UK study identified that the mortality in this group was around four times higher than those undergoing elective surgery, with rates identified of 21.7% versus 5.5%.[35]

Similarly, the majority of patients with diverticular disease present with sepsis or obstruction. At least a third of all inflammatory bowel disease present to the front door with acute symptoms. A small number of patients will lower gastrointestinal bleeding will require urgent admission and investigation. Civil violence when it affects the large bowel will also need to be managed through the emergency admission unit.

These patients must all be provided with access to the facilities for rapid resuscitation, early imaging and rapid surgical treatment to try and give them the best chance of an acceptable outcome. It is therefore essential that a colorectal unit should be in easy access of the emergency facilities with a dedicated intensive care unit and an emergency operating theatre suite that works 24 hours per day, seven days a week.

STOMA CARE

Stoma care is a recognised and important component of colorectal surgery. Despite this, the need for appropriately trained nursing personnel to supervise the management of stomas in hospital and the rehabilitation of patients into the community can still sometimes be an optional luxury and is periodically threatened by funding constraints in some centres or health services.[36] With the majority of patients with a stoma experiencing complications at some point,[37] the role is clearly vital from both the patient and the service provider point of view; a good stoma care nursing service will significantly improve the quality of life for patients with a stoma whilst providing financial remuneration in terms of saved inpatient bed days, repeated hospital attendances and even reoperations in some cases.

Stoma care clinical nursing specialists practice at an advanced level and work autonomously to deliver a high standard of care. The key aspects of the role are clinical, education, research and audit, consultancy and management.[38,39]

Details of stoma care management can be found in Chapter 8. The particular issues regarding stoma care management in the emergency setting are covered in Chapter 79.

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CHAPTER

4

Perioperative Care

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Pre-operative assessment is considered essential in the preparation of patients for surgery. The process starts informally from the point of contact with the patient's family doctor, who, when referring the patient for consideration for surgical intervention, should make a general assessment of the patient's overall fitness for anaesthesia and surgery. The surgical consultation is similar: the patient is briefly assessed for their fitness for surgery, whilst at the same time making an assessment of the surgical condition. The process of obtaining informed consent for the operation now requires rigour with an explanation of the condition, management options available and the risks and benefits associated with each option. The concerns of the patient need to be addressed in a clear and understandable manner. Once an agreement to proceed with surgery is made, the patient is then referred to the pre-operative assessment clinic for a formal, objective assessment of their risk factors and fitness for surgery.

A dedicated team of trained staff should manage the pre-operative assessment clinics. The service is often nurse-led with a consultant anaesthetist available for advice and for the assessment of high-risk patients. The nurses assesses the fitness of the patient for surgery based on protocols and guidelines discussed and agreed with the surgeons and anaesthetists. There is a general screening questionnaire to assess the patient's general fitness, medical co-morbidities, drug history, smoking and alcohol history, personal or family history of significant issues such as bleeding disorder or anaesthetic complications, as well as the patient's social circumstances. The screening process is designed to detect any cardiorespiratory problems or medical co-morbidities (such as asthma, diabetes, anticoagulation therapy, etc.) that would require further investigation and optimisation prior to surgery, as well as any social issues which may affect discharge planning. The process therefore helps facilitate same-day admission, early discharge planning and arrangement for day-case procedures or pre-emptive arrangement for the involvement of critical care facilities. Pre-operative assessment also provides an opportunity for the patient to ask further questions about his/her condition and the proposed treatment option as well as an opportunity for the patient to voice any concerns prior to the day of surgery. This helps ensure patients are both well-informed and well-prepared for their operations and post-operative recovery. A good and effective pre-operative assessment will therefore minimise any unplanned cancellation on the day of surgery and waste of theatre time and hospital resources, as well as avoid any unnecessary distress or inconvenience.

ROUTINE PRE-OPERATIVE TESTS FOR ELECTIVE SURGERY

The required pre-operative tests are dependent on the patient's risk factors and the type of surgery they are undergoing. For patients who are undergoing minor procedures (e.g. excision of skin lesion, examination under anaesthesia), pre-operative investigations are often not required apart from patients who are in the high-risk categories (ASA 3 or 4) where renal function and ECG tests may be considered. For major operations, which include all colorectal resections, pre-operative tests of full blood count and renal function are performed for all patients. For high-risk patients, older patients (aged above 65), patients on anti-hypertensives, diuretics and anticoagulant therapy, other blood tests such as coagulation screen, ECG and CXR should be carried out.[1] In our

practice, patients having major complex colorectal resections will undergo pre-operative cardiopulmonary exercise testing (CPEx) for an objective evaluation of their functional capacity to undergo surgery. The results are interpreted by a consultant anaesthetist to determine the patient's suitability to proceed to surgery and the need for post-operative critical care facility.[2]

RISK ASSESSMENT

The risk of an operation is multifactorial. It is influenced by the complexity of the surgery, the nature of the pathology, the urgency of the operation (elective versus emergency) and patient risk factors. Higher risk factors include major complex thoraco-abdominal surgery, procedures for cancer especially in the presence of nodal or distant metastasis, emergency operation and patients who are older and who have poorly controlled co-morbidities.

With an ageing population, many of our patients on whom we operate are of advanced age. Age is often associated with co-morbidities such as hypertension, cardiovascular problems and diabetes. The operative mortality risk for patients above age 80 is double the mortality risk for patients below age 70, and age is associated with an increased risk of post-operative cardiorespiratory complications.[3] Interestingly, the mortality risk for elderly patients undergoing surgery is not different from the mortality risk of their age-matched controls in the general population.[4] Therefore, old age itself is not a contraindication to surgery in patients who require surgery, but a reflection on the need for careful pre-operative assessment and planning for these patients.

Another common problem facing our population is obesity, although there are studies that suggest that obesity is not associated with increased mortality. It is felt that such patients have a chronic low-grade inflammatory status which enables them to mount an appropriate response to the surgical trauma and physiological stress.[5] Nevertheless, obese patients often have underlying diabetes, hypertension and obstructive sleep apnoea. They are at higher risk of wound complications, post-operative septic complications, reduced mobility and venous thromboembolism. Extra precautions should therefore be taken in this group of patients, with vigilant consideration of antibiotic prophylaxis, early physiotherapy input for post-operative rehabilitation and consideration of extended thromboprophylaxis to reduce the risk of thromboembolic disease.[6]

There are many risk predictive models, such as the simple ASA grading system or more sophisticated models such as POSSUM, P-Possum, CR-Possum and APACHE[7] (http://www.riskprediction.org.uk/). These models are useful in providing an objective risk assessment for informed consent, peri-operative planning and allowing meaningful comparison of surgical outcomes within a unit and between units. It is, however, important to remember that these models provide estimates of risks, and they do not guarantee outcomes. Some are also known to underestimate or even over-estimate risks of morbidities and mortalities, and should by no means replace our clinical assessment and decision-making process.

ENHANCED RECOVERY

Enhanced recovery, or fast-track surgery, is a multimodal and multidisciplinary approach to surgery. Its objective is to reduce surgical stress response and expedite the patient's recovery, which translates to a shorter hospital stay, quicker return to their baseline status and reduced overall cost. The programme involves integrated pre-operative, intra-operative and post-operative pathways, and for the programme to be successful, it requires participation from everyone involved in the patient's care as well as the patient themselves.

Pre-Operative Preparation

This includes adequate pre-assessment of the patient's fitness for surgery and optimisation of any medical conditions. Patients should also be counselled adequately not only with regards to the surgery itself, but on the anticipated journey from admission to discharge. In patients where formation of a stoma is anticipated, pre-operative stoma education can mentally and physically prepare the patients for the management of their stoma. Such pre-operative education encourages patients' active participation in their recovery process.

The practice of prolonged pre-operative fasting and mechanical bowel preparation is no longer routine practice. The merits of mechanical bowel preparation are discussed in a later section. For elective procedures, patients are required to fast for a minimum of 6 hours for solids and 2 hours for clear fluids to reduce the risk of gastric aspiration. In enhanced recovery programmes, patients are given pre-operative carbohydrate loads up to 2 hours before surgery in order to reduce the surgical stress response and minimise the post-operative effect of insulin resistance. We have previously compared oral carbohydrate load to either fasting state or pre-operative supplementary water which has shown an earlier return of gut function and shorter hospital stay in our patients undergoing colorectal resection.[8] Although subsequent review has not shown a reduction in post-operative complications, there continues to be a shorter post-operative stay which suggests a quicker return of gut function.[9]

Intra-Operative Strategies

Anaesthesia

The use of rapid short-acting volatile anaesthetic agents (e.g. sevoflurane), opioids (e.g. remifentanil) and muscle relaxants have helped to facilitate early recovery and reduced the need for prolonged post-operative monitoring or high dependency care. The use of regional anaesthesia