Diverticular disease of the colon: A 1968 view

Diverticulitis gained general attention at the turn of the last century and the first decade of the present one with the recognition of its mimicry of carcinoma of the sigmoid colon. The development of the barium enema made the demonstration of diverticula a routine matter, and, following Spriggs and Markes's distinction into the prediverticular state, diverticulosis, and diverticulitis, little then was added to the common teaching that the disease could be described simply in terms of diverticula and inflammation. A small minority had always held that there was some unusual spasm of the muscle in the affected area. Keith in 1910, giving a pathological demonstration on a handful of operative specimens, put the essentials of the problem very clearly and much of today's new writing is little more than illustrated recapitulations of his ideas. Edwards and some American radiologists were among the few who emphasized that there was something about the muscle itself that needed an explanation.

In the years 1963 and 1964 a series of papers on the pathology, radiopathological correlations, and studies of intraluminal pressures in the sigmoid colon combined to accentuate the evidence for a functional abnormality of sigmoid muscle, constantly seen in the chronic form of the disease and uncommonly the sole evidence of the disorder, present without a single sac or any sign of inflammation. Almy gives a useful summary of this stage. Today the interest is more concerned with what kind of abnormality is present, but facts are distressingly few and the simplest speculation can correlate all the known evidence. The need now is to take apart the clinical knowledge of the disease and to refashion it in the shape of questions that will interest and can be solved by physiologists. The present account is one such attempt, and its value will depend not on its ultimate correctness but on whether it solves the difficult problem of asking the right questions. Not that this should deter the interested worker. Basic understanding is so poor—there is, for example, no easily located article which records whether rigor mortis occurs in smooth muscle—that even the answer to the wrong questions is likely to yield useful information about colonic function.

The surgeon who opens the abdomen of the patient with chronic diverticulitis sees the colon in its resting state and finds the affected segment thickened and firm. Usually there is extra fat around the bowel, the mesentery thick and tense. Often he will palpate scybala in the diverticula and may see or feel signs of inflammation. The resected specimen is dropped into a pot of formol-saline for the pathologists who will between them record that the inflammation, if they do locate it, is too small to be held mechanically responsible for the muscular thickening. Histologically the muscle is not obviously abnormal, but there is a lot of it. The mucosa in the lumen is in excess and thrown up into redundant folds, and the ganglia of Auerbach’s plexus appear more numerous. The serosa is thickened and the blood vessels increased in number. Authors use the words ‘hypertrophy’ or ‘hyperplasia’ to characterize some of these observations, but in essence
the finding is that in the diseased area greater amounts than normal of every component of the wall are seen in each unit length of bowel, a series of observations which is simply explained on the single idea that the affected bowel is shortened. The belief that the thickness of muscle is solely due to hypertrophy is a tenable hypothesis, provided five other pathological processes are invoked to explain the increase in nerve tissue, the excess of mucosa in the lumen, the extra fat around the bowel and the thick mesentery, the increase in the thickness of the serosa, and the reduplication of blood vessels. Unless a new unitary concept can be found, Occam's law with its advice to economize on hypotheses, can hardly do less than urge as a working hypothesis that what the surgeon sees in the colon in its resting state is that the affected segment is shortened. No one has yet shown that this qualitative explanation can be complemented by a quantitative assessment, that contracted colon can have this degree of thickness of wall and the observed increase in the number of ganglion cells per centimetre. Nor does this idea rule out hypertrophy as an additional factor: indeed Arfwidsson\(^9\) has produced the only paper attempting to substantiate the use of this term in its strict meaning. In diverticular disease work hypertrophy seems to be implied, for which the intraluminal pressure measurements provide a cause in their record of extra activity. There is no suggestion that we are dealing with a disease in any way resembling obstructive cardiomyopathy\(^14\) with an obscure type of hypertrophy. Proof of work hypertrophy in diverticular disease merely returns one to the original question of what kind of muscular dysfunction is responsible for the increased work.

Radiologists\(^7\),\(^8\),\(^9\), who record graphically the position of the mucous membrane under distension at an unknown pressure for an unmeasured time, investigate a different aspect of colonic behaviour. They recognize a number of patterns reliably indicating the disease, of which the saw tooth sign is the most widely known, and correlating these with distended operative or postmortem specimens show the deformity to be an infolding of the side walls of the gut in very much the same fashion as normal haustration. They argue from simple inspection\(^4\),\(^5\) that this means the colon cannot elongate and allow the folds to flatten out as they would do in the normal. They find the cardinal abnormality is failure of the diseased segment to elongate fully, a feature independent of diverticula and inflammation. The importance of this is that the disease is diagnosed by the recognition of an abnormal pattern in distension, and in the absence of sacs this is the only way open to the pathologists\(^8\), who have as yet no histological criteria for the muscle disorder. This pattern, though not yet with the status of a numerical formulation, must use in its description a large number of bits of information and is as different from the hypertrophied colons of Chagas’ disease\(^15\) and chronic obstruction as is the style of Henry Moore from that of Michael-angelo. Its mimicry\(^16\), which needs confirmation and reassessment from other observers, by a mechanical trick of restraining the elongation of the taeniae while distending the circular muscle together with its destruction by dissecting off the taeniae\(^8\), is powerful support for the belief that in this disease the colon cannot elongate fully. It can be shown\(^17\) in operative specimens that the muscle in the affected area is still able to contract and relax, and this, with the surgeon’s evidence of a shortened resting state, the radiologist’s belief that it will not elongate fully, and the pathologist’s inability to explain these events in histological terms, makes ‘contracture’,
the conveniently vague word of Gasser,¹⁸ the best term for describing the muscle disorder. If this working hypothesis for 1968, drawn from the correlation of day-to-day clinical observations and resting on Occam's law, has a modicum of truth in it then questions for the physiologists flow out in dozens and it merely becomes a matter of selecting the right ones to investigate.

What is the nature of the initiating lesion? The patterns characteristic of the disease are seen as transient findings during a normal barium enema, that is, as a first guess the dysfunction is likely to be an exaggeration of a fairly simple normal event and not a random or completely unusual contraction as is corkscrew oesophagus. Should this be so, the problem immediately arises of distinguishing normal tone from this abnormal contraction in, for example, the action of smooth muscle relaxants. Because of the haustral structure of the human colon the radiologist finds it much easier to recognize the pattern indicating shortening than he does that of a minor contraction of circular muscle. This gives a bias difficult to estimate. Should the perpetuating mechanism of myostatic contracture lie in the connective tissue, it is possible that it forms more quickly and certainly in the taeniae, which are known to contain greater amounts of collagen and elastic than does the circular layer.¹⁸ At the moment there is no certain proof that the circular muscle is involved, so that investigation of the mechanism that allows a differential contraction of the two layers may give a clue to the site of the basic lesion. The rectum is believed to escape the disease even when the sigmoid is severely affected. Embryologically the muscle layers are said to arise from the same sheet of cells and one would expect any disease arising in the metabolism of the muscle cell itself occasionally to affect the rectum. Is this escape of the rectum a point in favour of a lesion in the controlling mechanisms outside the cell?

The contracture is found many hours after death of the individual and yet can be shown radiographically to be variable during life. Is there a single known contracture which has these two characteristics or must one look for two mechanisms, one a spasm which initiates the contracture and a second which causes it to persist? The latter view is summarized in the term 'myostatic contracture'¹⁷, and elucidation of the mechanism of perpetuation (connective tissue versus muscle proteins, for example) will be a help when the initiating mechanism is controllable by drugs. An identical postulate has been made by Short²⁰ for arterial smooth muscle in hypertension and by Fleischner et al.¹⁸ for the colon in the phrase 'spasm which persists becomes permanent'. One suspects the optimists will be attracted to myostatic contracture since it makes the apparently solid permanent look of diverticular colon a potentially reversible condition. In life, in radiographs for example, shortening may come in two parts at the same instant, a spasm which can be reduced by smooth muscle relaxants and a myostatic part not easily influenced by drugs.

Should it be accepted that the present radiological methods of assessing the amount of colon involved have some accuracy, then it is possible to state that on occasions the disease may be confined to a short length of bowel and remain so for years. What mechanisms limit the spread of contraction in the normal colon? The physiology of the whole circumference in 10 cm lengths rather than a few millimetre lengths has already been partly investigated.²¹ Work on smooth muscle relaxants²² with intraluminal pressure...
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recordings and cineradiology has shown that in some cases the spasm can be overcome, and this is manifestly a technique of promise. In the field of clinical medicine there are now large numbers of patients on potent drugs affecting the autonomic system. What happens to the diverticular disease of a hypertensive patient on methyldopa? Sigmoid myotomy, usually described as a simple operation in comparison with resection, is in physiological terms a slashing attack on the pathways of conduction around the bowel wall. An electro-physiological investigation may succeed in producing a rationale for the operation. Electron microscopy of muscle to determine the state of the cell organization, and of nerve cells to determine minor damage, is an obvious need.

Accepting Medawar's definition—if politics is the art of the possible then research is surely the art of the soluble—and viewing from the bedside, and given the talents and skills now widespread around the world, the initial approach to many of these problems does not look too difficult, and might be expected to reveal the site of the primary lesion. Rational treatment cannot be very far away.

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REFERENCES