Lifestyle and Disease

Diverticular disease of the colon

The first of the Western diseases shown to be due to a deficiency of dietary fibre

N. S. PAINTER

Summary

Diverticular disease of the colon is a new disease that appeared at the beginning of this century. It is now the commonest disease of the colon in the Western world, being found in 1 in 3 people of over 60 years of age.

The pathogenesis of the disease involves excessive segmentation, but this does not explain its aetiology. The historical appearance of the disease on the clinical scene and its geographical distribution suggest that it is due to the removal of fibre from carbohydrates. The author treated 70 patients with symptomatic diverticular disease with a high-fibre diet. The results of this and the effects of bran are discussed.

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Diverticular disease of the colon was the first disease to be shown to be caused by a deficiency of fibre in the diet. The demonstration that over 90% of the symptoms of uncomplicated diverticular disease can be relieved by fibre replacement led to the present interest in dietary fibre. This paper discusses the aetiology and pathogenesis of the disease and gives details of how bran was first used in its treatment.

Definitions

Colonic diverticula are acquired and so are environmental in origin. *Diverticulosis* used to imply that diverticula were present but were causing no symptoms. Patients with diverticula giving rise to symptoms were said to have *diverticulitis*. Now it is known that in most patients with diverticula abdominal discomfort, pain and other symptoms are caused by the colon and probably the rest of the gut behaving abnormally as it struggles with small, stiff stools. These result from a fibre-deficient diet, and excessive segmentation is required to move them. This leads to intermittent functional obstruction which causes discomfort and at times very severe colicky pain. This should be called painful diverticular disease to distinguish it from true infective diverticulitis.[1-3]

Manor House Hospital, London, England
N. S. PAINTER, M.S., F.R.C.S., F.A.C.S., D.OBST.R.C.O.G., *Senior Surgeon*

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Pathogenesis

The anatomy of colonic diverticula is shown in Fig. 1. The circular muscle forms a complete tube, but the longitudinal muscle is gathered into three taeniae. Between the taeniae the circular muscle is weakened by tunnels formed by the segmental blood vessels. Diverticula usually herniate through these tunnels, and the vessels therefore bear the same relationship to the diverticula that the spermatic cord does to an inguinal hernia.[4]

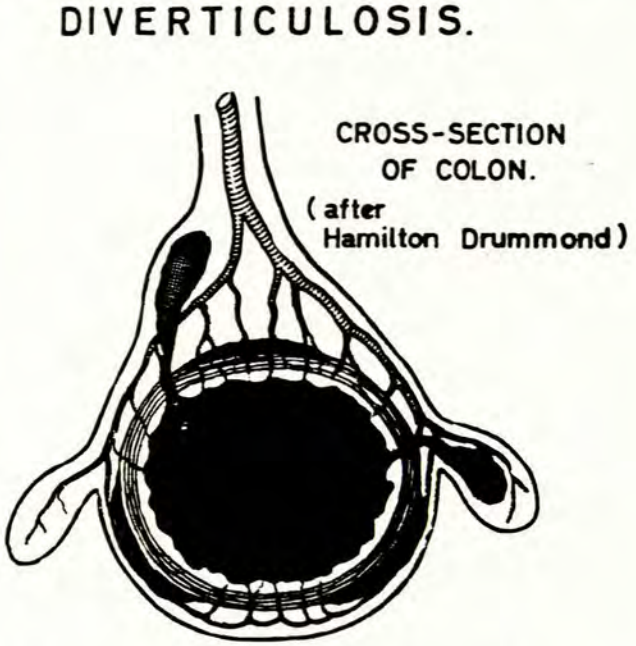

Fig. 1. Cross-section of colon showing colonic diverticula.

Diverticula are hernias and must be caused by weakness of the colonic wall, high intracolonic pressure or a combination of these two factors. Many theories have been put forward only to be dismissed.[3,5,6] The structure of the colon cannot be responsible for the disease as it is common to all patients and all races, whether or not they are affected by diverticulosis. No congenital weakness in the colon has been demonstrated, and both corpulent and thin subjects are affected. The incidence of diverticulosis increases with age, but nowadays it is also found in patients under 30 years of age. Hence many theories were put forward and debated until 1961, when the pressures in the human colon were first measured, both in healthy subjects and in those with diverticulosis. The effects of morphine, pethidine, prostigmine and propantheline bromide (Probanthine) on these pressures were studied, and I used simultaneous cine radiography to relate the configuration of the colon in a living subject to the pressures within it.[5-8] The colon's ability to

produce and to localize high pressures was shown to involve segmentation (Fig. 2). The top diagram in Fig. 2 shows a longitudinal section of colon containing three pressure-recording tubes. If the colonic wall contracts its contents are free to move through the open lumen and no significant pressure change will occur.

High intracolonic pressures may follow natural stimuli such as eating and emotion or the administration of drugs. In the middle diagram in Fig. 2 contraction rings on each side of the centre segment have narrowed the lumen on each side of it. If the segment contracts, its contents must move through a narrower lumen. The resistance thus offered results in a wave of pressure localized to this segment. The segmented colon acts as a series of 'little bladders', the outflow of which is obstructed at both ends and each of which harbour a different pressure. The bottom diagram in Fig. 2 shows how the centre segment has become isolated. Any contraction of this segment on its almost incompressible content will generate very high pressure that remains localized to this segment. Segmentation is essential to the transport and halting of the colon's contents. If the colon has to overwork it becomes thickened or 'trabeculated', the pre-diverticulosis state which can be seen on radiographs. The thickened colonic muscle becomes uneven in thickness and finally the mucosa herniates through the weakened parts of its wall. The sigmoid colon has to propel the most viscous faeces and hence is the commonest site of diverticular disease.[1]

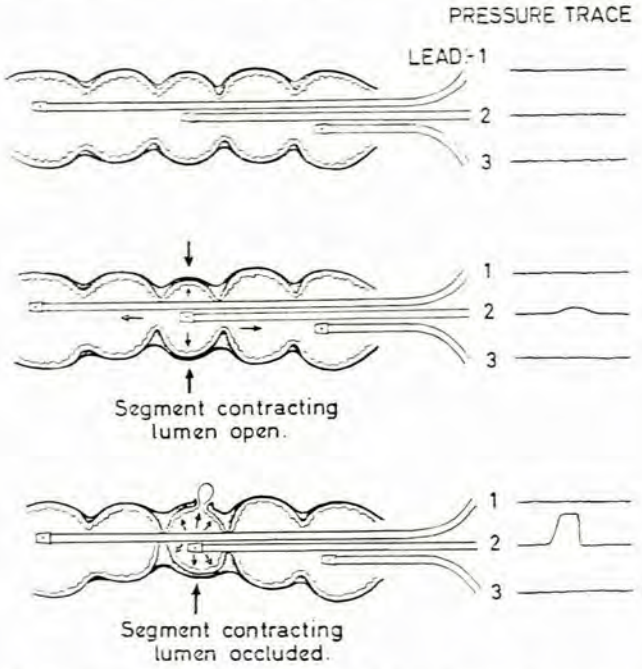

Fig. 2. Segmentation and the generation of high localized intracolonic pressures.

The demonstration of the role of segmentation explained the pathogenesis of diverticula, but the aetiology of the disease is still unknown. However, cine radiography showed that a colon with a narrow lumen initially segmented more efficiently than did a colon with a wide lumen. A low-residue diet was known to result in a narrow colon both in man and in rats.[9] This led me[5,6] to question the wisdom of prescribing a low-residue diet, until then the standard treatment for the disease.

Aetiology

Historical emergence as a new Western disease

It is difficult to 'prove' what causes a disease which takes half a lifetime to develop. However, diverticulitis appeared suddenly at the turn of the century, but only in certain populations. A study of its history and its geographical distribution suggests that it is caused by our modern over-refined diet. The evidence for this can be summarized as follows:

Colonic diverticula were described in the last century and were attributed to constipation.[10-12] They remained a pathological curiosity and were still almost unknown to clinicians until 1900. However, in the next decade diverticulitis appeared as a surgical problem so that by 1917 all its complications could be described. In 1920, Sir John Bland-Sutton called it a 'newly discovered bane of elders'.[13-16] This leaves little doubt that something had suddenly adversely affected the colon's environment.

As early as 1920 diverticula had become common in the UK, and by 1930 they were found in 5% of people aged over 40 years. Today 45% of Australian colons bear diverticula,[17] and by the age of 60 one-third of Westerners have diverticulosis. This rises to two-thirds by the age of 80. Diverticular disease, practically unknown in 1900, thus became the commonest colonic disease in only 70 years, the traditional lifespan of man.[2,3,18]

Geographical distribution

The dramatic appearance of diverticulosis and its rapid increase in incidence occurred only in the industrialized countries. It is still almost unknown in rural Africa, Asia and South America.

At Baragwanath Hospital, Johannesburg, which serves urbanized Blacks, no diverticula were found in the course of 2 367 autopsies and 600 barium enema examinations between 1954 and 1956.[19] In the last few years 16 cases have been discovered (I. Segal — personal communication, 1976). In Uganda only 2 diverticula were found in 4 000 autopsies in 15 years. In 1971 questionnaires sent to 37 doctors in rural hospitals revealed that together they had seen only 2 cases of diverticulosis.[3,18]

The disease is still rare in Asians in Singapore[20] and in Korea.[21] In Mexico it is found only in the well-to-do and not in the poor (personal observation). The position is similar in India.

Diverticulosis is rare to this day in peoples who have adhered to their traditional eating habits and who do not eat Western refined foodstuffs.

Changes in diet and the appearance of diverticular disease.

Diverticula take about 40 years to develop and were common in England in 1920. If this was due to a change in diet it would be expected that the British diet altered between 1870 and 1880. *In fact, it changed dramatically.*

Until 1870 'white flour' was available, but it was made traditionally by stone-grinding and sifting off some bran from wholemeal flour. Between 1870 and 1880 roller milling was introduced. Efficient metal rollers crushed the grain and made available the very white flour that we know today. Water-mills and windmills disappeared from the English countryside. Improvements in rail and sea transport, aided by the advent of refrigeration and canning, brought alternative cheap foods to Europe from all over the world. The pound increased in value, meat imports doubled, and even the poor could afford white flour and refined sugar. As a result the sales of wholemeal bread, formerly the staple source of energy, fell and continued to fall until about 10 years ago. The average *per capita* consumption of refined sugar, which contains no fibre, has risen from 60 lb. (27 kg) a year in 1960 to 150 lb. (67,5 kg) today. More fat and protein are eaten, as is more of some fruits and vegetables which are now available out of season. Cereal fibre consumption has

therefore dropped, firstly because less bread and flour are eaten, and secondly because modern white and most brown breads contain much less fibre.[22,23]

In the last century the biggest change in our diet has been a drop in cereal fibre consumption to one-fifth and possibly one-tenth of that previously eaten, and this change occurred very suddenly. It seems that the colon could not adapt to this change, and within 40 years diverticular disease had appeared on the clinical scene.

The American experience was similar. In 1937 American Blacks were less prone to the disease than Whites,[24] but now Black Americans suffer from the disease as often as do their White compatriots.[23] Similarly, the disease appeared in Tokyo only after World War II, while it is as common in Japanese born and bred in Hawaii as it is in White Americans.[3] *Diverticulosis is therefore not racial in origin,* but appears in peoples who change their eating habits and consume less dietary fibre. If this is so, it should appear in developing countries in about 30 years if their populations adopt Western eating habits. It is likely to be preceded by the appearance of appendicitis, as it was in America and Europe at the end of the last century. Already the incidence of appendicitis is increasing in urban Blacks in South Africa.

Transit times, stool weights and diverticulosis

The time between eating food and excreting its residue can be measured, together with the weight and consistency of the stools. These vary with the amount of fibre in the diet (Table I). The English fibre-deficient diet produces about 100 g of stiff stools daily, with an average transit time of 4 days. Ugandan villagers pass over 400 g daily, without straining, within 35 hours. This is not due to race, as English vegetarians eating more fibre pass about 200 g in 2 days, which is similar to the amount passed by South African boarding school children on a partly European diet. Likewise, patients at the Manor House Hospital, London, passed 170 g of stool daily in 41 hours once they had begun to take bran. The amount of fibre in the diet therefore affects the weight of the stools and the speed at which they traverse the intestine.[25,26]

A high-fibre diet leads to large, soft stools which require little pressure to propel them. Furthermore, colons which cope with large faecal volumes have a wide diameter and segment less efficiently than do the narrow colons of those eating low-fibre diets, which means that people on a high-fibre diet are less prone to develop diverticulosis.[5,6] In short, a high-fibre diet results in swiftly passed, soft stools which subject the sigmoid colon to little strain and do not favour the development of diverticula. The low-residue diet which was prescribed for nearly 50 years in the treatment of diverticulosis has been abandoned, as it is the cause of the condition! The epidemiological investigation of diverticular disease, appendicitis and colonic neoplasms shows that where a high-fibre diet is eaten these diseases are also rare or almost unknown.

Bran and a high-fibre diet in treatment

The low-residue diet became the mainstay of medical treatment for diverticular disease because it was thought that 'roughage' irritated the gut and that fragments of bone and stalk might perforate diverticula. No evidence of its efficacy was ever produced.

The recognition that the narrow colon which resulted from a low-residue diet could produce high pressures more easily threw doubt on the wisdom of the low-residue diet.[5,6] The observation that West African Blacks ate a bulky diet, had colons with a wide lumen and did not get diverticulosis prompted me to prescribe a high-fibre diet for diverticulosis. Initial results were encouraging, and so between 1966 and 1971 a high-fibre diet, including miller's bran, was given to 70 patients. All had symptomatic diverticular disease diagnosed by barium enema examination and had been investigated to exclude the presence of carcinoma or of any fibrous narrowing of the colon.[27]

Method of giving bran

The nature of the disease was described to each patient with

TABLE I. TRANSIT TIMES, STOOL WEIGHTS AND DIET*

Subjects	Country	Race	Type of diet	Mean transit time (h)	Mean weight of stools passed (g)	Comments
Naval ratings and wives	**UK**	**White**	**Refined**	**83,4**	**104**	**Shore-based personnel**
Teenage boarding school pupils	**UK**	**White**	**Refined**	**76,1**	**110**	**Institutional diet together with cakes, sweets, etc. from school shop**
Students	**SA**	**White**	**Refined**	**48,0**	**173**	**Ate more fruit than is usual in UK**
Nurses	**SA**	**Indian**	**Mixed**	**44,0**	**155**	**Less refined diet than that of Western world**
Urban schoolchildren	**SA**	**Black**	**Mixed**	**45,2**	**165**	**Partly Westernized diet**
Manor House Hospital patients	**UK**	**White**	**Mixed**	**41,0**	**175**	**UK diet plus wholemeal bread and added bran**
Senior boarding school pupils	**Uganda**	**Black**	**Mixed**	**47,0**	**185**	**Traditional Ugandan diet plus refined sugar, white bread, jam, butter**
Vegetarians	**UK**	**White**	**Mixed**	**42,4**	**225**	**Note similarity of values to those of Black groups**
Rural schoolchildren	**SA**	**Black**	**Unrefined**	**33,5**	**275**	
Rural villages	**Uganda**	**Black**	**Unrefined**	**35,7**	**470**	**Villagers not yet supplementing their diet with processed foods of Western type**

*Modified from Burkitt *et al.*[26] note the inverse relationship between stool weights and transit time.

the aid of a simple diagram, and the effect of fibre deficiency was explained. Each patient was asked to eat a 'high-fibre/low-sugar' diet and was given a packet of bran (bran is difficult to swallow dry and should be added to cereals, porridge or soup or washed down with fluid). Patients were asked to eat Kellogg's All-Bran and other bran-containing cereals, unrefined porridge or muesli. They were advised to use 100% wholemeal bread and plenty of fruit and vegetables and to reduce their intake of refined sugar, both brown and white. Women were reminded that refined sugar only yields energy or fat!

Initially, 2 tablespoonfuls of bran were taken 3 times a day with each meal, and after 2 weeks the amount was increased slowly *until the bowels opened easily twice a day without straining.* If patients did not strain, it was obvious that their colon did not have to overwork. Each patient was seen monthly until he or she had reached the amount of bran required. *This is essential*; some patients do not read instructions and do not increase their intake of bran. The patients were told that there is no 'dose' of bran and that once they had found the right amount they needed by trial and error, they should take this amount for life.

Results of the bran trial

Of the 70 patients, 3 became free of symptoms on All-Bran and wholemeal bread. One had taken senna for 20 years and the ganglia of his resected sigmoid had been destroyed. This was the only patient who came to surgery. Four others did not like bran and were given the bulk-former Normacol. This left 62 patients who took bran regularly. On average 6-8 teaspoonfuls of bran daily (about 14 g) abolished straining at stool, but some patients needed several tablespoonfuls to obtain this result. Bran caused flatulence in 39 of the 62 patients during the first few weeks — *patients must be warned of this and told not to stop taking the bran on this account!* It is a temporary symptom which disappears in about a month once the bran has taken effect.

The bowel habits of the 62 patients before and after bran had been introduced is shown in Fig. 3. Bran relieved constipation, so that soft stools were passed at least once a day. Those who voided hard 'sheep droppings' several times a day, often with episodes of passing mucus, also responded to the high-fibre diet. After bran had been introduced all the patients voided soft stools without straining, whereas before only 8 had not strained. Many said that they had now begun to defaecate completely; formerly they had always been conscious of something in their rectum.

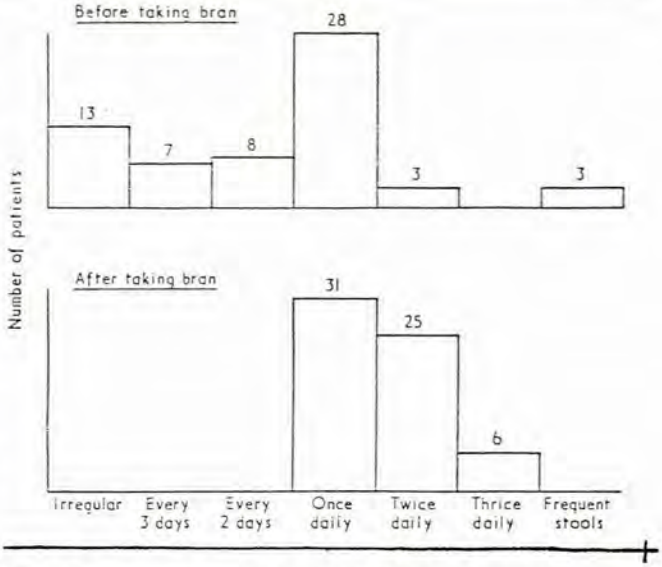

Fig. 3. Bowel habits of patients before and after taking bran.

Relief of symptoms by the bran diet

Patients with diverticular disease may present with dyspeptic complaints or with large-bowel symptoms. Excessive segmentation leading to intermittent functional obstruction may cause pain severe enough to be mistaken for left renal colic. Formerly this was attributed to 'diverticulitis' and treated by resection. Bran relieves even this pain, and so lessens the need for surgical intervention.

The main presenting symptoms of the 70 patients are shown in Table II (some had more than one main symptom, so the symptoms total 171). Fourteen symptoms (18,2%) were experienced by the 8 patients who did not take bran. Only 6 (3,8%) of the remaining 157 symptoms were not relieved or completely abolished by bran. The 'bran diet' therefore relieved or abolished 88,6% of symptoms, and this did not include the 3 patients whose symptoms abated with All-Bran and wholemeal bread.

Before beginning to take bran 49 of the 70 patients took laxatives. After they had begun to take bran only 7 took

TABLE II. PRESENTING SYMPTOMS IN 70 PATIENTS

	Before bran	Bran not tolerated	Symptoms after bran		
			Not relieved	Relieved	Abolished
Dyspeptic					
Nausea	11	1	1	2	7
Heartburn	2	—	—	2	—
Flatulence	2	—	1	1	—
Distension	36	4	2	14	16
Wind	13	—	—	4	9
Painful diverticular disease					
RIF pain or ache	7	—	0	5	2
LIF pain or ache	22	1	1	7	13
Lower or general abdominal pain	28	3	—	11	14
Severe colic	12	1	—	4	7
Bowel symptoms					
Tender rectum	4	—	—	1	3
Incomplete emptying of rectum	6	—	—	3	3
Constipation	28	4	1	8	15
Total symptoms	171	14	6	62	89

RIF = right iliac fossa; LIF = left iliac fossa.

occasional laxatives. The few who did not take bran were given Normacol, as it very rarely clumps and causes obstruction as do some bulk-formers. Bran therefore saves money and avoids the prolonged use of laxatives, a habit which is not without risk.

Two of the 62 patients had suffered an attack of 'acute diverticulitis' before taking bran. One woman had had 3 attacks of 'left renal colic'. Pyelography was negative and a barium enema examination showed diverticulosis of the sigmoid colon. She became symptom-free on bran and has remained so since 1968.

Before beginning to take bran 12 of the 70 patients, including this woman, had had recurrent attacks of the very severe colic which would formerly have been treated by sigmoidectomy. Eleven of those became symptom-free on bran and 1 obtained relief on Normacol. None of the 12 has come to operation.

'Roughage' has been said to irritate the gut and aggravate conditions such as peptic ulcers and hiatus hernia. This is not true. Man has eaten cereal fibre since the dawn of time, and bran becomes 'softage' when wet. Twenty-six of the patients had poor appetites, which improved in all cases after the introduction of bran. Patients with duodenal ulcers and hiatus hernias took bran without any ill-effects. Bran does no harm unless the patient is gluten-sensitive.

Some other effects of bran

Once this trial[27] had shown that bran was cheap, effective and safe, others looked at some of its effects. Bran relaxes the colon and reduces the intracolonic pressures better than simple bulk-formers.[28,29] Bran tablets each containing 2 g of bran reduced intracolonic pressure, the transit time and also altered the electrical activity of the diseased colon towards the normal.[30] Similar results have been reported by Brodribb[31] and Brodribb and Humphreys.[32]

After sigmoid myotomy or resection intracolonic pressures fall, but if the patient continues to eat the same diet they rise again within 3 years. In contrast, if patients change to a high-fibre diet after the operation, these pressures show no sign of rising after 5 years.[33] This shows that while operative intervention can deal with the complications of the disease it does not remove the cause of the condition. Only a high-fibre diet protects against a recurrence of symptoms.

Hyland and Taylor[34] followed up 100 patients who had suffered from symptoms or complications due to diverticular disease for 5-7 years; 25 had required an operation. On a high-fibre diet 91% remained free of symptoms. This was in marked contrast to previous reports in which no more than 60% of both medically and surgically treated patients remained symptom-free.[3] Before the adoption of the high-fibre diet, 17 planned resections for so-called 'diverticulitis' were performed annually at the Royal Berkshire Hospital, Reading, UK. Now only 1 such operation is performed in a year (C. Latto — personal communication, 1978). Bran therefore reduces the need for surgical intervention in diverticular disease.

Discussion

The relief of symptoms by bran shows that they are not due to the diverticula, which remain *in situ*, but result from the colonic muscle struggling with contents of abnormal consistency. If a low-residue diet can lead to the colon literally 'rupturing' itself, it is very unlikely that the colon (and usually only its sigmoid portion alone) is the only part of the gut to be adversely affected by this diet. The concept that fibre deficiency alters the behaviour of the proximal intestine as well as that of the colon would explain why bran alleviates the 'upper intestinal' symptoms associated with diverticulosis.

The irritable bowel syndrome is a vague, ill-defined condition. The name implies that the bowel itself is abnormal. It is not seen in rural communities but has appeared recently in urban South African Blacks who had adopted Western eating habits.[35] The disease is so common in the West that it is impossible, if only on evolutionary grounds, to believe that so many people could have congenitally abnormal intestines. Most patients improve on a high-fibre diet. The disease should be called the irritated bowel syndrome. Such a change of emphasis would stop us from blaming normal intestines for symptoms that are due to our over-refined diet, which contains many food additives.[27] Some of these have been shown to be toxic when fed to animals eating refined foods, but their adverse effects can be counteracted by fibre, including wheat bran.[36] Bran therefore has a detoxicating effect on substances which may play a part in causing some of the other intestinal diseases of civilization.

Conclusion

Diverticular disease of the colon is a deficiency disease caused by lack of fibre in the diet. The 'low-residue' diet formerly prescribed for its treatment is therefore contraindicated. A high-fibre diet will relieve the symptoms of uncomplicated diverticular disease and lessen the need for surgical intervention in painful diverticular disease.

A return to a diet containing the natural amount of fibre should prevent the appearance of the disease in succeeding generations. Such a change would dilute any noxious compounds in the colon's contents and speed their passage through the bowel. This might well decrease the incidence of colonic cancer.

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