

Recording of intestinal motility: routine or research?

In 1899 Bayliss and Starling wrote: 'On no subject in physiology do we meet with so many discrepancies of fact and opinion as in that of the physiology of the intestinal movements.' Regrettably, this remains valid today.

The methods that can be used for the routine study of the movements of the alimentary tract in man are radiology and manometry, but they offer only circumstantial evidence about events occurring in the muscle itself.

Radiology gives information about the calibre and outline of the alimentary tract and the movements of an artificial radio opaque bolus, but very little can be deduced about the force or strength of the muscle activity.

Manometry measures intraluminal pressures or the resistance of the bowel wall to stretch, and has the advantage over radiology that it permits periods of observation much longer than are possible with *x* rays. This is important as the movements of the intestinal wall are relatively slow. On the other hand, the pressure recorded from the lumen of the intestine is a function not only of muscular activity but also of the content of the bowel and the diameter of its lumen, so that information about muscular action obtained by this method may be clouded by the effects of other factors.

Intraluminal pressures can be measured accurately by open tubes and transducers passed into the lumen,¹ or by freely mobile pressure sensitive telemetering devices.^{2,3} Miniature balloons⁴ record pressures which approximate to that of intraluminal pressure and have the great advantage over open tubes of giving consistent recordings as they do not become blocked by bowel content.

The resistance to stretch of the muscle of the bowel can be estimated with a large balloon which, however, distorts physiological mechanisms by being an obstructive bolus in the lumen of the bowel and the recorded pressures by reason of its inherent elasticity.⁵ The resistance to stretch of the bowel wall is the nearest approximation which can be achieved to an estimate of 'tone'. Basal pressures are not helpful in estimating tone as the main contributor here is the hydrostatic force exerted by the tissues superficial to the recording device.^{6,7} Neither the recording of electrical potentials from the intestine nor our understanding of their significance is refined enough to be of practical use.

The pressure recorded from any segment of bowel is the resultant of the resistance to flow (including mucosal folding, the viscosity of the bowel content, and wall resistance to distension), and the forces increasing pressure in the segment.^{6,8} Most often, changes in intraluminal pressure indicate segmenting activity but progressive contractions of the bowel can raise the intraluminal pressure if they are of sufficient strength to force the content of the bowel against resistance or if they form a segment under tension as they sweep past the pressure recorder. It follows that intraluminal pressures more often denote resistance to transit than the presence of propulsive forces.⁸ In confirmation of this, it has been shown that, in general, intraluminal pressures are low in persons with diarrhoea where flow along the colon is free.^{9,10} Patients who are constipated tend to have normal or even excessive activity.⁹

THE OESOPHAGUS

Manometric procedures have uncovered mechanisms of movement at all areas of the gastrointestinal tract but so far they have found clinical application only in the oesophagus and to a lesser extent in the colon. Oesophageal pressure parameters are less complex than elsewhere in the gastrointestinal tract, in that the muscular activity in health is simple and predictable, the lumen is fairly constant, and, in the resting state, content is minimal. The intraluminal pressures reflect closely the activity

of the muscle itself. The lower oesophageal sphincter is accessible for accurate clinical testing, so that a background knowledge of pressure patterns in health and disease has developed, and many gastrointestinal centres study routinely the activity of the oesophagus and its lower sphincter.

A new dimension to our knowledge of the motor activity of the oesophagus, of the nature of oesophageal peristalsis,¹ of the occurrence of non-peristaltic contractions,¹¹ of the mechanisms of chest pain, and of the significance of the sphincters has been added by the application of manometric techniques. The lower sphincter exerts a closing pressure of only a few millimetres of mercury which is not enough of itself to resist the very high pressures which can be generated in the stomach.^{4,12-14} Indeed, using open-ended tubes, no significant high pressure zone has been detected in a proportion of persons,^{4, 12} but using miniature balloons resistance to stretch is always found.⁴ Thus, if a length of oesophagus is in place below the diaphragm, the sphincter maintains a closing force which keeps the mucosal surfaces of the oesophagus opposed, triggering a valve which can resist moderate gastric pressures.^{6,7,15,16} For control of gastro-oesophageal reflux two conditions must be satisfied: there must be a length of intraabdominal oesophagus, together with a pressure barrier at the gastro-oesophageal junction, although this need be minimal. Studies in dogs have indicated that if the sphincter is eliminated reflux occurs consistently even when a length of oesophagus remains in the abdomen.¹⁷ Conversely, in hiatus hernia, where the sphincter is displaced into the thorax, reflux can occur even when there is a good lower oesophageal pressure barrier.¹⁸

Manometry may reveal the disordered physiology in individual cases of hiatus hernia and reflux and can monitor the natural history of disorders such as diffuse spasm,¹⁹ but in the clinical context, has more to contribute in uncovering the cause of dysphagia. Dysphagia due to tonic or intermittent spasm of the lower oesophagus may be distinguished from that due to failure of the sphincter to relax, and typical pressure patterns in achalasia,²⁰ diffuse spasm,^{21,22,24,25} oesophagitis,²³ and visceral sclerosis involving the oesophagus have been well documented.¹ Pressure studies can be particularly valuable in early cases of achalasia in which the body of the oesophagus may not have dilated and where early operation, before disorganization of muscle has occurred, may be expected to produce a more satisfactory long term result. Spasm, achalasia, and incoordination can afflict the upper sphincter and manometry may show the quality and pattern of muscular disease although this sphincter has been less fully studied.^{26,27} However, those patients in whom manometry is decisive are few, and in most, good history taking and good radiology are still the keys to diagnosis.

COLON

In the colon the varying diameter of the lumen and the presence of a pultaceous content make interpretation of pressure records in terms of muscular activity more hazardous but, in general, the existence of pressure activity indicates resistance to flow;⁸ so that motility studies can guide the physician to discern if any given case of bowel dysfunction is a high or low pressure phenomenon. Thus, in diarrhoeal conditions, which are normally low pressure states, there would seem to be little rationale for the exhibition of preparations which will further diminish segmenting activity.

In the colon, pressure studies have been useful in outlining abnormal colonic mechanisms, especially in diverticular disease²⁸⁻³⁰ and the irritable bowel syndrome. In diverticular disease the muscle of segments bearing diverticula apparently generates greater intraluminal pressures than normal when stimulated by food or drugs such as prostigmine or morphine,³³ and it has been suggested that this hyperactivity is a factor in the pathogenesis of diverticulae. However, muscular activity in the wall of the bowel is more likely to result in intraluminal pressure changes when the lumen is narrow than when it is wide so that it is possible that some, at least, of the increased activity in diverticular segments is a function of the diameter of the bowel and does not necessarily represent greater muscular effort. Studies using miniature balloons, where the effect of luminal diameter is less important, do not show marked differences between the responses of normal subjects and patients with diverticular disease either to physiological or pharmacological stimuli.³⁰

In many patients with irritable colon there is also an increase in the pressure response to prostigmine.^{31,32} In some patients there is an exaggerated response to eating which can coincide with bouts of abdominal pain.³⁴ Whether this overactivity is a function of muscular hyperactivity or segmental narrowing, it can be interpreted fairly as representing resistance to transit. This, in turn, may cause segmental distension proximally which may contribute to the abdominal pain. In these relatively common disorders, radiological, bacteriological, and endoscopic examination may be unhelpful, but manometry can uncover the mechanism of the dysfunction and may spare the patient an untimely laparotomy.

A small but important use of pressure studies is in the characterization of idiopathic megacolon. This condition frequently presents as a low pressure state but in some patients normal activity can be promoted by the administration of prostigmine.³⁵ The response of the organ to cholinergic stimuli not only may be valuable in defining the nature of the muscular defect, but is necessary in the interests of logical treatment.³⁶

While pressure studies continue to contribute to our understanding of the mechanisms of functional disorder of the alimentary tract, clinical indications for intraluminal pressure studies are narrowly defined and further advance will only be made when data can be obtained directly from the muscle itself.

A. M. CONNELL

REFERENCES

- ¹Code, C. F., Creamer, B., Schlegel, J. F., Olsen, A. M., Donoghue, F. E., and Andersen, H. A. (1958). *An Atlas of Esophageal Motility*. Thomas, Springfield, Ill.
- ²Connell, A. M., and Rowlands, E. N. (1960). Wireless telemetering from the digestive tract. *Gut*, **1**, 266-272.
- ³Farrar, J. T., Zworykin, V. K., and Baum, J. (1957). Pressure-sensitive telemetering capsule for study of gastrointestinal motility. *Science*, **126**, 975-976.
- ⁴Atkinson, M., and Summerling, M. D. (1966). Oesophageal changes in systemic sclerosis. *Gut*, **7**, 402-408.
- ⁵Quigley, J. P., and Brody, D. A. (1950). Digestive tract: intralumen pressures: gastro-intestinal propulsion, gastric evacuation, pressure-wall tension relationships. In *Medical Physics*, vol. 2, edited by Glasser, pp. 280-292. Year Book Publishers, Chicago.
- ⁶Edwards, D. A. W. (1961). The anti-reflux mechanism: manometric and radiological studies. *Brit. J. Radiol.*, **34**, 474-487.
- ⁷Johnston, H. D. (1966). The fluid mechanics of the control of reflux. *Lancet*, **2**, 1267-1268.
- ⁸Connell, A. M. (1965). Significance of the pressure waves of the sigmoid colon. *Amer. J. dig. Dis.*, **10**, 455-462.
- ⁹— (1962). The motility of the pelvic colon. II. Paradoxical motility in diarrhoea and constipation. *Gut*, **3**, 342-348.
- ¹⁰Kern, F., Jr., Almy, T. P., Abbot, F. K., and Bogdonoff, M. D. (1951). The motility of the distal colon in non-specific ulcerative colitis. *Gastroenterology*, **19**, 492-503.
- ¹¹Cauthorne, R. T., Vanhoutte, J. J., Donner, M. W., and Hendrix, T. R. (1965). Study of patients with lower esophageal ring by simultaneous cineradiography and manometry. *Ibid.*, **49**, 632-640.
- ¹²Botha, G. S. M., Astley, R., and Carré, I. J. (1957). A combined cineradiographic and manometric study of the gastro-oesophageal junction. *Lancet*, **1**, 659-662.
- ¹³Dornhorst, A. C., Harrison, K., and Pierce, J. W. (1954). Observations on the normal oesophagus and cardia. *Ibid.*, **1**, 695-698.
- ¹⁴Fyke, F. E., Jr., Code, C. F., and Schlegel, J. F. (1956). The gastro-oesophageal sphincter in healthy human beings. *Gastroenterologia (Basel)*, **86**, 135-150.
- ¹⁵Creamer, B., Harrison, G. K., and Pierce, J. W. (1959). Further observations on the gastro-oesophageal junction. *Thorax*, **14**, 132-137.
- ¹⁶Johnston, H. D., and Laws, J. W. (1966). The cardia in swallowing, eructation, and vomiting. *Lancet*, **2**, 1268-1273.
- ¹⁷Vandertoll, D. J., Ellis, F. H., Schlegel, J. F., and Code, C. J. (1966). An experimental study of the role of gastric and esophageal muscle in gastro-oesophageal competence. *Surg. Gynec. Obstet.*, **122**, 579-586.
- ¹⁸Atkinson, M., Edwards, D. A. W., Honour, A. J., and Rowlands, E. N. (1957). The oesophagogastric sphincter in hiatus hernia. *Lancet*, **2**, 1138-1142.
- ¹⁹Kramer, P., Harris, L. D., and Donaldson, R. M., Jr., (1967). Transition from symptomatic diffuse spasm to cardiospasm. *Gut*, **8**, 115-119.
- ²⁰Creamer, B., Olsen, A. M., and Code, C. F. (1957). The esophageal sphincters in achalasia of the cardia (cardiospasm). *Gastroenterology*, **33**, 293-301.
- ²¹—, Donoghue, F. E., and Code, C. F. (1958). Pattern of esophageal motility in diffuse spasm. *Ibid.*, **34**, 782-796.
- ²²Kramer, P., Flesher, B., McNally, E., and Harris, L. D. (1967). Oesophageal sensitivity to mecholyl in symptomatic diffuse spasm. *Gut*, **8**, 120-127.
- ²³Olsen, A. M., and Schlegel, J. F. (1965). Motility disturbances caused by esophagitis. *J. thorac. cardiovasc. Surg.*, **50**, 607-612.
- ²⁴Atkinson, M., Edwards, D. A. W., Honour, A. J., and Rowlands, E. N. (1957). Comparison of cardiac and pyloric sphincters: a manometric study. *Lancet*, **2**, 918-922.
- ²⁵Creamer, B., Andersen, H. A., and Code, C. F. (1956). Esophageal motility in patients with scleroderma and related diseases. *Gastroenterologia (Basel)*, **86**, 763-775.
- ²⁶Fyke, F. E., Jr., and Code, C. F. (1955). Resting and deglutition pressures in the pharyngo-oesophageal region. *Gastroenterology*, **29**, 24-34.
- ²⁷Siegel, C. I., Hendrix, T. R., and Harvey, J. C. (1966). The swallowing disorder in myotonia dystrophica. *Ibid.*, **50**, 541-550.
- ²⁸Arfwidsson, S. (1964). Pathogenesis of multiple diverticula of the sigmoid colon in diverticular disease. *Acta chir. scand.*, suppl. **342**.
- ²⁹Painter, N. S., and Truelove, S. C. (1964). The intraluminal pressure patterns in diverticulosis of the colon. Parts I and II. *Gut*, **5**, 201-213.
- ³⁰Parks, T. G. (1966). An enquiry into the pathogenesis of diverticular disease of the colon. M.S. Thesis, Queen's University, Belfast.
- ³¹Chaudhary, N. A., and Truelove, S. C. (1961). Human colonic motility: a comparative study of normal subjects, patients with ulcerative colitis, and patients with the irritable colon syndrome. *Gastroenterology*, **40**, 1-17.
- ³²Wangel, A. G., and Deller, D. J. (1965). Intestinal motility in man. III. Mechanisms of constipation and diarrhea with particular reference to the irritable colon syndrome. *Ibid.*, **48**, 69-84.
- ³³Painter, N. S., and Truelove, S. C. (1964). The intraluminal pressure patterns in diverticulosis of the colon. Parts III and IV. *Gut*, **5**, 365-373.
- ³⁴Connell, A. M., Jones, F. A., and Rowlands, E. N. (1965). Motility of the pelvic colon. IV. Abdominal pain associated with colonic hypermotility after meals. *Ibid.*, **6**, 105-112.
- ³⁵— (1961). Colonic motility in megacolon. *Proc. roy. Soc. Med.*, **54**, 1040-1043.
- ³⁶Davidson, M., Slesinger, M. H., Steinberg, H., and Almy, T. P. (1955). Studies of distal colonic motility in children. III. The pathologic physiology of congenital megacolon (Hirschsprung's disease). *Gastroenterology*, **29**, 803-824.