

Diverticular Disease of the Colon— The Possible Role of “Roughage” in Both Food and Life

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Diverticular disease of the colon has been recognized for a long time, but understanding of its pathogenesis is relatively recent. Four phases may be defined in the accumulation of knowledge of this interesting malady: the clinical association of diverticular disease of the colon with the irritable colon syndrome; the demonstration of abnormal thickening of the colonic smooth muscle without inflammation in resected specimens of large bowel; clinical demonstration of abnormally increased intraluminal pressures in segments of colon containing diverticula; and the epidemiologic observations of predominance of diverticulosis among Western countries.

Clinically there is a close association between the symptoms of the irritable colon syndrome and the presence of diverticulosis of the colon (1, 2). Radiographically, the changes in the sigmoid colon of such patients include evidence of spasm with areas of narrowing and an irregular contour of the bowel wall, alterations quite similar and often indistinguishable from those of early diverticulosis of the sigmoid colon. Early in the formation of diverticula there is thickening of both the circular and longitudinal muscle layers of the sigmoid colon due to spasm and shortening of the muscle fibers (3).

Intrasigmoidal pressures are higher than normal in segments containing diverticula at rest, after the ingestion of food or drugs such as prostigmine and morphine, and during emotionally-charged discussions (4). Painter has emphasized that diverticulosis of the colon is quite rare in African and Asian countries where diets contain a high residue from unrefined carbohydrates and cellulose (5). As the rural areas become more “Westernized” and as more refined sugars and grains are consumed, the in-

cidence of diverticulosis rises. These observations have renewed interest in some earlier experiments of Carlson and Hoelzel who produced diverticula of the rat colon by feeding a low residue diet (6). These changes were not seen in rats who received diets containing increased bulk. The addition of bulk to the diet apparently reversed the diverticular process.

These observations in time led to therapeutic trials by Painter who, by treating patients with diverticular disease of the colon with high residue diets containing fruits, vegetables, and unprocessed bran, achieved symptomatic improvement in many (7).

Almost predictably, we now have the observations of Manousos and his colleagues (8) that diverticulosis is more common among the prosperous residents in urban sections of Greece than in the populations of rural areas of Greece. Differences in quantity of residue consumed in the diets between the urban and rural populations were not significant.

These observations, in their aggregate, would thus appear to document as important mechanisms in the pathogenesis of diverticular disease of the colon a) thickening of the circular and longitudinal muscle layers of the sigmoid colon due to spasm; resulting in b) shortening and narrowing of the bowel lumen; leading to c) segments of colon with increased intraluminal pressure; which cause d) the herniation of mucosa through the bowel wall where it is weakened by penetrating blood vessels.

The question now arises as to how these findings fit each other. Can they be utilized to provide a more complete account of the development of colonic diverticula? Differences in life style other than dietary exist between urban

and rural populations. Such factors as crowded living and working conditions, travel to and from work, noise and air pollution, competition, and emotional stress characterize an urbanized Western society and probably contribute to an excessive psychophysiologic stimulation of the sigmoid colon, resulting in spasm and increased intraluminal pressure. A high residue diet may provide the necessary bulk in the sigmoid colon to prevent the formation of high pressure segments—perhaps like a “safety valve.” In the absence of sufficient bulk in the food, the tension-induced high pressure segments within the colon would persist. In other words, the amount of “roughage” in life may be as important as the amount of roughage in the diet. Long-term controlled clinical studies obviously are necessary to determine if dietary manipulation or the administration of antispasmodics can influence the development or the prognosis of diverticulosis. In any event, we now have a useful pathogenetic concept around which to plan useful clinical and therapeutic studies towards the clarification of this intriguing disorder of “civilization.”

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