Disorders of colonic motility are thought to play a part in several diseases, especially the irritable bowel syndrome, and also ulcerative colitis, diverticular disease, and constipation. The genesis of abdominal pain or disordered bowel habit is generally ascribed to abnormal colonic motor function. Despite research contributions from many countries the normal and pathological motor function of the colon remains poorly understood. This is due in part to the relative inaccessibility of the organ, its regional differences in structure and function, and to the capricious nature of motor events in the large bowel. It is difficult to measure colonic function simultaneously in its various parts and it is erroneous to extrapolate findings from one part to another. Ritchie hoped that manometry would prove a 'safe and reliable method of studying colonic movements' and in 1965 Deller and Aveling expressed the view that understanding of intestinal motility may progress if pertinent problems were recognised. They pointed out that motility patterns in normal subjects needed to be described in more detail, that the relation between pressure and transit needed to be further investigated, and that the analysis and classification of pressure waves had to be standardised and quantified. Although some of these points have been considered, our understanding of colonic function has not increased much and measurements of colonic pressures have not found their way into routine clinical practice; measurements of transit have a more practical value in selected patients.

Why is it that clinical usefulness of colonic pressure measurements remains elusive? The answer is partly methodological and partly dependent on the physiology of colonic smooth muscle. Colonic motility can be conveniently considered under two related aspects, intraluminal pressure activity and transit of intraluminal contents. The second has been measured by studying the movements of barium, or radio-opaque shapes and more recently radioactive isotopes. Radio-opaque shapes are clinically the most useful for quantifying colonic transit; movement of barium involves unacceptably high doses of radiation and radioisotope studies need access to imaging equipment and the patient has to be immobilised during the scans. Formulae have been devised to measure segmental colonic transit with radio-opaque shapes. The need for repeated abdominal radiographs can be avoided by a modification of this technique and segmental transit can be measured from a single x-ray film taken after the serial ingestion of radio-opaque shapes for three days. This minimises exposure of the patient to radiation. If the stools, rather than the patient's abdomen are screened for the shapes, there is no radiation risk but this approach is inconvenient. Slow transit constipation can be distinguished from outlet obstruction by the ingestion of a capsule containing 20 shapes followed by an abdominal x-ray film taken on the fifth day; 80% retention of the markers signifies slow colonic transit. These simple methods can be used clinically to help establish a diagnosis and to measure the effect of treatment; they are also useful in research.

Colonic pressures can be measured with balloons, continually perfused catheters, freely mobile pressure sensitive radiotelemetry capsules, or solid state tube mounted strain gauges, each measuring different aspects of colonic motor function. Most studies use water perfused catheters. Solid state transducers have the important advantage that they can be used in the ambulatory patient and are becoming readily available. Hardware and software exist for prolonged computerised collection (including the ambulant patient) and analysis of data. The positioning of the recording devices in the colonic lumen and cleansing of the bowel before pressure measurements remain non-standardised. It seems logical to study unprepared bowel in an unsedated patient, but this limits studies to the distal colon or requires invasive and time consuming intestinal intubation.

Morphology of individual colonic pressure waves has eluded reliable classification. This has led to the concept of the motility index (area under the time pressure curve) to describe pressure activity. This has the advantage of being expressed as a number and is thus amenable to statistical analysis, but it does not recognise different types of colonic pressure waves. As pressure measurements obtained from colonic probes 5 cm apart can be very different, some workers have summed the pressure activity from adjacent channels to obtain a value for the colonic segment under study as a whole in an attempt to standardise analysis.

Colonic motor function has also been investigated with electromyography, the electrodes being attached to the mucosa in the intact human. A background slow variation in electrical potential has been described (basal electrical rhythm) with superimposed shorter duration, larger amplitude potentials (spike potentials or electrical response activity). The basal electrical rhythm controls the occurrence of the electrical response activity, which is thought to correlate with colonic smooth muscle contraction.

Normal motility of the small intestine in the fasting state is characterised by the cyclical appearance of the migrating motor complex. This is a high amplitude burst of contractions that start in the stomach and are propagated distally into the lower small bowel. After eating, this regular coordinated activity gives way to irregular, non-propagated pressure
activity (phase II), or periods of quiescence (phase I). Unfortunately in the colon the situation is far less clear. Instead of orderly motility patterns, which can be analysed relatively easily, irregular contractions occur that are apparently randomly distributed in location and time. Low amplitude segmenting contractions coexist with contractions of higher amplitude that can be present simultaneously at points up to 10 cm apart, but do not propagate proximally or distally. This type of segmenting pressure activity does not propel intraluminal contents over large distances and is probably responsible for the mixing of bowel contents. High amplitude (100 and 200 mm Hg) contractions, travelling distally over distances of at least 24 cm have also been recorded. These are termed high amplitude propagated contractions, or giant migrating contractions and differ greatly from the segmenting pressure activity that normally predominates. The high amplitude contractions travel over relatively long segments of the colon and appear consecutively in the more distal bowel, suggesting that this activity is coordinated and propulsive. These contractions occur infrequently, between four and five times a day, so that their recognition needs prolonged periods of intraluminal monitoring. Alternatively, they can be stimulated with contact laxatives such as dulcolax. They are thought to represent the manometric equivalent of colonic mass movements that were first described by radiologists at the turn of the century.

The relation between segmenting or propagating pressure activity and transit can be investigated by simultaneously measuring intraluminal pressures and transit of intraluminal radionuclide markers with a gamma camera. The isotope most commonly used for measurement of colonic transit is 99mTc-DTPA in liquid form, or it may also be incorporated into ispaghula husk to simulate the physical properties of faeces more closely. In normal subjects in the fasted state segmenting pressure activity is low and little transit of marker is seen. After a meal segmenting pressure activity increases and antegrade and retrograde transit of marker occurs in association with this activity. Movement in these circumstances is over short distances and occurs down a pressure gradient. Giant migrating contractions are associated with transit of marker over larger distances in a distal direction. Insight into abnormalities of transit and pressure activity in constipation or diarrhoea has been obtained with these techniques. In functional diarrhoea there is more and fro movement of intraluminal contents in the fasting state than in normal controls. After eating, although intraluminal pressure does not increase appreciably, the number of high amplitude propagating contractions increases. By contrast, in patients with chronic constipation fasting transit of marker was not recorded. Moreover, there was no postprandial increase in segmenting colonic pressure activity, no propagating contractions, and no postprandial transit of marker. Patients with idiopathic chronic constipation have a decreased number and duration of giant migrating complexes than healthy controls.

Measurements of colonic pressures are confounded by pronounced intra and interindividual variation of colonic motility indices depending on factors that are obscure and thus difficult to control. They probably include the nature of the intraluminal contents, the emotional and metabolic state of the patient, and the location of the recording devices. This situation makes it difficult to use basal colonic motility as a basis for comparisons and has led to the development of provocation tests in a controlled environment in an attempt to characterise differences between groups of patients. Appreciable variations between subjects, combined with the small numbers investigated in these invasive and time consuming studies, compound the difficulties of showing statistically significant differences and establishing a normal range of values.

Awakening stimulates colonic pressures, showing a link between the central nervous system and the bowel. Apart from awakening, the most powerful and reproducible physiological stimulus to segmenting colonic activity is a meal. The meal needs to contain 1000 kcal or more and have a relatively high fat content (>40%). High amino acid content in the meal abolishes the colonic response. It is not abolished by gastrectomy, which is related to the entry of food into the upper small bowel, and has a neural component. A cephalic phase of this response has recently been described in normal subjects and in the irritable bowel syndrome, again emphasising the link between the 'big brain' (central nervous system) and the 'little brain' (myenteric and submucous plexuses) in the gut. Unfortunately the colonic response to a meal is not consistent even in normal subjects and its absence does not point to a specific lesion in the nervous system-colon axis. For example, it is absent in such diverse conditions as constipation, diabetes, multiple sclerosis, and thoracic spinal cord injuries.

Experiments on the effects of various gastrointestinal polypeptide hormones and drugs on colonic motility have sought further clarification of normal motility patterns or effective treatments for motility disorders. Many hormones have been implicated in the colonic response to food, including cholecystokinin, gastrin, motilin, neurotensin, pancreatic polypeptide, and polypeptide Y. Cholecystokinin increases rectosigmoid segmenting pressure activity when administered exogenously, and the response can be reproduced by the administration of intraduodenal nutrients known to cause release of cholecystokinin. Motilin, administered exogenously at physiological doses, also increases the distal colonic motility index but these results were not confirmed with the motilin agonist erythromycin, which had no effect on segmenting sigmoid pressure activity or colonic transit in it. Gastrin increases rectosigmoid activity, and pentagastrin does increase the number of sigmoid colonic myoelectrical spike potentials in normal subjects. Despite much work, the roles of alimentary hormones in the control of colonic muscle in health and disease are not yet clear. In the studies with gastrin and its analogues an often uncontrolled variable has been gastric acid secretion, although the presence of gastric acid is not essential for a colonic response as it is not abolished by total gastrectomy. The role of acid secretion has been investigated in relation to the cephalic phase of the colonic response to food by means of H2 receptor blockade and continual aspiration of gastric juice, but the cephalic response occurs independently of the presence or absence of acid or the entry of gastric juices into the duodenum.

As well as arousal from sleep and the complex effects of food and polypeptide hormones, neurotransmitters have been shown to affect colonic motility. Thus selective or non-selective β blockade stimulates segmenting pressures in the distal colon, suggesting that this part of the bowel is under sympathetic inhibitory control, even in an unstressed situation. The calcium channel blocker nicardipine decreases rectal postprandial motor activity in normal subjects. It also inhibits motor activity induced by rectal distention and increases sensory thresholds for defaecation in patients with the irritable bowel syndrome. The same drug given intravenously prevents the postprandial increase in sigmoid segmenting pressure activity in patients with irritable bowel syndrome. These data suggest that calcium fluxes play a part in the normal functioning of the rectum and colon and imply potential use in the treatment of this disease. Another possible way of controlling colonic pressures is through 5-HT3 antagonists such as ondansetron, a compound that slows colonic transit in normal subjects. Its main effect, however, is not on sigmoid segmenting pressure activity.
when given intravenously at doses of 2·5 or 10 mg (Amin Z, Misiewicz JJ, personal communication).

In line with these uncertainties measurements of colonic pressures in the irritable bowel syndrome produce contradictory data and no common underlying abnormality has been defined. Several years ago there was much interest in the finding of an apparently abnormal basal electrical rhythm in irritable bowel syndrome. In normal subjects there are two components to the basal electrical rhythm, a predominant rhythm at a frequency of 6 cycles per minute with some at 3 cycles per minute. In irritable bowel syndrome the 3 cycles per minute component was increased independently of changes in bowel habit, but further, so that what happens to colonic pressures at the time clustered contractions are present in the small bowel is not known. It is possible that many patients with irritable bowel syndrome have a pancellular motor disorder, and that extra-alimentary innervations of the autonomic nervous system, such as the bladder, are involved. 46, 61

Areas of uncertainty extend to other conditions. Colonic diverticular disease is thought to occur in response to a combination of colonic wall weakness and high intraluminal pressures. Support for this hypothesis consists of high pressures recorded in the basal and postprandial period, and after prostigmine, but some investigators have challenged these findings. 49-60

There has recently been a revival of interest in changes in colonic motility in ulcerative colitis. Early studies showed decreased segmenting pressure activity in this disease with absence of the normal postprandial pressure response although the normal postprandial increase in myoelectrical spike activity was retained, suggesting some sort of electromechanical dissociation. These findings are consistent with decreased transit in the proximal and rapid transit through the sigmoid colon in patients with active colitis. 54 Studies with an animal model of colitis confirmed the decreased segmenting contractile activity and showed increased giant migrating complexes, which were often associated with defaecation. 70, 71

Thus research into colonic motor function remains a challenging and potentially rewarding area where progress has been facilitated by recent technological advances. More precise knowledge of control of colonic motor function, coupled with basic research into the pathways that control colonic muscle, should eventually result in the development of drugs for modifying colonic motor function.

At present the use of colonic motility tests in clinical practice is limited and will remain so until normal ranges for colonic pressures can be defined and more effective treatments for the various motility disorders identified. In patients disabled by severe dysmotility syndromes, however, they sometimes provide the only objective evidence of abnormality. This can be useful in the management of persons driven to distraction by the relentless normality of all conventional tests in the face of severe abnormal symptoms.

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