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Modification in the xylose absorption test as an index of intestinal function

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COMMENT This paper confirms the fact that a small oral dose of *d* xylose (5 g.) gives good evidence of small bowel malabsorption when its excretion is followed in the urine. Much better localization of the intestinal defect can be gained by measuring the two-hourly and five-hourly urinary excretion after this dose. A normal five-hour excretion may be accompanied by a lowered two-hourly excretion, thus still indicating the presence of upper small bowel disease.

The fraction of an oral dose of *d* (+) xylose excreted in the urine in five hours has been widely used as a test of the absorptive capacity of the small intestine (Fourman, 1948; Fowler and Cooke, 1960; Joske and Curnow, 1962; Sheehy and Floch, 1964). In subjects with normal renal function the five-hour urine excretion of xylose after an oral dose depends on four main factors: the stomach emptying time, the rate and duration of absorption of the xylose from the intestinal lumen, and the rate of metabolism of the xylose once absorbed. The rate of absorption depends on the functional integrity of the intestinal mucosa; absorption will continue as long as xylose remains in the small intestine.

The procedure frequently adopted in the study of patients with malabsorption is that of Helmer and Fouts (1937), who gave 25 g. of xylose and measured the xylose in the urine in the following five hours. Unfortunately this large dose of xylose is nauseating and causes diarrhoea in a third of patients (Chanarin and Bennett, 1962) and even, in our experience, in some normal people.

Butterworth, Perez-Santiagio, Martinez de Jesus, and Santini (1959) and Santini, Sheehy, and Martinez de Jesus (1961) found that a 5 g. dose did not cause diarrhoea but still distinguished intestinal disease from normal subjects. Rinaldo and Gluckmann (1964), however, found that this smaller 5g. dose only revealed the more severely affected patients. We have found that with either the 5 or 25 g. dose the test is insensitive in that it fails to distinguish the borderline cases. We felt that variation in the dose of xylose given, or a more careful study of the excretion of xylose, especially in the first five hours, might increase the sensitivity of the test. We have, therefore, investigated xylose excretion after doses ranging from 0·5 to 25 g. and followed the pattern of excretion in the urine. A preliminary account of this work, with a description of a modified xylose excretion test has been published previously (Morgan and Sammons, 1964) and the limitations of this test emphasized (Sammons, 1965). Additional evidence supporting the modified test is now presented together with a more detailed discussion of the concepts involved.

METHODS

ESTIMATION OF XYLOSE The method of Roe and Rice (1948) was used. One ml. of the test solution was always retained and used as a standard.

TECHNIQUE All subjects were fasted overnight but fluid intake was not restricted and where necessary it was encouraged. This was essential in patients who were dehydrated and in children. A dose of *d* (+) xylose (B.D.H.) was given in 250 ml. distilled water at 9.0 a.m. Solid food, but not fluid, was withheld until a normal meal was given, usually at 2.0 p.m. Urine was collected in timed periods as described in the results. A request for a routine xylose tolerance test after a 5 g. dose now requires that the urine be collected in two consecutive periods of two and three hours' duration. The two-hour and five-hour excretion and the two : five-hour excretion ratio are reported.

NORMAL SUBJECTS These were members of the hospital staff and patients who had no evidence of gastrointestinal disease or impairment of renal function.

PATIENTS WITH ENTEROPATHY The results in 44 subjects

have been given to illustrate various aspects of the xylose tolerance test. All had a serum urea level less than 50 mg. per 100 ml. and no proteinuria. Four had gluten-induced enteropathy affecting the upper small intestine with marked steatorrhoea; the creatinine clearance was more than 70 ml./min. in all of them. Four others had steatorrhoea but a completely normal absorption of xylose, and four had had a partial gastrectomy. Twenty-eight patients had a low two-hour and four had a low three-hour excretion. None of these had a disease which primarily affects the upper small intestine but some of them had other evidence suggestive of malabsorption.

TABLE I

TWO- AND FIVE-HOUR XYLOSE EXCRETION IN NORMAL SUBJECTS

Subject	*Dose (g.)*							
	0·5	*1·0*	*1·5*	*2·0*	*5·0*	*10·0*	*15·0*	*25·0*[1]
Two-hour Excretion (% dose)								
A	28	29	28	29	22	17	17	13
B	31	24	28	25	28	20	17	13
	27							
C	16	23	26	28	27	15	19	13
D	21	23	21	19	14	13	9	7
E	25	26	19	24	18	17	16	14
	26							
Average	25	25	25	25	22	16	16	12
Five-hour Excretion (% dose)								
A	39	33	41	44	33	24	30	27
B	53	42	39	39	45	36	26	26
	46							
C	30	38	40	41	40	24	26	24
D	38	34	33	33	28	30	18	22
E	37	39	32	31	31	30	31	30
Average	40	37	37	38	35	29	26	26

[1]All subjects found the 25 g. dose nauseating and three out of five had diarrhoea afterwards.

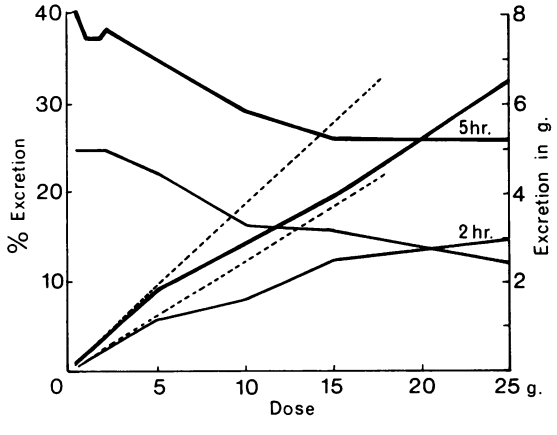

FIG. 1. *Pattern of excretion after varying doses in five normal subjects. Dotted lines indicate departures from linearity after 5 g. dose.*

RESULTS

XYLOSE EXCRETION AFTER DIFFERENT DOSES The pattern of excretion in five normal persons after doses varying from 0·5 to 25·0 g. is given in Table I and illustrated diagrammatically in Fig. 1 which also shows the departure from linearity for doses above 5 g. Table I also shows the consistency of the results in these persons.

EXCRETION OF XYLOSE AFTER A 5 G. DOSE Table II shows that after a 5 g. dose in 52 normal subjects the results for the five-hour excretion of xylose are identical with the results reported by others, the mean value being 35% with a standard deviation of 6·2 and a range of 23 to 48.

TABLE II

XYLOSE EXCRETION (% DOSE) IN NORMAL SUBJECTS USING A 5 G. DOSE

	No. of Tests	*Mean*	*S.D.*	*Minimum*	*Maximum*
Butterworth *et al.* (1959)	29	34	5	—	—
Santini *et al.* (1961)	125	36	6	24	48
Present series					
Five-hour	52	35	6·2	23	48
Two-hour	40	23	4·5	14	34
Two-hour : five-hour (%)	40	62	10·1	39	81

Table III shows the cumulative excretion of xylose over the 24 hours after a 5 g. dose in 29 tests on 19 normal persons and 10 tests on four patients with gluten-induced enteropathy. The five-hour excretion was similar in the two groups but the patients with gluten-induced enteropathy put out less in the first two hours.

TABLE III

CUMULATIVE XYLOSE EXCRETION OVER 24 HOURS AFTER A 5 G. DOSE

Time (hr.)	*No. of Tests*	*Percentage Dose* *Mean*	*S.D.*	*Min.*	*Max.*	*% 24-hour Excretion*
Normal Subjects						
2	29	22·9	±4·5	14·2	29·9	54
5	29	35·7	±7·1	22·8	47·9	84
8	29	39·0	±7·1	23·2	51·4	92
12	29	40·7	±7·3	24·5	52·2	96
24	29	42·5	±8·0	25·8	53·3	100
Patients with Gluten-induced Enteropathy						
2	10	6·4		4·1	8·4	25·0
5	10	19·0		13·3	26·5	75
8	10	23·1		16·9	32·6	92
12	10	24·4		17·9	34·5	96
24	10	25·3		18·2	35·6	100

The excretion of xylose in the first five hours after a dose was studied in more detail. Figure 2 shows the excretion of xylose in each of the first five hours

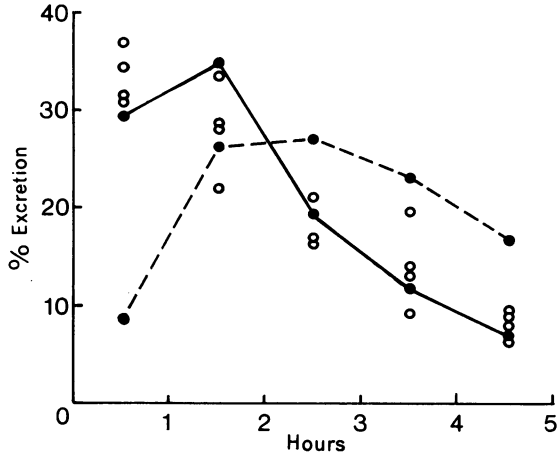

FIG. 2. *Hourly excretion of xylose expressed as percentage of total five-hour excretion after 5 g. dose.*
● ——● *Mean values in 16 normal subjects.*
● - - - - ● *Mean of 10 tests on four patients with gluten-induced enteropathy.*
(○) *Four other patients with partial gastrectomy.*

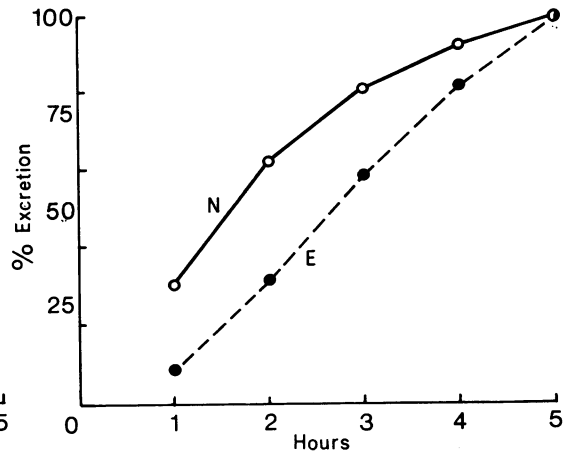

FIG. 3. *Cumulative excretion of xylose expressed as percentage of total five-hour excretion after a 5 g. dose in (N) 16 normal subjects; (E) in four subjects with gluten-induced enteropathy.*

after 5 g. of xylose in 16 normal subjects and in the four patients with gluten-induced enteropathy and in four cases after partial gastrectomy. The excretion in each hour was calculated as a proportion of the total five-hour excretion. The normal subjects put out more than half of the five hours' excretion in the first two hours while the patients with gluten-induced enteropathy put out less than a third of their five hours' excretion in the first two hours (Fig. 3). The maximum excretion of xylose in four patients with partial gastrectomy was in the first hour (Fig. 2) and this would be expected from the rapid entry of the xylose into the small intestine of these patients. On the basis of these results we chose to measure the excretion of xylose in consecutive periods of two hours and three hours after a 5 g. dose. Table II also includes the results of the two-hour excretion and the 2 hr./5 hr. ratio in 40 of these normal subjects, the mean value for two hours being 23% with a standard deviation of 4·5 and a range of 14 to 34. The mean 2 hr.:5 hr. percentage ratio is 62 with a standard deviation of 10·1 and a range of 39 to 81. Table IV shows the reproducibility of this test in one normal person and one patient.

Tables V and VI show the results of this modified xylose excretion test in 32 patients with their clinical and biochemical abnormalities.

Table VII shows the findings in four patients with a normal xylose excretion test in spite of steatorrhoea.

TABLE IV

REPRODUCIBILITY OF TEST

	Xylose Excretion (%) in	
Day	*Two Hours*	*Five Hours*
1	22·1	31·0
2	21·9	33·7
3	22·3	34·4
4	24·5	35·8
5	24·5	34·1
6	23·6	38·0

Subject with gluten-induced Enteropathy

	Excretion (%) Dose Xylose							*Hourly (%) Five-hour Excretion*					
Day	*1*	*2*	*3*	*4*	*5*	*Two-hour Total*	*Five-hour Total*	*1*	*2*	*3*	*4*	*5*	*2/5*
1	0·9	4·0	4·7	3·3	2·3	4·9	15·2	5·9	26·3	30·9	21·7	15·1	32·2
3	1·3	5·2	5·5	4·2	2·8	6·5	19·0	6·8	27·3	28·9	22·1	14·7	34·1
5	0·6	3·5	3·1	3·7	2·4	4·1	13·3	4·5	26·3	23·2	27·8	18·0	30·8

TABLE V

RESULTS OF MODIFIED XYLOSE EXCRETION TESTS IN SELECTED CASES

Case No.	Age (yr.)	Sex	Two-hr. Xylose Excretion	Five-hr. Xylose Excretion	2:5-hr. Ratio	Faecal Fat (g./24 hr.)	Serum Iron (µg./100 ml.)	Serum Folate (µg./ml.)	Figlu	Serum B_{12} (pg./ml.)	Schilling Test (%)	Haemoglobin (g./100 ml.)	
1	56	F	5·7	30·7	18·5	6·2	—	1·7	—	—	8·0	4·6	Dimorphic anaemia with diarrhoea
2	54	M	10·9	36·2	30·0	1·7	58	—	Pos.	—	—	10·3	Dimorphic anaemia; angular stomatitis and glossitis; hiatus hernia
3	37	F	12·8	36·8	35·0	11·0	26	—	Pos.	100	—	9·6	Dimorphic anaemia; steatorrhoea; markedly dilated duodenum on *x*-ray; ? cause
4	37	F	10·8	29·0	37·0	3·0	50	1·7	Neg.	269	—	10·9	Dimorphic anaemia; buccal ulceration; stool nitrogen 3-5 g. per day
5	16	M	11·9	29·5	40·0	3·7	52	4·5	—	—	—	9·5	Iron-deficiency anaemia; slight dilatation of the ileum; occult blood loss
6	38	F	10·8	39·0	28·0	—	69	—	—	—	—	6·8	Iron-deficiency anaemia
7	43	F	8·0	28·0	29·0	—	60	—	—	243	—	9·0	Iron-deficiency anaemia; occult blood loss
			21·4	31·8	62·0	—						11·4	
After oral iron treatment													
8	16	M	12·0	30·0	40·0	—	34	0·5	—	—	—	8·9	Jejunitis (operative finding)
9	47	M	13·0	27·5	47·0	—	—	—	—	10	1·9	7·9	Addisonian anaemia
10	61	F	7·0	32·0	22·0	1·6	—	2·4	—	79	0·4	2·3	Addisonian anaemia; diarrhoea
11	61	F	10·9	29·8	37·0	1·3	—	—	Pos.	90	13·3	4·5	Epilepsy; megaloblastic anaemia due to mysoline
12	15	F	11·9	33·0	36·0	2·2	145	4·3	—	1,000	—	5·8	Subacute monoblastic leukaemia (patient died at home).
13	25	M	13·6	36·8	37·0	8·1	65	—	—	—	—	9·7	Duodenal ulcer; mild steatorrhoea
			18·4	38·0	48·0							14·7	Occult blood loss; complete recovery on medical treatment
After treatment													
14	55	M	13·8	30·8	45·0	30·0	—	—	—	—	—	11·9	Post-gastrectomy malabsorption
15	58	M	11·6	37·0	31·0	10·0	116	—	Pos.	—	—	13·2	Osteomalacia, post-gastrectomy malabsorption
16	38	F	10·0	40·0	25·0	—	—	2·3	—	—	—	12·8	Chronic ulcerative colitis, distal
17	59	M	10·7	29·1	37·0	3·0	—	—	—	265	—	13·0	Chronic ulcerative colitis, distal
18	21	F	13·7	32·4	42·0	2·7	40	4·7	—	172	—	12·3	Acute ulcerative colitis, total
19	21	F	10·0	28·0	36·0	5·4	34	0·6	—	—	—	9·8	Acute ulcerative colitis, total
20	39	F	13·0	33·0	39·0	—	28	3·0	—	273	—	10·1	Acute ulcerative colitis, distal
21	22	M	9·0	33·4	27·0	6·5	80	—	Pos.	595	—	13·0	Pityriasis rubra pilaris
22	28	M	11·4	31·1	37·0	3·0	—	—	Pos.	507	—	14·0	Hypogammaglobulinaemia; diarrhoea
23	12	M	13·2	29·7	45·0	8·0	—	—	—	—	—	—	Hypogammaglobulinaemia; steatorrhoea
24	63	F	11·5	28·1	40·1	7·0	—	Low	Neg.	217	—	12·3	Hypoproteinaemia; mild steatorrhoea
25	59	M	9·9	33·4	30·0	3·0	—	—	Neg.	—	—	14·3	Osteoporosis (stool nitrogen 2-3 g. per day)
26	21	M	10·2	29·9	52·0	4·5	30	—	—	—	—	9·6	Crohn's disease, lower ileum
27	23	F	4·8	28·0	20·0	—	—	—	Pos.	85	—	10·8	Crohn's disease ileum and caecum; fistulae; megaloblastic anaemia
28	59	M	9·8	29·2	38·5	12·6	—	—	—	—	—	13·1	Crohn's disease, ileum Hypoproteinaemia, wasting; partial villous atrophy of upper jejunum

TABLE VI

RESULTS OF XYLOSE EXCRETION TESTS IN FOUR SUBJECTS

Case No.	Age (yr.)	Sex	Two-hr. Xylose Excretion	Five-hr. Xylose Excretion	2:5-hr. Ratio	Faecal Fat (g./24 hr.)	Serum Iron (µg./100 ml.)	Serum Folate (µg./ml.)	Figlu	Serum B_{12} (pg./ml.)	Schilling Test (%)	Haemoglobin (g./100 ml.)	
29	31	F	16·4	24·0	68·0	8·0	—	—	—	—	—	11·0	Crohn's disease; resection of 95 cm. lower ileum
30	40	M	14·0	18·0	78·0	21·0	40	—	Neg.	60	—	8·1	Crohn's disease, jejunum and ileum; fistulae and blind loops on radiograph
31	29	F	14·4	17·2	83·0	25·0	80	—	Neg.	40	—	7·4	Crohn's disease, lower ileum, fistulae and blind loops on radiograph
32	32	M	14·0	24·0	58·0	—	—	—	—	—	—	15·2	Ulcerative colitis; lactose intolerance

TABLE VII

CASES WITH NORMAL XYLOSE TEST BUT ABNORMAL FAECAL FAT

Case No.	Xylose Excretion			Faecal Fat (g.)	Diagnosis
	Two-hr.	Five-hr.	2:5 Ratio		
33	20·9	35·8	58	10	Crohn's disease terminal ileum
34	20·1	35·6	57	8	Crohn's disease lower jejunum, ileum, and colon
35	17·2	32·8	54	50	Crohn's disease, ileum; protein-losing enteropathy
36	18·2	43·2	42	15	Carcinoma of caecum; subacute obstruction

DISCUSSION

RELATIONSHIP BETWEEN DOSE AND EXCRETION OF XYLOSE The amount of xylose excreted in five hours increased as the dose increased. There was a linear relationship between the dose given and the amount excreted for the lower doses up to 5 g. (Fig. 1). When the large doses are used the percentage of dose excreted is smaller. This deviation from linearity was evident for the two-hour and five-hour excretions.

The mean five-hour excretion after 25 g. was 25% which is very close to the values for normal subjects recorded in the literature (Sheehy and Floch, 1964). When this same dose is given intravenously to normal subjects about 40% is excreted in the urine in five hours (Fourman, 1948; Butterworth *et al.*, 1959) and this suggests that the absorption of a 25 g. dose is incomplete, even in normal subjects. The mean value for the five-hour excretion after a 5 g. dose was 36% which also agrees closely with the literature (Table II). Two subjects given 5 g. intravenously excreted 40 and 41% of the dose in five hours, suggesting that absorption of this smaller dose is complete. Complete absorption would explain the absence of intestinal disturbances when this dose is used; incomplete absorption would explain the diarrhoea which follows a 25 g. dose.

The irregular occurrence of diarrhoea after a 25 g. dose (Benson, Culver, Ragland, Jones, Drummey, and Bougas, 1957; Chanarin and Bennett, 1962) could account for the wide variation in results reported in normal subjects and the difficulty in defining the lowest result which can be accepted as normal. A dose of 5 g., however, gives reproducible results in normal and abnormal subjects (Tables IVa and IVb).

Finally, it is worth pointing out that this dose of 5 g. can be used successfully in children (Hubble and Littlejohn, 1963) and in our experience it does not cause diarrhoea, even in the smallest child.

PATTERNS OF EXCRETION In normal subjects most of the five-hour excretion was in the first two hours (Fig. 3). The lower limit of normal would appear to be 14% excretion in two hours or approximately 23-2 S.D. In patients with enteropathy most of the five-hour excretion was in the last three hours. The difference in the pattern of excretion could be explained by faulty absorption from the small intestine. This may or may not be influenced by stomach emptying time. The delay in excretion in the patients with intestinal disease could be due entirely to delay in stomach emptying, but since the xylose was always given as a water solution after an overnight fast and as there was no radiological evidence of slow stomach emptying in any of the subjects who were radiographed this explanation would seem unlikely.

The amount of xylose absorbed from the intestine depends on the rate of absorption and the duration of absorption; and the rate of excretion depends on the blood level (Wyngaarden, Segal, and Foley, 1957). Maximum excretion, like the maximum concentration of xylose in blood (Thaysen and Müllertz, 1962), is achieved in two hours and absorption is probably complete in this time in the normal person (McCance and Madders, 1930). The later excretion of xylose represents clearance of previously absorbed xylose. The slow two-hour excretion of the patients with enteropathy differentiates them from normal subjects and implies that in such patients there is a slower rate of absorption (Fig. 3). That the five-hour figure was normal in nearly all of these selected patients shows that prolonged absorption at this slower rate resulted in nearly complete absorption. The process of absorption is illustrated diagrammatically in Figure 4. The accumulation of xylose in the body can be represented in normal subjects by line A. When

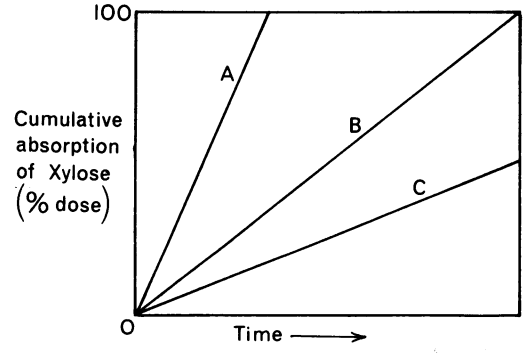

FIG. 4. *Process of absorption illustrated diagrammatically. The time axis represents the time during which absorption can occur from the small intestine. For explanation of lines A, B, and C, see text.*

damage to the intestine is gross the rate of absorption will be slow and absorption prolonged but xylose will leave the small intestine before absorption is complete, and therefore the five-hour excretion will be less than normal (line C). The degree of damage, however, could be such that although absorption is slow it will continue until all the xylose is absorbed (line B). There is obviously a limit to the time for which absorption can continue since the xylose will eventually leave the small intestine. The longest time is probably not more than five hours in the absence of obstruction and during that time if the amount of xylose absorbed per unit time were constant the accumulation of xylose in blood and urine would be linear (Fig. 1). The minimum value for the fraction of the five-hour excretion occurring in the first two hours would then be about 20% (Fig. 4, line B) and a value less than this would indicate that there was considerable delay in the stomach emptying, so that only a small fraction of the xylose was available for absorption.

These considerations form the theoretical basis of the modified xylose excretion tests, which included measurement of the two-hour and five-hour excretion and calculation of the two-hour to five-hour excretion ratio. With this modification, impairment of xylose absorption was demonstrated in 28 cases (Table V) even when the total five-hour xylose absorption was normal. In some of these cases there were other abnormalities suggestive of malabsorption. This supports the view that the low two-hour excretion of xylose is usually (if not always) attributable to upper intestinal malabsorption rather than delayed stomach emptying.

In most cases the abnormality of xylose absorption would have been detected by measurement of the two-hour excretion alone, but occasionally accurate interpretation depended on the five-hour excretion as well. In patients 29, 30, 31, and 32 (Table VI) the excretion and therefore the absorption of xylose in the first two hours was normal or nearly normal but little more was excreted in the following three hours and the five-hour figure was below normal. This suggested that only the uppermost part of the intestine was intact and able to absorb xylose whereas the lower intestine was diseased. All four patients had a disorder affecting the lower intestine. In these cases another explanation might be the metabolism of xylose by intestinal bacteria in the lower small bowel. However, we consider this improbable as we have been able to show, in a collaborative experiment with Dr. Bridgwater, that incubation of a 2% (w/v) solution of xylose in peptone broth with nine different strains of xylose-fermenting intestinal bacteria failed to destroy any xylose in five hours and only a maximum of 12% in 24 hours.

We have not considered in this paper the great majority of patients in whom steatorrhoea correlates with impaired xylose excretion both at two hours and five hours. We have, however, encountered four patients (Table VII) with steatorrhoea whose xylose absorption by all our criteria was normal, a result which suggested that the steatorrhoea was due to a disease confined to the most distal small intestine.

These last cases illustrate that a normal xylose excretion pattern does not entirely exclude malabsorption. We believe nevertheless that it is a useful screening test and is rendered more sensitive by the procedure we have recommended.

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