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Progress report

The irritable bowel

There is no subject other than the weather so prone to uninformed comment as bowel function. Ignorance notwithstanding, gut disorder is a significant health problem. There are more physician visits in the United States for the irritable bowel syndrome than for inflammatory bowel disease and the days in hospital are similar in the two conditions.[1] The irritable bowel syndrome is the most common disease category seen by gastroenterologists.[2] This review will discuss the irritable bowel stressing developments which have occurred since 1978.[3]

Prevalence

No practising physician would deny that the irritable bowel syndrome is common. In Bristol, 14% of 301 adults who were *not* patients said they had abdominal pain relieved by defaecation more than six times per year.[4] These subjects had the following symptoms more often than others: altered frequency and consistency with pain onset, distension, mucus, and a feeling of incomplete evacuation after defaecation. These symptoms appear to be more common in the irritable bowel syndrome than in organic gastrointestinal disease.[5] A further 7% had abdominal pain unaffected by bowel movements, 6% had straining greater than 25% of bowel movements (constipation), and 4% had loose, runny stools greater than 25% of bowel movements (diarrhoea). Thus 30% of this group of people seemed to have gut dysfunction. Similar figures have been reported by Drossman[6] and Whitehead.[7] More accurate epidemiology must await a survey of a true random sample of the population and a precise definition of the disorder.

It is of note that only 20% of subjects with gut symptoms had ever seen a doctor about them.[4] We do not know why this minority sought medical attention, but it is unlikely that severity of symptoms was the only factor. Worry about serious disease, anti-cancer propaganda, and the availability of free health care lead many to consult a doctor. Mistrust of medical opinion and unrealistic expectation of treatment may generate repeated consultations especially in Canada and the United States where it is easier than in Britain to be referred to several specialists.

Quest for definition

If epidemiology and clinical studies are to progress there is need for a clear definition of the irritable bowel syndrome. The literature before 1940 emphasised the presence of mucus in the stools as a marker.[8–10] Several writers have been satisfied to define the irritable bowel syndrome by what

it isn't: that is not organic disease. Obviously it is important to establish that no structural or biochemical abnormality exists, but there must be more emphasis on what the irritable bowel syndrome is. As there is no agreed upon pathophysiological marker we must define the irritable bowel syndrome by its symptoms.

The irritable bowel syndrome is not a single, homogenous syndrome. There appear to be several subgroups, and these may have very different pathophysiologic relationships (Table 1). The spastic colon, for example, consists of abdominal pain usually related to defaecation, scybalous constipation with an empty rectum, and sometimes periods of diarrhoea. Diarrhoea may occur without pain or constipation, and constipation may occur with an atonic colon and rectum full of stool. These are very different from gas and chronic abdominal pain unrelated to bowel habit. Clearly these disparate symptom complexes should not be lumped together when searching for psychologic, pathologic, or physiologic markers of the irritable gut. Furthermore, treatment may be different for each syndrome. For therapeutic trials, the type of patients participating should be clearly defined if we are to detect useful treatment applicable to each group. A working group, sponsored by the US National Institutes of Health, is currently striving to define and classify these syndromes.

Undoubtedly those conditions which we now call functional will some day have a rational explanation. Before the recognition of lactose intolerance,[11 12] patients with that disorder would have been considered to have the irritable bowel syndrome. Idiopathic bile salt malabsorption is another organic explanation for a syndrome previously thought to be functional.[13] Precise definition of the clinical syndromes of the irritable gut is an important step towards recognition of other psycho-patho-physiologic markers in this group of disorders.

Search for a pathophysiologic marker

MOTILITY

Resting sigmoid tone is often decreased in diarrhoea and increased in constipation.[14 15] One can imagine the sigmoid behaving as a sphincter holding back stool in the constipated. In those with diarrhoea a lax sigmoid sphincter might allow liquid faeces to trickle through into the rectum where it prematurely triggers the defaecation reflex. Unfortunately this neat hypothesis does not hold up in many cases. Whitehead found more frequent fast contractions in diarrhoea dominant irritable bowel syndrome patients than those with constipation.[16] His patients, however, had pain while those in other studies tended to have painless diarrhoea. The colon moves in ways too subtle and complicated to be accurately assessed by our primitive methods. The proximal colon and small bowel are even more difficult to observe yet no doubt play an important role in the genesis of disordered bowel habit and pain.[17 18] One study suggests that small bowel

Table 1 *Syndromes of the irritable gut*

1 Spastic colon	3 Painless diarrhoea
2 Constipation – spastic	4 Gas
–atonic, painless	5 Chronic abdomen

transit is more rapid in patients with diarrhoea because of the irritable bowel than in controls.[19]

Some patients suffer postprandial abdominal pain and distension which is accompanied by exaggerated sigmoid pressure responses.[20 21] This 'gastrocolonic response' is mediated by afferant neural receptors in the gastroduodenal mucosa, efferant cholinergic neurones, and opiate receptors.[22] It is believed that segmental colon contractions cause localised obstruction, proximal dilatation, and abdominal pain. Ritchie inflated balloons in the rectums of volunteers and irritable bowel syndrome patients and thereby reduced abdominal pain, urgency, and gaseousness.[23] Although the pain was usually hypogastric it could be felt anywhere in the abdomen. Discomfort occurred with less balloon distension in those with irritable bowel syndrome. Swarbrick more recently showed the effect of balloons inflated at various points throughout the colon.[24] In 48 patients with the painful type of irritable bowel the pain could be reproduced in any part of the abdomen and in 29 of these the induced pain was of the same quality and site as the presenting complaint. Many physicians will be surprised to learn that distension of the colon can produce pain in the back, shoulders, sacro-iliac region, thigh, and perineum.[24] This reminds us of older work in which distension of the splenic flexure reproduced chest pain.[25] It is noteworthy that colon distension usually produces pain locally but can do so anywhere in the abdomen and beyond.

It is of further interest that asymptomatic patients with diverticular disease have a normal response to balloon distension while those with symptoms have a response similar to those with irritable bowel syndrome.[26] We found that among patients referred for barium enema, symptoms of the irritable bowel syndrome were equally prevalent in normal subjects and in those with uncomplicated diverticular disease.[27] These results suggest that the symptoms of uncomplicated diverticular disease are those of a coexistent irritable bowel.

MYOELECTRIC ACTIVITY

Smooth muscle electrical activity in the rectosigmoid of man consists of two basic, intermittent slow wave rhythms: one about three cycles per minute and the other six to 12 cycles per minute. These appear to originate from the inner or circular smooth muscle layer. Snape and his colleagues[28] found that the three cycle per minute activity was greater in their subjects with the irritable bowel syndrome than in controls. They later provided evidence that this three cycle per minute electrical activity was associated with increased three cycle per minute motor activity.[29] Taylor and others have confirmed this and found that the abnormal electrical rhythm persisted during asymptomatic intervals.[30]

Presenting contrary evidence, Latimer[31] compared colonic contractions and slow wave frequency in controls, irritable bowel patients with pain, and psychoneurotic patients with no bowel symptoms yet psychometric criteria similar to the irritable bowel syndrome group. At one recording site, the number and duration of contractions in the irritable bowel syndrome group were greater than normal subjects yet in all other motility and electrical criteria they were alike. The irritable bowel syndrome group did not differ significantly from the psychoneurotic group by any motility or electrical criterion.

French investigators used a different technology to record electrical activity throughout the colon.[32] They found two types of action potentials: long spike bursts and short spike bursts. The former may originate in longitudinal muscle, while the latter are action potentials superimposed on the slow waves originating in circular smooth muscle. In contrast with the slow waves these spike bursts correlated with mechanical activity in the colon. Short spike bursts of electrical activity coincided with abdominal pain and were more plentiful in the painful, constipated patient than in controls. In contrast, they were diminished in patients with painless diarrhoea.

This area now seems confused and will remain so until methodology is simplified and standardised. Some studies are done with empty rectums, some others not. Diet is not controlled. The exact symptomatology of the irritable bowel syndrome group studied is unclear and patients who enter such studies are highly selected. Recording sites and equipment are variable. Finally, in the words of Latimer, 'there is no convincing physiological model to explain how the finding of an abnormal electrical control activity can account for the clinical features of the irritable bowel syndrome'.[31] Much more research and collaboration is necessary before we can accept an abnormal electrical control activity (slow or fast) as a physiologic marker of the irritable bowel syndrome.

ENDOCRINE

One mechanism of disordered colon activity might be abberant gut hormone secretion or sensitivity. Diarrhoea may result from thyrotoxicosis, carcinoid glucogonoma, islet cell tumours of the pancreas, or medullary carcinoma of the thyroid.[33] Constipation has been associated with hyperparathyroidism and hypothyroidism. Harvey and Read showed that cholecystokinin increased colon contractions and reproduced postprandial abdominal pain when administered to irritable bowel syndrome subjects.[34] Although physiologic concentrations of cholecystokinin stimulate colon contractions which are not inhibited by atropine, this hormone does not appear to mediate the 'gastrocolonic response'.[35] Valberg reproduced right upper quadrant abdominal pain in young women with cholecystokinin injection yet no gall stones were present.[36] He concluded that this is a useful test for 'biliary dyskinesia'. It is, however, by no means certain that it is the gall bladder that is at fault here. Cholecystokinin might just as likely cause contraction of the common bile duct or even the colon.

Motilin apparently stimulates the myoelectric complexes in the small bowel.[37] Adrenal medullary activity is increased in 'nervous diarrhoea',[38 39] yet irritable bowel syndrome subjects have increased sigmoid sensitivity to parasympathomimetic drugs.[31 40 41] One study measured fasting and postprandial blood concentrations of several gut hormones by radioimmunoassay.[42] All irritable bowel syndrome patients had pain, but it was unclear if they had symptoms at the time of the study. At any rate no differences were detected between patients and controls. Apparently there is no simple gut hormone profile that will help us identify irritable bowel syndrome patients.

There is a need for further study during symptoms or after manipulations such as colon distension, physical, or mental stress. The endocrine

responses should be correlated with other physiologic features such as motility and myoelectric activity. Little is known about the roles of local paracrine, neurotransmitter or intracellular messenger substances.

Search for a psychological marker

It is the common experience that acute emotion affects gut function. Most of us respond to interviews, examinations, or tragedies with a variety of gut symptoms ranging from 'butterflies' through diarrhoea to vomiting. Acute emotion or stress may alter both colon[14] and small bowel function.[18] It is widely believed that chronic emotion or stress may be responsible for irritable bowel syndrome symptoms in some people. It appears, however, that a given emotion may elicit different responses in different subjects.

Many papers have been published which seem to show that patients with the irritable bowel syndrome are more neurotic, anxious, or depressed than others.[39 42–48] Most of these studies suffer from three defects. The first is that most individuals suffering with functional gut complaints do not see a physician.[4] Those who consult a doctor, become referred to a specialist, and then submit themselves to psychological testing, must be a small subset of irritable bowel syndrome sufferers whose very neurosis may have brought them to a physician. As mentioned, Latimer found similar gut motility and myoelectric abnormalities in irritable bowel syndrome subjects and patients with psychoneurosis without an irritable bowel.[31] Secondly, few of these studies attempt to classify the irritable bowel syndrome into subgroups which may have different psychologic and physiologic implications. Thirdly, it has been pointed out that many of the psychometric tests used in these studies have yet to be validated.[49]

A study by Whitehead[16] suffers from similar difficulties in patient selection. He does, however, attempt to differentiate painful diarrhoea from painful constipation. In this instance no psychologic differences could be detected. Further, although psychopathology was shown in irritable bowel syndrome patients, it could not be quantitatively correlated with motility or the severity of symptoms.

In an attempt to circumvent these problems, Whitehead[7] conducted random telephone interviews in Cincinatti. Subjects were said to have the irritable bowel syndrome if they had abdominal pain or gaseous distension, and constipation or diarrhoea in the previous year without an organic diagnosis. These representatives of the general adult population might be considered to have the spastic type of irritable bowel syndrome. Compared with subjects who said they had peptic ulcer disease or the remainder of those interviewed, this group had more somatic complaints, viewed cold and flu more seriously, consulted physicians more often for minor complaints, and as children were more likely to have been pampered by their families when ill. Whitehead concludes that irritable bowel syndrome sufferers are more prone to chronic illness behaviour and that this behaviour is learned.

Thus we remain uncertain whether the role of psychoneurosis in the irritable bowel syndrome is one of cause, effect, or in some cases coincidence. Although the importance of attending to the emotional problems of patients with the irritable bowel syndrome is unchallenged, much more work is necessary before we can call the condition a

psychoneurosis. Almy[50] points out that we have little understanding of the 'true nature of the irritable bowel syndrome, in particular whether it represents a qualitative or merely quantatitive departure from the psychophysiological reactions of normal people'.

Search for a cause

FIBRE

Hippocrates was aware of the stool bulking effect of bran and several scientific studies in the 1930s confirmed this.[51] It was the work of Burkitt and his colleagues a decade ago, however, that drew attention to the small, slow moving stools of Western societies compared to those of African natives.[52] He blamed this phenomenon on our fibre-free, processed foods. The ensuing 'fibre hypothesis' attributes many Western diseases to this phenomenon. Constipation, and the irritable bowel were said to be rare or non-existent in Africa. Largely on the assumption that fibre prevents an irritable bowel, bran has become popular as a treatment. It should be pointed out that bran, which tends to be the chief vehicle of Western fibre, is different chemically from that found in the African diet. Furthermore, we cannot be sure from any published work that the irritable bowel syndrome indeed does not exist in Africans.

It seems likely that fibre deficiency is not the cause of the irritable bowel syndrome, but may be a contributing factor in many cases. Certainly a complete and lasting cure cannot be guaranteed through fibre replacement. One recent study showed that although fibre consistently increased stool output in 21 healthy volunteers, personality factors were also important determinants of stool production.[53] An outgoing personality plus a positive self image were associated with high stool output. Those with low output had a greater increase after ingesting bran.

FOODS

Bran occasionally increases irritable bowel syndrome symptoms. This led one group to search by means of elimination diets for specific food intolerance.[54] Of 21 patients with abdominal pain and diarrhoea owing to the irritable bowel syndrome, 14 were found to be intolerant to one or more of wheat, dairy products, coffee, tea, and citrus fruits. The authors found no evidence of an immunologic cause for these intolerances, but rectal prostaglandin E_2 concentrations rose after ingestion of the offending food in those who developed diarrhoea. If confirmed, this is very important information. The procedure reported, however, is lengthy. In most irritable bowel syndrome patients, diarrhoea is not a predominant feature. Therefore this mechnism may apply to the diarrhoea phase of the illness, or to a subgroup of irritable bowel syndrome sufferers. In 1965, it was thought that lactose intolerance would explain many cases of the irritable bowel syndrome,[11 12] but that has not proven to be true.

FAT (GASTROCOLONIC RESPONSE)

We need to know more about the effect of food components on the colon. The so-called gastrocolonic response has been recognised but its mechanism has not.[21] Individuals with abdominal pain after meals have an exaggerated sigmoid pressure response which coincides with pain.[20 21] At

the University of Pennsylvania it has been shown that myoelectric activity and motility increased concomitantly after meals, and that the magnitude of this increase was proportional to the caloric levels of the meal.[55] The postprandial colonic electrical spike and motor activity was prolonged in the irritable bowel syndrome.[56] Snape and his colleagues later showed that the gastrocolonic response to a standard meal was because of its fat component.[57] The early gastrocolonic response was inhibited by an anticholinergic agent suggesting that it is mediated by a neural or cholinergic mechanism.[22 58] Fat also caused a delayed peak of activity which was inhibited by the concomitant administration of protein or aminoacids. This late response may be transmitted by a hormone.[34 57] These observations suggest that a low fat, high protein diet may benefit irritable bowel syndrome patients whose pain predictably follows meals.

DRUGS

Drugs are so commonly used in modern life that we cannot ignore their potential to disturb bowel function. Antacids, antibiotics, beta blockers, and narcotics all have well known adverse effects on the gut which trigger irritable bowel symptoms. Many patients choose to conceal their use of laxatives from their physician.[59] Of 27 patients with unexplained diarrhoea referred to one centre, the cause was found to be surreptitious use of laxatives in seven and diuretics in two.[60] None proved to have one of the rare endocrine causes of diarrhoea.

DYSENTERY

In their classic study of the irritable bowel Chaudhary and Truelove[40] found that one quarter of patients dated their symptoms from an attack of gastroenteritis. They stated that they responded favourably to treatment perhaps as no serious underlying psychologic or physiologic abnormality existed. Although this damaged gut syndrome seems well recognised I know of no scientific approach to the matter.

Approach to rational diagnosis

HISTORY

Most irritable bowel syndrome patients can be recognised from the history. How do physicians accomplish this? Certainly the lack of clues of organic disease is important. Severe weight loss, blood in the stool, anaemia, or fever cannot be explained by the irritable bowel syndrome. Bright red blood on the outside of stools may reflect haemorrhoids or fissure.[61] Longstanding symptoms in a well nourished patient, and lack of family history of ulcer or cancer are important as well. There may also be non-verbal communication that assists the experienced physician.

We must, however, strive for more positive features of the irritable bowel. Pain relieved by defaecation, looser and more frequent stools with onset of pain, abdominal distension, mucus in the stool, and a sensation of incomplete evacuation after defaecation seem to be more frequent in spastic colon subjects than in those with organic disease[5] (Table 2). The more of these symptoms that are present, the more likely is the subject to have the irritable bowel.

Although the syndrome seems to be equally prevalent in men and

Table 2 *Symptoms found more frequently in irritable bowel syndrome (spastic colon type) than organic disease*[5]

1 Pain relieved by defaecation	4 Abdominal distension
2 More frequent stools with pain onset	5 Mucus in the stool
3 Looser stools with pain onset	6 Feeling of incomplete evacuation after defaecation

women,[4] most gastroenterologists report that their patients tend to be women, especially young ones. The high prevalence of proctalgia fugax in irritable bowel syndrome subjects may be due to this predominance of women among patients.[62 63] In India, where women are discouraged from seeking medical help, more men are seen with the irritable bowel syndrome.[64] Headache[65] and backache[61] are said to be surprisingly common. Decreased lower oesophageal sphincter pressure and abdominal oesophageal contractions have been noted in patients with the irritable bowel syndrome when compared with controls.[66] Heartburn and globus, however, seem to be no more common in irritable bowel syndrome subjects than in normal subjects.[61 67]

EXAMINATION

Physical findings are few. Some have noted tenseness and anxiety with cool clammy hands, neurodermatitis and brisk reflexes,[68] but these do not seem to be specific for the irritable bowel. They may not be present in those who do not see a specialist. The sigmoid colon may be palpated in the left lower quadrant of the abdomen.[69] Fielding has presented evidence that the following are irritable bowel syndrome signs: excessively tender and palpable colon, pain on rectal examination, empty or nearly empty rectum, and hard or firm faeces.[68] Also tapping the posterior rectal mucosa on digital examination produced pain in 70% of irritable bowel syndrome subjects compared with 5% of controls. It should be noted, however, that 72% of Fielding's irritable bowel syndrome subjects were women as opposed to only 51% of controls. The right iliac fossa squelch sign is described as a sensation on palpation of a 'fine deep-seated surgical emphysema nearly always accompanied by a squelching sound'.[70] Fielding observed it in 18 of 50 new outpatients all of whom had diarrhoea. When the patients improved, the squelch disappeared. This sign may turn out to be one of diarrhoea due to any cause. Abdominal scars are more common than in the general population and the operative notes may indicate adhesions, uterine suspension, or removal of a normal gall bladder or appendix.[40 71–73] The patient seldom exhibits any signs of chronic disease such as malnutrition, anaemia, or fever.

Sigmoidoscopic examination is important as much, I believe, for therapeutic as diagnostic ends. No patient can see his colon and many can be reassured by someone who has. A vigorously contracting sigmoid supports a diagnosis of spastic colon. On occasion insufflation of air into the rectum may reproduce the patient's pain. A full rectum which fails to stimulate the defaecation reflex may indicate atonic constipation. The presence of melanosis coli, a brown pigmentation of the mucosa, is a telltale sign of chronic laxative abuse.[59 74] A negative examination, even if it has no real diagnostic value, may have placebo effect.[75]

INVESTIGATION

The irritable bowel syndrome would not present much of a problem to physicians were it not for the fear that the symptoms might represent organic disease. Of course, the irritable bowel syndrome does not provide any insurance against organic disease, so the physician must be wary. In the young, inflammatory bowel disease is the main concern. Careful abdominal examination and sigmoidoscopy should detect most cases. If the haemoglobin, white blood count, erythrocyte sedimentation rate, and temperature are normal and the symptoms are typical of the irritable bowel, no further tests may be needed.

Dyspareunia, or irregular menses might indicate a gynaecologic examination.[76] In patients over 40, the risk of an occult cancer is such that most physicians will insist on an air contrast barium enema or colonoscopy at the initial visit. It should be repeated thereafter only if disease is found, there is a family history of carcinoma, or if the symptoms change. Among patients referred for barium enema, irritable bowel symptoms are similarly prevalent in normal subjects and in those with uncomplicated diverticular disease.[27] The latter are often asymptomatic[77–79] and when symptoms do occur they may be those of a coexistent irritable bowel. The presence of fever, leucocytosis, and severe abdominal pain with tenderness and guarding may indicate the presence of a complication of diverticular disease such as a pericolic abscess. Epigastric pain, especially if it goes to the back and is present day and night, might indicate carcinoma of the pancreas. Ultrasound, computerised tomography, or endoscopic retrograde pancreatography, and cytology may be helpful in this difficult situation depending upon the availability of the tests and the degree of suspicion. Even these tests are fallible.[80]

What is not indicated in the investigation of the irritable bowel is a shot gun approach with barium enema, gastrointestinal series, small bowel enema, cholecystogram, intravenous pyelogram, and CT scan on every patient with gut symptoms. Not only does this cost a great deal and present a radiation hazard for patients, but the lack of precision reflects the doctor's uncertainty thereby undermining the patient's confidence.

Treatment

GENERAL

Satisfactory management of the irritable bowel syndrome demands much of the art and science of medicine. It is a common experience that lasting cures are unusual. In a prospective study of 50 patients on a variety of treatments there was remarkably little change in symptoms a year later.[81] Thus the physician's duty is to help the patient to understand and cope with his symptoms and to avoid any therapy which might be harmful.

Treatment begins with the history and physical examination. One should ensure that the patient's symptoms are taken seriously. I doubt if any doctor ever says, 'It's all in your head!', but some patients get that message anyhow. Questions during the interview about diet, stress, emotional state, and drugs help draw the patient's attention to these factors, and prepare him for the advice which will be offered later. A thorough examination and crisp investigation reassures the patient that nothing is overlooked.

Fear of cancer is frequent in the irritable bowel, and the stress it

generates may actually aggravate the symptoms.[71–73] Even if not mentioned by the patient, such anxiety about serious disease is often on his hidden agenda. It must be firmly laid to rest as soon as the physician feels sufficiently confident to do so.

PLACEBOS

From various irritable bowel syndrome drug trials, it is apparent that over one third of patients benefit from placebos.[82–84] This has several implications. First, no potentially harmful therapy should be accepted unless it has been shown to be better than placebo. Secondly, there may be situations where pure or impure placebos may be of benefit, although some degree of deception is involved. Finally, there is great therapeutic benefit to be derived from the physician–patient encounter itself.

It may be very difficult to prove drug efficacy. For example, there have been many drug trials of the efficacy of anticholinergics in the irritable bowel syndrome, and reviewers have concluded that any short term benefit over placebo does not justify the expense, unwanted effects, and pill taking psychology that such therapy engenders.[3 82 83] In the United States pharmaceutical houses include a note in their advertisements stating that anticholinergics are 'possibly effective' in the irritable bowel syndrome. Nonetheless, some physicians find these drugs useful in certain circumstances.

Lack of classification of the irritable bowel may result in the lumping of disparate syndromes into a trial so that efficacy in certain instances may be hidden by lack of efficacy in others. Whatever the explanation, few if any, treatments have been shown to be better than placebo.

This brings us to the second implication of the placebo response. Many physicians cannot accept the deception implied by the use of a deliberate placebo.[75] It seems that if a pure placebo such as a sugar pill is to work, the patient should believe that there will be pharmacological effect. This thesis may not be entirely true, as controlled clinical trials demand informed consent and yet the placebo is still effective. In a small number of neurotic patients, a placebo was effective when the patients knew the pills that they were given were inert.[84] It seems that the symbolic giving of pills has therapeutic value. The use of impure placebos – that is, drugs whose effects are not due to their pharmacologic properties – risks double deception if both patient and physician come to believe in their efficacy.[85]

The third issue is perhaps the most important. Brody[75] says that there are important symbolic elements of the physician–patient relationship. 'A clinical approach that makes the illness experience more understandable to the patient, that instills a sense of care and social support, and that increases a feeling of mastery and control over the course of the illness will be most likely to create a positive placebo response and to improve symptoms.' Patients who received preoperative explanations of incisional pain, reassurance that backup medication was at hand, and pain avoidance techniques required half as much medication and were discharged earlier than a cohort of patients undergoing similar operations who received only the standard preoperative anaesthetic visit.[86] Such a doctor–patient interaction involves no deception and should be the cornerstone of the treatment of the irritable bowel syndrome. With the use of diagrams, the patient can be helped to understand how gut symptoms occur.

Contributing factors gathered during the interview can be discussed, and the fear of serious disease obviated. Only in this way can the patient be expected to deal rationally with his symptoms and not to shop from physician to physician, or physician to charlatan seeking a cure for the incurable.

DIET

There is evidence for[87–90] and against[91 92] the use of bran or other bulking substances in the irritable bowel syndrome. The most convincing study was in patients with symptomatic diverticular disease[87] whose symptoms may be assumed to be those of a coexisting irritable bowel.[27] All of these studies have imperfections, however, lack of definition of the syndrome, varying doses or types of bran, and a short period of follow up. Nevertheless, fibre does shorten transit time and increases stool bulk.[52] It seems to be harmless, and many gastroenterologists believe it is useful,[93 94] particularly in patients whose dominating symptoms are constipation and hard stools.[95]

One tablespoon full of raw bran with each meal is a good starting point. The patient must take it regularly and over several months, before failure is admitted.[87] There will be bloating in some, and some will claim that bran is intolerable. In these cases psyllium powder seems like a reasonable alternative although here again efficacy may depend on the placebo effect.[96] It should be recalled that psychological factors apparently affect stool output as well as fibre.[53]

From the experimental work cited above,[57] a low fat diet, or a diet rich in protein might be expected to benefit some patients with postprandial abdominal pain due to the irritable bowel. Dietary intolerances such as that due to lactose[11 12] should be recognised, but some are difficult to identify unless one conducts an elimination diet (see under Search for a cause). Many patients repeatedly blame their symptoms on a previous meal leading to unreasonable exclusions. This irrational fear of food must be addressed.

DRUGS

I know of no controlled studies suggesting that tranquillisers or antidepressant drugs by themselves are beneficial in the irritable bowel syndrome. Clearly, if the patient is anxious or depressed such therapy may be indicated for those reasons. As pointed out, however, the irritable bowel is often a life-long condition.[81] Therefore one should avoid long term use of mind affecting drugs.

As mentioned above, the irritable bowel syndrome does not consistently respond to anticholinergics.[3 82 83] Recent publication of small, short term clinical trials do not settle this issue.[97–100] Because of the smooth muscle relaxing properties of these drugs, however, many physicians believe that postprandial pain when due to a spastic colon might be alleviated by their administration before meals.[56 83] Such use takes advantage of the drug's short duration of action and minimises side effects. This particular subgroup of patients with postprandial pain is not common, and most patients will not benefit from these drugs. Dicyclomine is said to act by directly relaxing smooth muscle rather than by blocking acetylcholine.[101] In one trial, however, anticholinergic side effects were noted in 69% of

patients receiving the drug.[98] It may be that anticholinergics are more effective when combined with a bulking agent.[100–102]

Some patients with predominant and persistent diarrhoea may benefit from antidiarrhoeal medication such as diphenoxylate[104] or loperamide.[105] A report that cholestyramine might benefit some individuals with the diarrhoea type of irritable bowel appeared in 1970.[106] It now appears that the rare responsive patient has idiopathic bile salt malabsorption.[13] Carminative oils such as peppermint are known to have a smooth muscle relaxant effect.[107] In a small double blind cross over trial, 0·2 ml of peppermint oil in gelatin capsules was found to be superior to placebo in alleviating symptoms over a three week period.[108] There are conflicting reports of the usefulness of dopamine receptor blocking drugs in this condition,[109 110] but the beta blocking drug timolol appears to be of no benefit.[111]

Many other drugs and drug combinations have come and gone but none have shown consistent pharmacologic efficacy. It is likely that the psychic, humeral, and neural interactions that lie behind the irritable bowel syndromes are so complex that no drug can be expected to have a lasting, broad-spectrum effect.

Conclusions

The irritable bowel syndrome and its variants appear to affect about one third of the population, but most sufferers do not see a doctor. Progress in our understanding of this disorder is hampered by imprecise definitions, and the lack of a pathophysiologic marker. There is evidence of abnormal gut motility and myoelectric activity, and a suggestion that nerves and hormones play an important role. Diet, drugs, emotions, and infections are undeniable, but variable, contributing factors. While academicians grapple with aetiology it is the physician's duty to precisely and positively diagnose the syndrome, so that he may explain and reassure. The irritable bowel syndrome is a great problem to doctors and patients because of the worry that symptoms might indicate serious pathology such as inflammatory bowel disease or cancer.[112] The short term therapeutic response to placebo is very high and no diet or medication consistently outperforms it. Bran and other bulking agents seem safe and are probably most effective when constipation is present. Peppermint oil shows some promise, and anti-cholinergics may be tried in persistent postprandial symptoms. It is most important, in this lifelong condition, that the risks of investigation and treatment not excede those of the disease. As only a minority of irritable bowel syndrome sufferers bring their complaints to a physician it is important to find out why the patient consults. The stressed, the unloved, and the cancer-phobic will have very different needs in diagnosis and in treatment.

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