Chronic Inflammatory Bowel Disease, Ulcerative Colitis, Crohn's Disease and Colorectal Cancer

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Chronic inflammatory bowel disease is a multidimensional experience that involves a permanent alteration in a person's lifestyle. Chronic inflammatory bowel disease can have a major impact on all aspects of a person's life. Home, social, work and sex lives can be disrupted, which can lead to the impairment of emotional and psychological function, as well as physical debilitation. The consequences of inflammatory bowel disease can be costly in terms of healthcare, both in relation to primary and secondary healthcare settings and to human sufferings (Rowlinson, 1999a).

Contents

Learning Objectives

By the end of the chapter you should be able to demonstrate knowledge of

- Factors associated with the epidemiology and aetiology of ulcerative colitis and Crohn's disease.
- Differences between the pathophysiology and clinical manifestations of ulcerative colitis and Crohn's disease.
- How ulcerative colitis and Crohn's disease are diagnosed and treated.
- Nursing interventions that reflect the chronic nature of the disorders and the potential impact physically, psychologically and socially.
- The role of the specialist nurse.
- The potential complications of chronic inflammatory bowel disease, including colorectal cancer.

Definition

Inflammatory bowel disease (IBD) is an all-embracing term for the chronic inflammatory disorders of the intestine. Ulcerative colitis and Crohn's disease are idiopathic chronic inflammatory disorders of the gastrointestinal tract, characterized by periods of relapse and remission. There is some overlap in their clinical features; the major difference between them, however, is that ulcerative colitis is confined to the large bowel whereas Crohn's disease can occur in any part of the gut, from mouth to anus. Both diseases can have a major impact upon all aspects of a patient's life. There is a significant risk of carcinomatous change in patients with long standing inflammatory bowel disease.

Epidemiology

Ulcerative colitis and Crohn's disease are more prevalent in Europe and North America (Allison *et al.*, 1998). The incidence of IBD varies widely between populations: Crohn's disease appears to be rare in the underdeveloped world, yet ulcerative colitis, although still unusual, is becoming more common. In the West, the incidence of ulcerative colitis is stable at 10 per 100 000 while that of Crohn's disease is increasing and is reported to be 5–7 per 100 000 (Palmer and Penman, 1999). An exception to this trend is in Scandinavia where the incidence of ulcerative colitis has risen sharply (Allison *et al.*, 1998).

In the United Kingdom (UK) inflammatory bowel disease affects between 15 and 30 people per 10 000 of the population (Rowlinson, 1999a). Ulcerative colitis affects approximately 95 000 people in the UK, which is about 1 in 600 of the population and Crohn's disease affects approximately 55 000 people in the UK, which is about 1 in 1000 of the population. Approximately 5500 new cases of ulcerative colitis are diagnosed each year and 3000 new cases of Crohn's disease (National Association for Colitis and Crohn's Disease [NACC], 2001b). The incidence of ulcerative colitis in the UK may be as high as 26 per 100 000

Table 12.1 A comparison of the incidence of ulcerative colitis and Crohn's disease

Ulcerative colitis	*Crohn's disease*
■ Incidence stable in the West ■ Most commonly diagnosed in the 20–40 age group	■ Incidence increasing ■ Commonly presents at adolescence and in young adults. The incidence among children is rising

of the population with a prevalence of 80–120 per 100 000 of the population. The incidence of Crohn's disease is generally considered to be 5–7 per 100 000 of the population with a prevalence of 30–60 per 100 000 of the population (Table 12.1) (Allen, 1999; Long and Cooper, 1997; Clark and Kumar, 1994).

The incidence of IBD is lower in the non-white races and people of Jewish descent are more prone to IBD than non-Jewish people (Clark and Kumar, 1994). Ulcerative colitis begins most frequently in people 20–40 years of age; Crohn's disease commonly presents in puberty and young adults. Young children and the elderly, however, can be affected by both disorders (Curry, 1995). There has been a rise in the incidence of Crohn's disease among children. The first epidemiological study in children to be carried out in England and Wales showed that the incidence of Crohn's disease in South Glamorgan more than doubled in 11 years (Kmietowicz, 1997). A second incidence peak occurs in the seventh decade (Palmer and Penman, 1999). The incidence between the sexes is equal in both diseases (Finley, 1999).

Aetiology and Pre-disposing Factors

The cause or causes are unknown. Immunological and infective agents have been proposed and researched as possible causes of both conditions, but without conclusive results (Finley, 1999). It has been suggested that the pathogenesis of IBD represents an interaction between genetic and predisposing factors, exogenous and endogenous and modifying factors (Travis, 1998).

Aetiology and pre-disposing factors

- Genetics
- Immunological factors
- Microvasculature
- Infection
- Environmental factors
- Stress
- Smoking
- Dietary factors
- Geographical factors

Genetics

There is an overlap of clinical, radiological, endoscopic and histological features. In addition, shared epidemiological characteristics, such as similar distribution with respect to geography, age, gender, race, ethnicity, occupation and social class suggest the possibility that the two disorders have a common genetic basis (Jewell, 1998). While they may share some genetic predisposing factors and immunological mechanisms, the weight of evidence however, suggests that they are genetically and otherwise fundamentally distinct disease processes (Podolsky, 1991).

Familial clustering has been observed and if both parents are affected the risk to their children is very high. Also the first presentation of the disease in familial cases occurs at an earlier age compared with those without a family history (Rowlinson, 1999a). First-degree relatives of patients with IBD have a ten-fold increased risk of developing the same disease. Studies of twins confirm a strong genetic link; the risk is higher for an identical twin of an affected patient than for a non-identical twin. Among monozygotic twin pairs there is a concordance of about 25 per cent for each of the diseases. In families in which two or more relatives have one of the IBDs, the affected first-degree relatives are not always concordant with the same disease (Allison *et al.*, 1998). There appears to be a relationship in some families between ankylosing spondylitis, the HLA phenotype B27 and IBD (Walsh, 1997).

Immunological Factors

The cellular events involved in the pathogenesis of ulcerative colitis and Crohn's disease involve activation of macrophages, lymphocytes and polymorphonuclear cells with the release of inflammatory mediators (Palmer and Penman, 1999). Both food and bacterial antigens have been implicated in the disorders but there is no clear evidence for either (Long and Cooper, 1997). It has been noted that there are deficiencies and abnormalities of the intestinal mucous layer in IBD, with associated abnormality of the mucous glycoproteins. In ulcerative colitis in particular, the immune response is mounted against a specific colon-associated protein. Mucus is an essential component contributing to intestinal mucosal defence and in ulcerative colitis colonic mucus is qualitatively and quantitatively abnormal. There may also be deranged restitution of damaged mucosa in both disorders. These factors may underlie the development of IBD (Rowlinson, 1999a). It remains unclear whether immunological abnormalities are the primary or secondary event in the pathogenesis (Clark and Kumar, 1994).

Microvasculature

The study of the microvasculature of resected gut has given new insight into the pathogenesis of Crohn's disease. It suggested that vascular injury and focal

arteritis were early pathological events leading to micro-infarction. It has also been shown that there is an association between ulcerative colitis and thrombotic events and histological examination of rectal biopsies have detected fibrin thrombin in the mucosa in patients with IBD. Research into the use of anticoagulants is underway (Rowlinson, 1999a).

Infection

No infectious agent has yet been shown to transmit the disease; however, infectious agents have long been considered to be a cause of Crohn's disease (Walsh, 1997). Upper respiratory tract infections, the measles virus and *Mycobacterium paratuberculosis* have all been implicated, although incontrovertible evidence of their relevance has failed to emerge (Rowlinson, 1999a).

Professor John Hermon-Taylor is convinced, however, that a human pathogen, probably the *Mycobacterium avium* subsp. *paratuberculosis* (MAP), causes 95 per cent of Crohn's disease. In Western Europe and North America, subclinical infection with *M. paratuberculosis* is widespread in domestic livestock, and infected animals shed mycobacteria in their milk. *M. paratuberculosis* causes Johne's disease in cattle and both Johne's disease and Crohn's disease are present in Scotland in almost epidemic proportions (NACC, 2001a). It is known that *M. paratuberculosis* can accumulate in the intestine and sit there for years and that it can cause chronic inflammation of the intestine in many animal species.

M. paratuberculosis has also been found in the inflamed tissue of people with Crohn's disease (Hermon-Taylor, 2000). Complete destruction of *M. paratuberculosis* by pasteurization is not assured. Studies have shown that the temperature used for pasteurization does not destroy all the mycobacteria. A study of milk bought at retail outlets, discovered that one in four bottles tested positive for *M. paratuberculosis* (NACC, 1997). *M. paratuberculosis* is quite often present in people's bodies without appearing to cause any ill effects, but Hermon-Taylor believes that drinking pasteurized milk could be a risk for people who are susceptible to Crohn's disease. *M. paratuberculosis* appears to be highly resistant to standard antituberculosis drugs but Hermon-Taylor claims that using new drugs to which MAP is more susceptible can make patients a lot better, some lastingly so. Current genetic research, funded by the Ileostomy Association, is attempting to make a therapeutic vaccine to help the immune system of people with Crohn's disease eradicate MAP themselves (Hermon-Taylor, 2000).

The role of the bacterium is not clear-cut, however, as *Mycobacterium* has not been consistently found in involved tissues of people with Crohn's. The organism has not been shown to appear, persist, or recur with the clinical course of the disease. People with Crohn's disease have been shown to improve if their immune system is suppressed. For example, people go into remission if their T-cell counts are low because of AIDS or a recent bone marrow transplant.

In these cases a *Mycobacterium* infection would worsen, but people with Crohn's improve. The heterogeneity of Crohn's may, however, allow a role for *M. paratuberculosis*, as there may be similar types of Crohn's disease, each determined by various genes. One type may involve this organism. It is also possible that the organism is one of many bacteria that may trigger the inflammatory response in Crohn's disease. It is therefore important to cast the net wider in examining the development of the disease (NACC, 2001a).

Data published by the Royal Free Hospital's Inflammatory Bowel Study Group (IBSG) suggested that measles vaccination in infancy might triple the risk of developing Crohn's disease and more than double the risk of developing ulcerative colitis (Thompson *et al.*, 1995; Wakefield *et al.*, 1993; 1998). However, other extensive research has not shown a link between measles, mumps and rubella (MMR) vaccination and bowel disease and experts from around the world, including the World Health Organization (WHO), have agreed that there is no link between MMR and bowel disease (Yarwood, 2001). Dr Andrew Wakefield, the IBSG leader, has stressed that no changes to immunization schedules should be made until the exact nature of the link has been investigated. He does, however, believe that the research conducted so far cannot simply be ignored. Studies linking prenatal exposure to measles virus and Crohn's disease have also showed conflicting results (Kmietowicz, 1997).

Research published by Scott Montgomery in 1999, found that children who had caught measles and mumps in quick succession seemed much more likely to develop IBD as adults. The statistics indicate the likelihood of developing ulcerative colitis to be seven times greater than normal and for Crohn's disease to be four times greater. The findings did not establish a link between children having measles infection on its own and the subsequent development of IBD. The study looked at viruses caught naturally and not weakened viruses contained in the MMR vaccination and it was not therefore possible to make any conclusion about MMR on the basis of this study. As with any new research, this finding will need to be tested in other groups of people and by other researchers before it can be accepted as a certain cause of IBD (NACC, 1999).

Data collected by Health Promotion England show that when parents turn to health professionals for advice they want clear, consistent information based on facts.

Nursing action points

- Advise clients that
  - □ extensive research has failed to show a link between MMR and IBD.
  - □ measles kills and disables children. In nearly 30 years, more than 500 million doses of MMR have been given in over 90 countries.

(Yarwood, 2001)

The increase in IBD over the past 50 years parallels the increasing use of antibiotics in human and veterinary medicine. Antibiotics can promote the proliferation of toxic bacteria and make them more invasive. It has also been suggested that Crohn's disease is an infectious disease caused by a mutated form of an organism of the normal bacterial flora, which becomes a super germ under constant selection pressure from antibiotics (Hoffman, 1997).

Environmental Factors

Evidence suggests that the cause is environmental. When a population migrates from areas of low to high incidence, the incidence in that population subsequently rises (Long and Cooper, 1997). A seasonal variation of relapses, with the highest relapse rate occurring from September to February has been suggested. This may indicate that microbiological factors are involved in the exacerbation of symptoms as some infectious agents occur with seasonality, peaking in late summer and autumn. Also patients may be less inclined to seek help during holiday periods or have less access to medical help (Rowlinson, 1999a).

Stress

Emotional factors and stress have not been shown to cause IBD, despite the demonstrated link between stress and bowel disorders. Although life stressors have not implicitly been shown to influence the clinical course of ulcerative colitis, the regulatory effects of stress on the neurogenic system and the inflammatory response cannot be discounted (Walsh, 1997).

Smoking

Ulcerative colitis is commoner in non-smokers and ex-smokers and there also appears to be a rebound effect in smokers that quit (Rowlinson, 1999b). Studies have suggested that smokers were more likely to develop Crohn's disease; heavy smokers had a higher risk of relapse and smokers with small bowel involvement had more frequent bowel movements, suffered more pain, were admitted to hospital more frequently and had an increased risk of surgical intervention. There may also be a tendency to arterial thrombosis in patients with IBD who smoke and multifocal gastrointestinal infarction has been demonstrated in Crohn's disease and proposed as a possible pathogenic mechanism (Rowlinson, 1999a).

Dietary Factors

Dietary factors are thought to play a role. Swedish researchers have found that people who eat at fast food restaurants regularly and who eat too much sugar may increase their risk of developing Crohn's disease. New

unnatural substances have been introduced into the diet of people in the West, including fluoridated and chlorinated water, the residue of dental fillings and many common antacids containing aluminium and other hazardous materials. Toothpaste, food additives and synthetic food ingredients are possible dietary elements requiring further research. Infant feeding patterns may be related to the incidence of IBD according to some studies that have found a correlation between lack of breast-feeding and susceptibility to IBD. Also, the premature introduction of allergenic food such as cow's milk, soy and difficult to digest fruit juices may accelerate the development of IBD in children (Hoffman, 1997).

Geographical Factors

The incidence of Crohn's disease is higher in urban than in rural areas but no such geographical relationship has been shown in ulcerative colitis. Migrant studies have shown that environmental factors play a role in the development of these disorders. Crohn's disease is rare in Hong Kong Chinese yet is more common among Chinese migrants in Vancouver. People who migrate from the West Indies to Europe may increase their risk of developing Crohn's. Although there is a low prevalence of ulcerative colitis among Bangladeshi immigrants to the UK, the incidence of the disease among Sikhs and Hindus from Asia living in the UK is higher than among those remaining in Asia (Allison *et al.*, 1998).

Anatomy and Physiology of the Intestinal Tract

The Small Intestine

The small intestine is a convoluted tube extending from the pyloric sphincter in the epigastric region to the ileocaecal valve in the right iliac region where it joins the large intestine. It is the longest part of the alimentary canal, but its diameter is only about two and a half centimetres (Marieb, 2001) (Figure 12.1).

The small intestine is subdivided into three parts:

- The duodenum
- The jejunum
- The ileum

The duodenum is about 25 centimetres long. It is the first part of the small intestine; it is a short curved portion, roughly C-shaped and curves around the head of the pancreas (Jackson and Bennett, 1988). At its mid point there is an

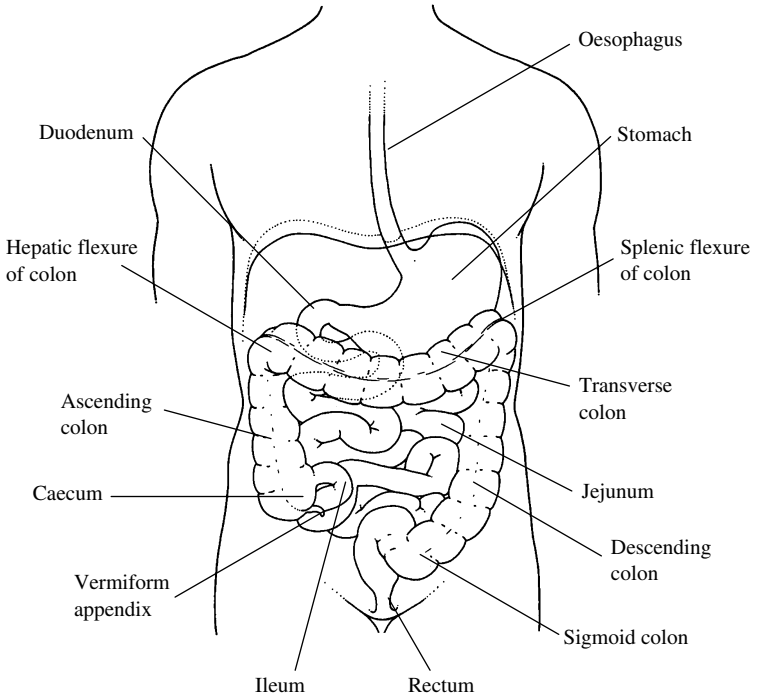

Figure 12.1 Digestive tract in body outline

opening, to the pancreatic duct and the bile duct, guarded by the hepatopancreatic sphincter.

The jejunum is the middle part of the small intestine and is about two metres long.

The ileum or terminal part is about three metres long and ends at the ileo-caecal valve, which controls the flow of material from the ileum to the caecum and prevents regurgitation (Wilson and Waugh, 1998).

Structure of the Small Intestine

The wall of the small intestine has the same four coats as the rest of the alimentary canal.

Coats of the small intestine:

- Serous coat
- Muscular coat
- Sub-mucous coat
- Mucous membrane lining

- The serous coat is formed of peritoneum. The abdominal organs are covered by a serous membrane, the peritoneum. It consists of a closed sac, containing a small amount of fluid, within the abdominal cavity. The parietal layer lines the abdominal wall and the visceral layer covers the organs within the abdominal cavity. The stomach and intestines, deeply invaginated from behind, are almost completely surrounded by peritoneum and have a double fold, the mesentery, which attaches them to the posterior abdominal wall (Wilson and Waugh, 1998).
- The muscular coat has a thin external layer of longitudinal fibres and a thick internal layer of circular fibres.
- The submucous coat contains blood vessels and nerves.
- The mucous membrane has three special features:

1. It is thrown into circular folds, which are permanent and are not obliterated when the intestine is distended. They increase the area for absorption.
2. It has a velvety appearance due to the presence of fine hair-like projections called villi, each containing a lymph vessel called a lacteal and blood vessels (Jackson and Bennett, 1988). The walls of the villi consist of columnar epithelial cells, or enterocytes, with tiny microvilli on their free border. Goblet cells that secrete mucus are interspersed between the enterocytes. These epithelial cells enclose the network of blood and lymph capillaries (Wilson and Waugh, 1998). Digested foodstuffs are absorbed through the epithelial cells into both the capillary blood and the lacteal. Between the villi, the mucosa is studded with pits or openings that lead into tubular intestinal glands called intestinal crypts or crypts of Lieberkuhn. The epithelial cells that line these crypts secrete intestinal juice (Marieb, 2001).
3. It is supplied with simple tubular type glands that secrete intestinal juice (Jackson and Bennett, 1988).

The small intestine contains considerable amounts of lymphoid tissue that deals with bacteria that may be absorbed through the intestinal wall. The lymph nodes are numerous in the mucosa at irregular intervals throughout the length of the small intestine. The smaller ones are known as solitary lymphatic follicles and larger nodes, situated towards the distal end of the ileum are called aggregated lymphatic follicles or Peyer's patches (Wilson and Waugh, 1998). The Peyer's patches increase in abundance towards the end of the small intestine, reflecting the fact that the large intestine contains huge numbers of bacteria that must be prevented from entering the bloodstream (Marieb, 2001).

Functions of the small intestine:

- Onward movement of its contents, which is brought about by peristalsis, segmental and pendular movements.

- Completion of chemical digestion of carbohydrates, protein and fats in the enterocytes of the villi
- Protection against infection by microorganisms that have survived the antimicrobial acid in the stomach, by the solitary and aggregated lymph follicles
- Secretion of the hormones cholecystokinin and secretin. These hormones are produced by cells in the walls of the duodenum and stimulate the secretion of pancreatic juice. The presence in the duodenum of acid material from the stomach stimulates the production of these hormones
- Absorption of nutrient material

(Wilson and Waugh, 1998)

The Large Intestine

The large intestine is about one and a half metres long. It begins at the caecum in the right iliac fossa and terminates at the rectum and anal canal, deep in the pelvis (see Figure 12.1). Its diameter is greater than that of the small intestine but it is less than half as long. It forms an arch round the coiled up small intestine. Its major function is to absorb water from indigestible foodstuffs and eliminate them from the body as semisolid faeces. Over most of its length the large intestine exhibits features not seen elsewhere. Except for its terminal end, the longitudinal muscle layer of its muscularis is reduced to three bands of smooth muscle called teniae coli. Their tone causes the wall of the large intestine to pucker into pocket like sacs called haustra. A unique feature of the large intestine is its epiploic appendages. These are small, fat filled pouches of visceral peritoneum that hang from the surface. Their significance is not known (Marieb, 2001).

Parts of the Large Intestine

The large intestine is divided into seven parts.

- The caecum
- The ascending colon
- The transverse colon
- The descending colon
- The sigmoid colon
- The rectum
- The anal canal

- The caecum lies in the right iliac fossa. It is a dilated portion, which has a blind end inferiorly and is continuous with the ascending colon superiorly. Just below the junction of the two the ileocaecal valve opens from the ileum (Wilson and Waugh, 1998). Attached to its posteromedial surface is the blind, wormlike vermiform appendix, which contains masses of lymphoid tissue (Marieb, 2001).
- The ascending colon passes upwards from the caecum to the level of the liver where it bends acutely to the left at the hepatic flexure to become the transverse colon.
- The transverse colon is a loop of colon, that extends across the abdominal cavity in front of the duodenum and stomach to the area of the spleen where it forms the splenic flexure by bending acutely downwards to become the descending colon.
- The descending colon passes down the left side of the abdominal cavity then curves towards the midline. After it enters the true pelvis it is known as the sigmoid colon.
- The sigmoid describes an S-shaped curve in the pelvis then continues downwards as the rectum.
- The rectum is a slightly dilated part of the colon that is about 13 centimetres long. It leads from the sigmoid colon to the anal canal.
- The anal canal is a short canal and leads to the exterior. There are two sphincter muscles that control the anus. The internal sphincter consists of smooth muscle and is under the control of the autonomic nervous system and the external sphincter, formed by striated muscle is under voluntary nerve control (Wilson and Waugh, 1998).

The Structure of the Large Intestine

- Outer serous coat of peritoneum
- Muscular coat
- Sub-mucous coat
- Mucous membrane lining

The arrangement of the longitudinal muscle fibres is modified in the colon. They do not form a smooth continuous layer of tissue but are collected into three bands, called teniae coli, situated at regular intervals round the colon. They stop at the junction of the sigmoid colon and the rectum. As these bands are slightly shorter than the total length of the colon they give a sacculated or puckered appearance to the large intestine. The sacculations are called haustrations or haustra (Wilson and Waugh, 1998). The longitudinal muscle fibres spread out and completely surround the rectum and anal canal. Thickening of the circular muscle layer forms the anal sphincters. In the submucous coat there is more lymphoid tissue than in any other part of the alimentary tract, providing non-specific defence against invasion by resident and other microorganisms. In the mucosa that lines the colon and upper part of the

rectum there are large numbers of goblet cells forming simple tubular glands, which secrete mucus. They are not present beyond the junction between the rectum and the anus. The lining membrane of the anus consists of squamous epithelium, which is continuous with the mucous membrane lining of the rectum above and merges with the skin beyond the external sphincter (Wilson and Waugh, 1998).

Functions of the Large Intestine

- Absorption of water and salts
- Defecation
- Microbial activity

Absorption. Absorption of water continues in the large intestine along with mineral salts, vitamins and some drugs.

Defecation. The large intestine does not exhibit peristaltic movement as seen elsewhere in the digestive tract. Only at fairly long intervals does a wave of strong peristalsis sweep along the transverse colon forcing its contents into the descending and sigmoid colon. This is known as mass movement and when this forces faeces into the rectum, nerves endings in its wall are stimulated. Defecation is a reflex action but the brain can inhibit the reflex until it is convenient to defecate. If defecation is delayed the sensation of fullness in the rectum passes and more water is absorbed (Jackson and Bennett, 1988).

Contents of faeces

- Water makes up 60–70 per cent of the weight
- Fibre
- Dead and live microorganisms
- Epithelial cells from the walls of the tract
- Fatty acids
- Mucus, which helps to lubricate the faeces

Microbial Activity. Although most bacteria entering the caecum from the small intestine are dead some are alive. Together with bacteria that enter via the anus, these constitute the bacterial flora of the large intestine. These bacteria colonize the colon and ferment some of the indigestible carbohydrates, releasing irritating acids and a mixture of gases. About 500 millilitres of gases are produced each day, much more when certain carbohydrate-rich foods are eaten. The bacterial floras also synthesize B complex vitamins and most of the vitamin K the liver requires to synthesize some of the clotting proteins (Marieb, 2001). These bacteria are commensals in humans; they include *Escherichia coli*, *Enterobacter aerogenes*, *Streptococcus faecalis* and *Clostridium perfringens*. They may become pathogenic if transferred to another part of the body (Wilson and Waugh, 1998).

Pathophysiology

In both diseases the intestinal wall is infiltrated with acute and chronic inflammatory cells, however, there are differences in the distribution of disease and in histological features (Palmer and Penman, 1999) (see Table 12.2).

Ulcerative colitis is predominantly a mucosal disease of the colon. The rectum is almost always involved; proximal extension occurs in about half of patients who present initially with disease confined to the rectosigmoid region. In some cases the whole colon may be affected (pancolitis). There is usually an abrupt cut off between affected and unaffected colon. A variety of appearances may be encountered depending upon the activity and duration of the disease. In acute-onset severe disease (toxic megacolon) most of the bowel is distended and plum-coloured and the walls are paper thin and friable. More usually, the disease is confined to the mucosa, with only mild reactive changes in the submucosa. In contrast to Crohn's disease, the bowel wall is not thickened and fibrous, and the outer surface of the bowel may appear normal (Allison *et al.*, 1998). Initially there is reddening and oedema of the mucosa with bleeding points. This is followed by ulceration, which is usually superficial. In long standing pancolitis the bowel becomes shortened and narrowed with lack of haustrations (Miller *et al.*, 1994). The mucosal surface is characterized initially by hyperaemia and capillary fragility. With advancing disease these changes lead to irregular ulceration, which may become confluent, with undermining of the adjacent mucosa. Residual mucosal bridges then disappear and areas of the mucosal surface become denuded. This means that most of the mucosa has been lost leaving only small islands of congested mucosa, overlying a smooth and inflamed submucosal base (Allison *et al.*, 1998). Inflammation at the base of the crypts of Lieberkuhn damages the epithelial cells in the crypts and forms abscesses. The mucosa surrounding the ulcerations may appear 'heaped up', because the muscularis

Table 12.2 A comparison of ulcerative colitis and Crohn's disease

Ulcerative colitis	*Crohn's disease*
■ Mucosal involvement	■ Transmural involvement
■ Inflammation is confluent	■ Skip lesions
■ Granulomas absent	■ Granulomas present
■ Crypt abscesses common	■ Crypt abscess uncommon
■ Reduced number of goblet cells	■ Normal number of goblet cells
■ Colon affected	■ Any part of the gastrointestinal tract affected
■ Strictures uncommon compared with Crohn's disease	■ Strictures (narrowing of the lumen of the bowel)
■ Anal lesions such as fissures and fistulas are less severe and less common	■ Fistulas and fissures are features
■ Curative surgery	■ No cure

mucosa tends to contract. These are known as pseudopolyps and are a diagnostic finding (Doughty and Jackson, 1993). Acute and chronic inflammatory cells infiltrate the lamina propria and the crypts. Goblet cells lose their mucus and may become distorted in long-standing cases Inflammation is confluent and is more severe distally (Palmer and Penman, 1999).

In Crohn's disease any part of the gastrointestinal tract from mouth to anus can be affected but the commonest site is the terminal ileum. In some cases the whole of the colon and/or the small intestine can be involved (Clark and Kumar, 1994). The inflammatory changes affect isolated segments of all layers of the intestinal wall, which becomes oedematous and thickened. Crohn's disease can involve one small area or multiple areas with relatively normal bowel in between (skip lesions). The change from the affected area is abrupt. The mesenteric lymph nodes are enlarged and the mesentery thickened (Palmer and Penman, 1999). Aphthous ulcers are probably the earliest pathological finding. These begin as microabscesses in lymphoid follicles and develop into superficial ulcers surrounded by normal or mildly oedematous mucosa. As the disease process continues, the ulcers become more invasive forming fissures that may extend into the submucosal layer (rake ulcers). The rake ulcers run longitudinally and transversely and tend to coalesce, creating a 'cobblestone' appearance of the bowel wall. With advanced disease, fissures can penetrate the bowel wall to create fistulas or abscesses. Narrowing (strictures) of the lumen of the bowel wall also occurs as the disease advances (Doughty and Jackson, 1993). Fistulas can develop between adjacent loops of bowel or between affected segments of bowel and the bladder, uterus or vagina and may appear in the perineum (Palmer and Penman, 1999). There are focal aggregates of epithelioid histocytes, which may be surrounded by lymphocytes and contain giant cells. These are non-caseating granulomas and are a hallmark of Crohn's (Clark and Kumar, 1994). This feature is seen in about 50 per cent of cases (Allison *et al.*, 1998).

Clinical Manifestations

Ulcerative Colitis

The first attack is usually the most severe and in most cases will be followed by relapses and remissions of the disease. Emotional stress, infection, gastroenteritis, antibiotics or non-steroid anti-inflammatory drugs may provoke a relapse (Palmer and Penman, 1999). The usual presenting symptom is the passage of frequent loose, small volume, brown motions with fresh blood and mucus either on the surface of the stools or intermixed with them. Patients with mild attacks open their bowels up to five times a day, with moderate attacks up to ten times a day and with severe attacks over ten times a day (Long and Cooper, 1997). In an acute attack, 20 liquid stools a day may be passed and diarrhoea occurs at night, with urgency and incontinence that is severely disabling for the

patient. Occasionally blood and mucus alone are passed (Clark and Kumar, 1994). The pain is usually located in the lower left quadrant and is colicky in nature. After defaecation, pain may subside. In very severe cases tenderness in the left lower quadrant, guarding and abdominal distension may occur (Walsh, 1997).

The clinical features depend upon the site and activity of the disease:

Proctitis

Proctitis causes rectal bleeding and mucus discharge accompanied by tenesmus. Some patients pass frequent, small volume liquid stools but some may experience constipation and pass small pellet stools. No constitutional symptoms occur.

Proctosigmoiditis

Proctosigmoiditis causes bloody diarrhoea with mucus. Most patients are well, having no constitutional symptoms. Small minorities of patients, who have very active, limited disease, develop fever, lethargy and abdominal discomfort.

Extensive Colitis

Extensive colitis causes bloody diarrhoea with mucus. In severe cases anorexia, malaise, weight loss and abdominal pain occur. The patient is toxic with fever, tachycardia and signs of peritoneal inflammation. Fever and tachycardia, usually indicate severe widespread and active disease rather than infection. Anaemia is usually hypochromic and microcytic and due to blood loss (Palmer and Penman, 1999).

Toxic Megacolon

This is a rare, very serious complication resulting from a spread of the mucosal inflammation to the submucosal, muscular and possibly the serosal layers of the colon; the smooth muscle is paralysed allowing the colon to passively dilate and the barrier functions of the epithelium appears to be lost, permitting the uptake of bacterial toxins and antigens. Clinical manifestations include:

- Severe abdominal distention
- Abdominal pain and tenderness
- Chills
- Fever
- Anorexia
- Nausea and vomiting
- Bloody diarrhoea
- Leucocytosis

The goal of treatment is to prevent perforation and peritonitis, which carries a high mortality rate (Doughty and Jackson, 1993).

Crohn's Disease

The patient with Crohn's disease may have occasional acute episodes of illness but more often has mild intermittent symptoms. The commonest initial presentation is with terminal ileum disease, often with coexistent oral and anal involvement. Less common initial presentations are acute right iliac fossa pain simulating acute appendicitis and acute colitis identical to ulcerative colitis. General symptoms include the following:

- Fever
- Malaise
- Weight loss (may be due to malabsorption or avoidance of food, since eating provokes pain. Some patients present with features of fat, protein or vitamin deficiency)
- Amenorrhoea
- In children: failure to thrive, growth retardation, delayed puberty
- In pre-pubertal and pubertal children there may be no history of diarrhoea or abdominal pain (Long and Cooper, 1997)

Gastrointestinal Symptoms

As a result of stricture formation many patients with ileal disease complain of colicky abdominal pain, usually in the right iliac fossa. Diarrhoea is usually moderate and may contain excess fat. Flatulence, nausea, borborygmus and increased peristalsis may be present (Walsh, 1997). Urinary frequency and dysuria may result from inflamed bowel adjacent to the urinary tract. Air in the urine and faeces via the urethra or vagina indicate a fistula from bowel to genitourinary tract (Long and Cooper, 1997).

The gastrointestinal symptoms depend primarily upon the site of disease.

Ileal Disease

Ileal disease causes colicky, abdominal pain, principally due to subacute obstruction, although an inflammatory mass, intra-abdominal abscess or acute obstruction may be responsible (Palmer and Penman, 1999). Pain is often associated with diarrhoea that is soft or semi-liquid and does not contain blood or mucus. If steatorrhoea is present the stools will be foul smelling and fatty. When the terminal ileum is involved, the pain is in the periumbilical region. Initially the ileal pain is peristaltic and intermittent; later it becomes more constant and may be noticed in the lower right quadrant. Cramps of regional enteritis are not closely associated with defaecation, and unlike cramps with colonic disease are not relieved by passing stool or flatus. A constant aching soreness or tenderness usually indicates advanced disease (Walsh, 1997).

Crohn's Colitis

Crohn's colitis presents in an identical way to ulcerative colitis with bloody diarrhoea, passage of mucus and constitutional symptoms including lethargy, malaise, anorexia and weight loss. Rectal sparing and the presence of perianal disease favour a diagnosis of Crohn's disease rather than ulcerative colitis. Many patients present with symptoms of both small and large bowel disease. A few have isolated perianal disease, vomiting from jejunal strictures or severe oral ulceration.

Physical examination often reveals evidence of weight loss, anaemia with glossitis and angular stomatitis. Abdominal tenderness, most marked over the inflamed area is present. An abdominal mass due to matted loops of thickened bowel or an intra-abdominal abscess may occur. Perianal skin tags, fissures or fistulae are found in at least 50 per cent of patients (Palmer and Penman, 1999).

Acute arthritis, portal pyaemia, liver abscess, and dermatological, ocular and vascular complications tend to occur during acute relapse of bowel disease. IBD can be considered as a systemic illness and in some patients extraintestinal complications are a dominant clinical feature. Some occur during a relapse of intestinal disease; others appear unrelated to intestinal disease (see Table 12.3) (Palmer and Penman, 1999).

Table 12.3 Complications of inflammatory bowel disease

Intestinal	*Extraintestinal*
Inflammation ■ Severe, life-threatening inflammation of the colon (occurs in ulcerative colitis and Crohn's disease)	**Seronegative arthritis** ■ Acute arthritis affecting medium sized joints ■ Ankylosing spondylitis ■ Sacroilitis
Perforation ■ Perforation of the small intestine or colon (can occur without the development of toxic megacolon)	**Dermatological** ■ Erythema nodosum ■ Pyoderma gangrenosum ■ Oral aphthous ulcers
Haemorrhage ■ Life-threatening acute haemorrhage (due to erosion of a major blood vessel)	**Ocular** ■ Conjunctivitis ■ Iritis ■ Episcleritis
Fistula and perianal disease ■ Fistula and perianal disease (specific complications of Crohn's disease and not ulcerative colitis)	**Hepatic and biliary** ■ Primary sclerosing cholangitis (ulcerative colitis only) ■ Gallstones ■ Autoimmune hepatitis ■ Fatty liver ■ Portal pyaemia and liver abscesses ■ Amyloidosis ■ Cholangiocarcinoma

Table 12.3 (Contd.)

Intestinal	*Extraintestinal*
Cancer ■ Cancer (patients with extensive colitis of more than eight years duration are at increased risk of colon cancer). The cumulative risk for patients with ulcerative colitis may be as high as 20 percent after 30 years but is probably less for Crohn's colitis	**Renal** ■ Oxalate calculi (small bowel Crohn's) ■ Amyloidosis ■ Ureteric obstruction (Crohn's) **Vascular** ■ Deep vein thrombosis ■ Portal or mesenteric vein thrombosis

Investigative Tests

History

After taking a history the doctor will assess each individual to decide the type and extent of investigations required (Table 12.4).

Medical and Surgical Treatment

Medical Management

Aims are to bring about remission and maintain this for as long as possible. This may involve correcting fluid and electrolyte balance, malnutrition and anaemia.

Table 12.4 Investigative tests

Tests	*Investigations*
General examination	■ Recent weight change ■ Appetite ■ Bowel frequency ■ Pain ■ Energy levels/altered lifestyle
Abdominal observation and palpation	Observable abdominal distention or visible peristalsis. Palpable mass or areas of tenderness.
Ano-rectal examination	
Visual inspection	Inspection of perianal skin, noting any skin tags, anal fissure, anal fistula, skin discolouration.
Protoscopy	Visual examination of the rectal mucosa.
Sigmoidoscopy	Direct examination of the anal canal rectum and sigmoid colon, using a rigid or flexible instrument.

Table 12.4 (Contd.)

Tests	*Investigations*
	Rectal sparing, perianal disease and discrete ulcers suggest Crohn's disease rather than ulcerative colitis.
Rectal biopsy	Taken during sigmoidoscopy to determine disease extent, as this is underestimated in endoscopic appearance alone and to seek dysplasia in patients with long-term disease.
Colonic examination	
Plain abdominal x-rays	In severe colitis to observe for any colonic dilatation and assess the extent of the disease, which can be judged by the distribution of air in the colon.
Barium enema	Less sensitive than colonoscopy for the investigation of colitis. In long standing ulcerative colitis the bowel becomes shortened and loses haustra to become tubular and pseudopolyps are visible. In Crohn's disease the appearance may be similar to ulcerative colitis but skip lesions, strictures and deep ulcers are characteristic Contrast studies of the small bowel are normal in ulcerative colitis. Barium enema is contraindicated in acute disease.
Colonoscopy	The whole bowel can be viewed with a fibreoptic scope. May show active inflammation with pseudopolyps or a complicating carcinoma. In ulcerative colitis the macroscopic and histological abnormalities are confluent and most severe in the distal colon and rectum. In Crohn's colitis the endoscopic abnormalities are patchy with normal mucosa between the areas of abnormality and 'cobblestoning' of the mucosa. Aphthoid or deep ulcers are common.
Radionucleide scans	Radio-labelled white cell scans can show areas of active inflammation. This test is less sensitive than conventional tests but is useful in severely ill patients in whom invasive tests are to be avoided.
Ultrasound and CT scanning	Helpful in delineating abscesses, masses, thickened mesentery or other extraluminal problems in Crohn's disease.
Small bowel examination	
Barium enema and follow through	To identify disease in the upper gastrointestinal tract.
Specimens	
Stool analysis and culture	
Observation	To observe consistency and presence of blood, mucus, pus or steatorrhoea.
Microbiological examination	To rule out infective agents.
Stool weight	Measure stool weight per 24 hours.
Blood analysis	
Full blood count (FBC)	For noting any anaemia, raised white cell count or raised platelets.
Erythrocyte Sedimentation Rate (ESR) or C Reactive Protein (CPR)	ESR or CPR may be raised in active disease.
Liver Function Test (LFT)	Particularly noting albumin level as a nutritional indicator.
Urea and electrolytes (U & E)	To indicate fluid balance deficiencies and general state of health.

Palmer and Penman, 1999; Curry, 1995; Clark and Kumar, 1994.

The principles of drug treatment are similar for ulcerative colitis and Crohn's disease.

Drug therapy

- Corticosteroids
- Aminosalicylates
- Immunosuppressants
- Antibiotics
- Nicotine

Corticosteroid Therapy

Corticosteroids are first line treatment and are prescribed for their anti-inflammatory and immunosupressant actions. Steroid foam or liquid retention enemas, from which systemic corticosteroid absorption is insignificant, are used to treat active proctosigmoiditis. In severe proctosigmoiditis, where patient is unable to retain enemas or patient has active, extensive colitis, oral corticosteroids are given. Severe active colitis can be treated with intravenous methylprednisolone. Once improvement occurs, a reducing regimen of oral prednisolone is commenced.

Systemic Steroid Side Effects: Mood changes, acne, weight gain and dyspepsia are common but resolve as soon as the dosage is reduced. More rarely, hypertension and hyperglycaemia may result. Long term, high dose therapy is avoided because of the risks of metabolic bone disease and infection (Palmer and Penman, 1999).

Aminosalicylate Therapy

Sulphasalazine, mesalazine, balsalazineside or olsalazine are given during an attack of ulcerative colitis and for long-term use to keep the disease in remission to prevent inflammation from developing. These are also given for Crohn's colitis. For small intestine Crohn's disease, a slow release mesalazine preparation is used. These drugs may be given as tablets, enemas or suppositories.

Side Effects. Headaches, nausea, skin rashes, diarrhoea, anorexia, oligospermia, renal toxicity, blood dyscrasias.

Nursing action points

- Be aware that side effects such as headaches, nausea and anorexia are dose related.
- Inform young male patients of the potential side effect of oligospermia, with sulphasalazine therapy, though this is reversible once the drug is withdrawn.
- Be aware that the newer range of drugs are as effective as sulphasalazine but have significantly lower side effects.

(Rowlinson, 1999b)

Immunosuppressant Therapy

Immunosuppressant therapy with drugs such as azathioprine, methotrexate or cyclosporine is valuable in refractory inflammatory bowel disease in helping to achieve and maintain clinical remission, reducing steroid use and avoiding surgery. These drugs work by blocking the immune reaction that contributes to inflammation.

Side Effects. Nausea, vomiting, diarrhoea and a lowered resistance to infection (Rowlinson, 1999b).

Antibiotic Therapy

A fistula complicated by infection or a stagnant area or loop of intestine in which there is an overgrowth of bacteria may be treated with antibiotics such as, metronidazole, ampicillin, cephalosporin, tetracycline or sulphonamide (Walsh, 1997). The use of antibiotics may be to eradicate atypical mycobacteria. Rifambutin and clarithromycin have been used to treat people with Crohn's disease and analysis of the outcomes showed significant improvement in disease severity and a reduction in inflammatory markers. However, not all people respond to the treatment, possibly due to existing drug resistance (Hermon-Taylor, 2000).

Nicotine

In various clinical trials both nicotine gum and transdermal patches have been found to be clinically effective in the treatment of ulcerative colitis, although there was a high incidence of side effects, especially among those patients that had never smoked (Rowlinson, 1999b).

Infliximab, the first treatment approved specifically for the treatment of Crohn's disease is an anti-tumour necrosis factor (anti-TNF) substance. TNF is a protein produced by the immune system that may cause the inflammation associated with Crohn's disease. Anti-TNF removes TNF from the bloodstream before it reaches the intestines. It has been used to treat moderate to severe cases that do not respond to standard therapies.

Nutrition

There is no specific therapeutic diet for patients with IBD. Many patients find that certain foods aggravate their symptoms and these are therefore best avoided. Such foods vary widely from patient to patient but include fatty foods, dairy products, caffeine or raw, high fibre foods that are difficult to digest.

Sometimes IBD is treated with an elemental diet, which is a liquid consisting of all the nutrients in a pre-digested form, ready for absorption through the wall of the small intestine. Usually nothing but the diet and water are allowed for weeks or months at a time. The diet can be rather unpalatable and should be taken in sips to avoid increasing the diarrhoea. In some cases it may be given via a nasogastric tube.

Medical intervention may be necessary in order to restore nutritional and electrolyte balance during active disease. Nutritional deficiencies are common due to

- Decreased nutritional intake
- Increased nutritional requirements due to inflammation, infection and fever
- Increased nutritional losses due to malabsorption, diarrhoea, bleeding
- Nutritional interference by drugs, for example, steroids

(Curry, 1995)

In fulminating disease, enteral nutrition may not be possible and total parenteral nutrition (TPN) will be required. If enteral nutrition is possible, an elemental residue free diet may be necessary for a short while until a low residue diet can be introduced. As the inflammation settles dietary restrictions can be reduced. During periods of remission a normal well-balanced diet is recommended. It should contain sufficient kilocalories to restore and maintain weight, as well as being high in protein and carbohydrates and low in fat. Supplements of vitamins, iron, folic acid, zinc and potassium may be required (Miller *et al.*, 1994).

Fish oil preparations have been shown to have anti-inflammatory properties and have been recommended for use in a number of chronic inflammatory diseases and it is claimed that they may have some positive benefits in prolonging periods of remission in patients with Crohn's disease (Medicinenet.com, 1999).

Nursing action points

- Identify what type of diet the patient normally has.
- Advise the patient to note food or drinks that aggravate their symptoms so that they can be avoided in future.
- Note how much of a normal diet is eaten and tolerated.
- Record prescribed supplements on the appropriate chart.
- Be aware that nutritional supplements may be recommended, especially for children whose growth has been slowed. Special high-calorie liquid formulas may be used for this purpose.

Surgical Management

Surgery may be appropriate in the following situations:

- Loss of occupation or education.
- Disruption of family life.
- Disease complications such as arthritis and pyoderma gangrenosum that are unresponsive to medical treatment (Palmer and Penman, 1999).
- Imminent risk of perforation.
- A long history of moderately active disease with few or no periods of remission.
- Failure to respond to medical treatment.
- Intestinal obstruction and abdominal or perianal fistulae.
- Pre-cancerous change or cancer (Curry, 1995).

The type of surgery will depend upon the specific diagnosis. Up to 60 per cent of patients with extensive ulcerative colitis eventually require surgery, involving removal of the entire colon and rectum, which cures the patient. The choice of procedure is either panproctocolectomy with ileostomy or proctocolectomy with ileo-anal pouch anastomosis.

The indications for surgery in Crohn's disease are similar to those for ulcerative colitis. Operations are often necessary for dealing with fistulae, abscesses or perianal disease and may be necessary to relieve small or large bowel obstruction. Up to 80 per cent of patients eventually require some form of surgery, but unlike ulcerative colitis surgery does not cure the patient. Surgical intervention should therefore be conservative in order to conserve as much viable intestine as possible and to avoid a short bowel syndrome.

Surgical procedures

- Localized segments of Crohn's colitis may be managed by segmental resection.
- Extensive colitis may require total colectomy (ileo-anal pouch is to be avoided because of the high risk of disease recurrence in the pouch and subsequent fistula, abscess formation and pouch failure).
- Perianal disease is managed as conservatively as possible by drainage of the abscess and avoidance of resection or reconstructive procedures.
- Obstructing or fistulating small bowel disease may require resection of affected tissue.
- Multiple or recurrent strictures may require strictureplasty in which the stricture is not resected but incised in its longitudinal axis and sutured transversely (Palmer and Penman, 1999).

Nursing Interventions

Nursing care will inevitably be determined by the presenting symptoms.

Nursing action points

Symptoms	Nursing interventions
Diarrhoea	
The need to defecate is often associated with a degree of urgency and tenesmus. The stool may also have an offensive smell.	■ Position the patient close to a lavatory. ■ If the patient is unable to reach a lavatory due to weakness or urgency, provide a bedside commode. ■ Monitor faecal loss on a stool chart, recording consistency, presence of blood, mucus and pus. ■ Provide soft tissues and wipes. ■ Provide a pleasant air freshener; this may alleviate embarrassment. ■ Maintain privacy and dignity. Patient may monitor his or her own stool output if appropriate.
Faecal incontinence	
This may be a problem. Incontinence associated with frequent bowel movements can lead to excoriated perianal skin.	■ Assist the patient to maintain personal hygiene. ■ Provide disposable pants and pads. ■ Help the patient to wash perianal skin and apply barrier cream if excoriation or soreness is a problem.
Weakness and general malaise due to a combination of inflammatory disease, frequent bowel movements and weight loss. Interrupted sleep adds to the tiredness.	■ Give assistance with maintaining the activities of living; ensure that the patient has adequate rest. ■ Provide a quiet environment.
Loss of appetite	
The disease process may suppress appetite	■ Offer small appetizing meals that are light and easy to digest.

and patients may be reluctant to eat as they have a misconception that eating will increase bowel frequency. Nausea and vomiting may also be experienced.	■ In between snacks may be required. ■ Be aware that dietary supplements may be prescribed to maintain nutritional intake. ■ Administer prescribed anti-emetics as appropriate. ■ Monitor body weight.
Abdominal pain Persistent, colicky abdominal pain is characteristic of Crohn's disease. The pain of ulcerative colitis is severe prior to defecation and is usually relieved by defecation.	■ Monitor the patient's pain and report any changes. ■ Encourage the patient to report any aggravating factors. ■ Ensure the patent's comfort by providing extra pillows.
Anxieties These may be related to lack of understanding of the disease, fears of having to have a stoma or fears of being unable to cope with work or taking care of the family. Altered body image due to weight loss may be a problem. The increasing incidence of Crohn's disease among young children can lead to problems at adolescence. This can be a very difficult time as changes in nutritional state may lead to a pallid and sickly appearance that the adolescent perceives as decreasing body image (Pullen, 1999).	■ Give full explanations of planned treatment and investigations. ■ Correct any misconceptions. ■ Give time for patient to express any anxieties and discuss the effects that the disease has upon the individual and significant others. ■ Identify specific psychological problems and provide the relevant support, for example, counselling. ■ Liaise with stoma therapist if a stoma is indicated. ■ Liaise with a medical social worker. ■ If appropriate and desired arrange a meeting with another person who has had similar experiences of the disease and treatment. Contact NACC for further patient information (Curry, 1995).

A professional and caring approach will help the nurse to establish a trusting partnership in care. This is essential for those practitioners involved in the long-term care of patients, as compliance with maintenance drug therapy and other relevant advice will determine the potential course of the disease. A prime aim will be to reduce stress, as there is growing evidence that both chronic and everyday stress can cause psychophysiological reactions such as colonic hyperactivity. Patient participation in care gives back an element of control over his or her life. While not all patients will want to be fully informed or involved in decision-making they should be given the choice. The role of the nurse is to support those individuals who wish to be fully involved, while relieving those for whom this responsibility is perceived to be too overwhelming. The effects upon the family should also be taken into account. It is not uncommon for family members, in caring for the individual with IBD to overlook their own needs. This can result in family tensions, fatigue, feelings of resentment and decreased ability to cope (Walsh, 1997).

Connection

Chapter 2 (Family-Centred Care) discusses how the health of relatives can be compromised by the burden of care and explores ways in which nurses can support such family members.

Nursing action points

- Encourage patients to take control of their own lives where appropriate.
- Discuss stress management techniques.
- Assess how relatives are coping with the impact of caring.

The Impact of IBD

Some individuals may feel it necessary to hide their illness from employers for fear of discrimination or due to the socially unaccepted nature of the disease and the associated embarrassment. Despite poor attendance, affected children show a normal educational outcome in terms of leaving age and the uptake of further education (Mayberry *et al.*, 1992). The fear of diarrhoea and incontinence may inhibit social life. It is common for individuals to locate the nearest lavatory in any new situation. Personal relationships can be affected. Fear of faecal incontinence, abdominal pain, dyspareunia and perianal disease can have a major impact upon sexuality. Fertility in male patients has not been shown to be different from the normal population and women with IBD have the ability to

conceive. Fewer children are born to such women, possibly through choice or medical advice (Rowlinson, 1999c).

Nursing action points

- Be aware of the potential embarrassment that this condition may cause patients.
- Be aware that patients may find it difficult to initiate discussion relative to sexual dysfunction.

The Role of the Clinical Nurse Specialist

In adapting to a chronic illness or significantly altered body function, feeling understood and supported is of prime importance. This is difficult to achieve if patients see different staff at each clinic attended. Many patients only ever attend an outpatient clinic or their GP's surgery and even then may not see the same doctor. The need for continuity and the benefits of being seen by a familiar person have been recognized and nurse specialist posts are being developed in many gastroenterology departments. These posts will enable patients to obtain specialist advice and pastoral care. They will enable patients to make informed choices on future treatment and complementary therapies. Nurse specialists will be able to make referrals to other specialists, ward and community nurses and to disseminate their expertise to an array of practitioners (Finley, 1999).

A nurse whose role is totally devoted to patients with IBD can be invaluable in providing educational, emotional and psychological support. Such nurses do not replace doctors or 'general' nurses but provide a complementary service. Drawing upon research, and their own experience and intuition, they are in a unique position to influence the quality of care offered to patients. By combining both medical and nursing functions to provide a service that is acceptable to patients, the specialist nurse can assist patients to attain a higher quality of life through purposeful interventions designed to minimize symptoms, reduce the frequency and intensity of exacerbations and enhance psychosocial well-being (Rowlinson, 1999c).

Conclusion

There are marked differences in the manifestations and severity of the disease and in the ways that individuals adapt and cope with symptoms. The fact that only the worst exacerbations require serious medical intervention may lead to both patients and healthcare professionals underestimating the physical and psychological impact that the chronic disease has upon the individual and his or her family. It may be true that individuals do not often feel very ill but they may not often feel entirely well, such is the nature of IBD.

Colorectal Cancer and IBD

Individuals with chronic ulcerative colitis are at increased risk of developing colorectal carcinoma, particularly if there is long-standing or extensive colitis. It is generally accepted that the risk of colorectal cancer does not begin until eight to ten years after ulcerative colitis is diagnosed. Thereafter the risk increases by approximately 0.5–1.0 per cent, per year. The risk is smaller and less well defined (Solomon and Schnitzler, 1998). The relative risk of colorectal cancer is increased in both ulcerative colitis and Crohn's disease in those patients whose colitis started before the age of 25 years. Whether the absolute risk is greater in the younger age group or merely reflects the fact that the expected number of carcinomas increases with age is uncertain. The number of patients with Crohn's disease who actually develop cancer is small because many patients with extensive colitis undergo colectomy early in the course of the disease to relieve persistent symptoms that do not respond to medical treatment (Gillen *et al.*, 1994).

Aetiology

Epithelial cells lining the colon are normally subject to rapid turnover. They form and mature and are then lost by a process of sloughing within a period of two to three days. This rapid turnover prevents the accumulation of damaged or defective cells. In some cases the renewal cycle breaks down and abnormal tissue structures begin to appear. This can range from hyperplasia (excessive production of normal cells) to dysplasia (clumps of abnormal cells) to polyps, comprised of dysplastic cells (Campbell, 1999). The increased cell proliferation that occurs in ulcerative colitis may predispose the mucosa to mutational events, thereby increasing the risk of cancer (Noffsinger *et al.*, 1996). Histopathological grading of tumour tissue gives a measure of its aggressiveness and may relate to prognosis and treatment.

Connection

Chapter 14 (Cancer – an Overview) addresses the aetiology and pathophysiology of malignant changes in body cells.

Screening

The signs and symptoms of IBD may mask any symptoms of malignancy and monitoring any changes in the pattern of disease is important. The most

significant predictor of the risk of malignancy is the presence of dysplasia in colonic biopsies; there is however, controversy over the efficacy of colonoscopy and the role of prophylactic surgery. Research is underway to identify genetic and biochemical markers that may prove useful for predicting cancer risk (Solomon and Schnitzler, 1998).

Treatment

Recent research into colorectal cancers has resulted in the increased use of combination therapies, which incorporate local treatments (surgery and radiotherapy) and systemic therapy (cytotoxic chemotherapy), either to cure or offer palliation for the disease. The choice will depend upon the stage of the disease and the prognosis at the time of presentation. The primary treatment for potentially curable cancer is resection of the tumour, the surrounding tissue and the draining lymph nodes, followed by restoration of intestinal continuity. The location of the tumour determines the surgical approach. If the tumour is extensive, palliative resection of the affected portion of the bowel can relieve obstruction, alleviate the local effects of the tumour and prevent perforation and haemorrhage.

Radiotherapy may be given pre-operatively to downstage a tumour or make it smaller. Post-operative radiotherapy is given less frequently and is reserved for patients considered to be at high risk of residual disease, for example when histology demonstrates the presence of malignant cells at the margins of the resected tissue. Palliative radiotherapy provides relief from symptoms of colorectal cancer and for the treatment of distant metastases.

Chemotherapy may be commenced prior to surgery to reduce the tumour size and offers early systemic treatment for undetectable micro-metastases if given at a time when malignant cells are likely to be more responsive to treatment. Adjuvant chemotherapy attempts to treat occult metastases following surgery or radiotherapy with curative intent. Palliative chemotherapy aims to improve the duration of survival and the quality of life in advanced disease when a high incidence of toxicity and patient distress is clearly inappropriate (Lunn *et al.*, 1999).

The Role of the Nurse

Health Promotion

Nurses have a role to play in making patients and family aware of the potential significance of changes in the pattern of symptoms and the importance of colonoscopic surveillance.

Surgical Intervention

The patient will require physical and psychological support during the pre- and post-operative periods. A greater degree of spiritual well-being may help to mitigate the demands of illness imposed by colorectal cancer according to a study by Fernsler *et al.* (1999). They also point out that younger patients with colorectal cancer may experience more intense illness-related problems than older patients. It is important for nurses to be aware of this and to plan appropriate assessment and interventions accordingly.

Side Effects of Treatments

Patients having radiotherapy or chemotherapy are likely to experience one or more of a range of common physiological toxicities and a range of psychological reactions. In addition to the patient's response to the disease itself, nursing assessment, within a structured theoretical framework, enables monitoring and recording of toxicity. This enables prompt nursing interventions to resolve problems and alleviate patient distress. Information and support are key elements of the nursing role, enabling the patient to prepare for the experience of toxicity, to take preventative measures and to develop coping strategies (Campbell and Lunn, 1999).

Colorectal Cancer Nurse Specialist

In many cases the colorectal cancer nurse specialist takes a technical role and is involved in screening and follow up endoscopy. Other key functions involve health education and the provision of information prior to screening and following diagnosis (Campbell and Borwell, 1999).

Conclusion

Patients with IBD have an increased risk of developing colorectal cancer. If the disease is detected at an early stage it is easily treated and potentially curable. People with colorectal cancer have specific needs at each stage of the disease and as patients will be nursed in a variety of settings. It is important that all nurses who come into contact with them have a clear understanding of the nature of the disease, its treatment and impact upon the patient and his or her family. The specialist nurse has an important role to play in providing expertise and continuity of care for the patient and family. This allows for sensitive issues around sexuality and continence to be discussed (Campbell and Borwell, 1999). A holistic and individualized approach to care will take account of the potential impact that a diagnosis of colorectal cancer may have upon a person who has already experienced perhaps many years of IBD.

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