treatment clinics and in general practices. Additional investigations of the alcohol clinical index are planned in other contexts, such as specialist medical wards (for example, gastroenterology and psychiatry) and emergency service departments, where one might expect a higher prevalence of excessive drinking and alcohol related morbidity among patients.

The present study has provided a comprehensive comparison of the relative diagnostic value of clinical and laboratory information for the identification of alcohol abuse. Our findings underscore the value of selected items from the medical history and clinical signs, which can be combined to form an objective index. Previously, incomplete knowledge of the diagnostic power of specific clinical items has prevented firm recommendations about indicators of excessive drinking.¹¹ Our advice is to administer the alcohol clinical index routinely during clinical examination and to corroborate it by a brief questionnaire on alcohol problems such as the Michigan alcoholism screening test or CAGE (cut down on drinking, annoyed by others criticizing your drinking, guilt feelings over drinking, eye-opener or drink in the morning),^{15 20 28} as well as by laboratory tests determining mean corpuscular volume and γ -glutamyltransferase activity.¹⁴ This practical strategy could make significant inroads on identifying drinking problems that often remain undetected in medical wards and general practices.

References

- Wilkins RH. *The hidden alcoholic in general practice: a method of detection using a questionnaire*. London: Clerk, 1974.
- McIntosh ID. Alcohol-related disabilities in general hospital patients: a critical assessment of the evidence. *Int J Addict* 1982;17:609-39.
- Holt S, Stewart IC, Dixon JM, Elton RA, Taylor TV, Little K. Alcohol and the emergency service patient. *Br Med J* 1980;281:638-40.
- Buchan IC, Buckley EH, Deacon GLS, et al. Problem drinkers and their problems. *J R Coll Gen Pract* 1981;31:151-3.
- Kristensson H, Lunden A, Nilsson BE. Fracture incidence and diagnostic roentgen in alcoholics. *Acta Orthop Scand* 1980;51:205-7.
- Jankowski CB, Drum DE. Criteria for the diagnosis of alcoholism. *Arch Intern Med* 1977;137:1532-6.
- Skinner HA, Holt S, Allen BA, Haakonson NH. Correlation between medical and behavioral data in the assessment of alcoholism. *Alcoholism* 1980;4:371-7.
- Fisher JC, Mason RL, Fisher JV. A diagnostic formula for alcoholism. *J Stud Alcohol* 1976;37:1247-55.
- Lee K, Moller L, Hardt F, Haubek A, Jensen E. Alcohol-induced brain damage and liver damage in young males. *Lancet* 1979;ii:759-61.
- Skinner HA, Holt S, Israel Y. Early identification of alcohol abuse: critical issues and psychosocial indicators for a composite index. *Can Med Assoc J* 1981;124:1141-52.
- Holt S, Skinner HA, Israel Y. Early identification of alcohol abuse: clinical and laboratory indicators. *Can Med Assoc J* 1981;124:1279-94.
- Ryback RS, Ehardt MJ, Felsher B, Rawlings RR. Biochemical and hematologic correlates of alcoholism and liver diseases. *JAMA* 1982;248:2261-5.
- Chick J, Kreitman N, Plant M. Mean cell volume and gamma-glutamyl transpeptidase as markers of drinking in working men. *Lancet* 1981;ii:1249-51.
- Skinner HA, Holt S, Schuller R, Roy J, Israel Y. Identification of alcohol abuse using laboratory tests and a history of trauma. *Ann Intern Med* 1984;101:847-51.
- Bernadt MW, Mumford J, Taylor C, Smith B, Murray RM. Comparison of questionnaire and laboratory tests in the detection of excessive drinking and alcoholism. *Lancet* 1982;ii:325-8.
- Bernadt MW, Mumford J, Murray RM. A discriminant-function analysis of screening tests for excessive drinking and alcoholism. *J Stud Alcohol* 1984;45:81-6.
- Cushman P, Jacobson G, Barboriak JJ, Anderson AJ. Biochemical markers for alcoholism: sensitivity problems. *Alcoholism (NY)* 1984;8:253-7.
- National Council on Alcoholism. Criteria for the diagnosis of alcoholism. *Ann Intern Med* 1972;77:249-58.
- Ringer C, Kufner H, Antons K, Feuerlein W. The NCA criteria for the diagnosis of alcoholism. *J Stud Alcohol* 1977;38:1254-73.
- Selzer ML. The Michigan alcoholism screening test: the quest for a new diagnostic instrument. *Am J Psychiatry* 1971;127:165-8.
- Skinner HA, Sheu WJ. Reliability of alcohol use indices: the lifetime drinking history and the MAST. *J Stud Alcohol* 1982;43:1157-70.
- Dixon WJ, ed. *Biomedical computer programs*. Berkeley: University of California Press, 1981.
- Sox HC. Probability theory in the use of diagnostic tests. *Ann Intern Med* 1986;104:60-6.
- Fleiss JL. *Statistical methods for rates and proportions*. New York: Wiley, 1981.
- Johnson RD, Williams R. Prevention of hazardous drinking: the value of laboratory tests. *Br Med J* 1985;290:1849-50.
- Le Go PM. A propos de la lutte contre l'alcoolisme dans une grande collectivité de travail. *Bull Acad Natl Med* 1967;151:197-205.
- Babor TF, Weill J, Treffardier M, Bernard JY. Detection and diagnosis of alcohol dependence using the Le Go grid method. In: Chang NC, Chao HM, eds. *Early identification of alcohol abuse*. Rockville, Maryland: National Institute on Alcohol Abuse and Alcoholism, 1985. (Research monograph 17.)
- Skinner HA, Allen BA, McIntosh MC, Palmer WH. Lifestyle assessment: just asking makes a difference. *Br Med J* 1985;290:214-6.

(Accepted 24 April 1986)

Does super efficient starch absorption promote diverticular disease?

J R THORNTON, A DRYDEN, J KELLEHER, M S LOSOWSKY

Abstract

The amount of starch escaping absorption in the small intestine was measured in eight patients with symptomatic diverticular disease and eight controls. Unabsorbed starch was calculated from breath hydrogen measurements after a potato meal compared with the hydrogen response to lactulose. The proportion of unabsorbed starch was low in all the patients (mean 3.3%) and was only about a quarter of that in the controls (12.4%; $p < 0.01$).

These findings confirm that unabsorbed starch provides an important quantity of carbohydrate reaching the colon and suggest that super efficient starch absorption, by reducing this provision, may promote the development of diverticular disease.

Department of Medicine, St James's University Hospital, Leeds LS9 7TF

J R THORNTON, MD, MRCP, tutor in medicine, honorary senior registrar

A DRYDEN, BSC, SRD, dietician

J KELLEHER, PHD, senior lecturer in biochemistry in relation to medicine

M S LOSOWSKY, MD, FRCP, professor of medicine

Correspondence to: Dr Thornton.

Introduction

Recent work has shown that dietary starch is not completely absorbed by the small intestine.^{1,2} The degree of malabsorption of starch varies with different food sources.^{1,3} Furthermore, one recent study found wide variation in starch malabsorption among people eating the same meal, with between 2% and 20% of the starch escaping absorption in the small intestine.²

There is considerable evidence that a low intake of dietary fibre predisposes to diverticular disease.⁴⁻⁶ Fibre is the main exogenous source of colonic carbohydrate. In view of the possibility that unabsorbed starch may also contribute a sizable quantity of carbohydrate to the colon we hypothesised that people who absorb starch particularly well, such that little reaches their colon, might be more at risk of diverticular disease. We call this better than average starch absorption "super efficient."

Subjects and methods

We studied eight patients with symptomatic diverticular disease. They had presented complaining of lower abdominal pain, with or without altered bowel habit, and had at least 10 colonic diverticula seen on barium enema.

All were improved by a high fibre diet. They were compared with eight controls individually matched for sex (five women, three men) and age (mean age of patients 66 (SEM 4) years, controls 64 (3) years). None of the subjects had had gastrointestinal surgery, none was taking any regular medication, and none had taken antibiotics within the past three months. No subject gave a history to suggest lactose malabsorption. In order to allow a reasonable interval for their usual colonic bacterial activity to return the patients were studied for a median of 10 months (range 5-21) after diagnostic investigation of their large bowel. All the subjects were in good health when studied and all abstained from alcohol for at least 24 hours before each study day.

After a 14 hour fast on two separate days one to three weeks apart the subjects consumed either 6.5 g of the non-absorbable disaccharide lactulose or a carefully standardised meal of potatoes. Meals were allocated in random order. The potatoes were peeled, cut to roughly equal size, and boiled for 20 minutes. This meal provided 60 g starch, 3.3 g fibre, 1.3 g sugars, 4.6 g protein, and 0.3 g fat. The lactulose was diluted in 200 ml water and a similar quantity of water was drunk with the potato meal. Subjects were required to consume the potato meal or lactulose over 10-15 minutes, to remain largely sedentary, and were not allowed to smoke during the test.⁷

Hydrogen is one of the products of bacterial fermentation of carbohydrate entering the colon. Respiratory hydrogen is derived almost entirely from colonic bacterial action, and its pulmonary excretion is an accurate indicator of production of hydrogen in the colon.⁸ End expiratory samples of breath were collected in a modified Haldane-Priestley tube and hydrogen measured by a Key-Med monitor every 15 minutes until hydrogen generation was complete. Usually eight to 12 hours of measurements was necessary. The amount of hydrogen produced from carbohydrate in the colon is linearly proportional to the amount of carbohydrate substrate.⁹ Since it also varies considerably with different colonic flora⁹ the lactulose was used to provide a measure of each subject's colonic bacterial capacity to produce hydrogen. The quantity of potato starch entering the colon was calculated for each subject from the relative amounts of hydrogen generated by the potatoes and the lactulose.¹⁰ Since fermentation of the small amount of fibre in the potatoes would add to the hydrogen production,¹¹ and assuming 60% breakdown of this fibre, 2 g was deducted from each calculated value as an approximate correction. Mouth to caecum transit time of the head of the potato meal or lactulose was taken as the first sustained rise in breath hydrogen of five or more parts per million.¹² The statistical significance of differences was calculated by the Mann-Whitney U test.

Results

The percentage of unabsorbed starch was low in all the patients (mean 3.3 (SEM 0.5)%) and was only about a quarter of that found in the controls (12.4 (1.8)%; $p < 0.01$) (figure). Mouth to caecum transit time was not significantly different between the patients and controls for either the potato meal (321 (SEM 37) and 272 (37) minutes respectively) or the lactulose (128 (17) minutes and 140 (16) minutes). All the subjects had low concentrations (less than 10 ppm) of fasting breath hydrogen, excluding the possibility that any of them had bacterial overgrowth in the small bowel.¹⁴

Discussion

This study shows that patients with symptomatic diverticular disease absorb starch better than controls and consequently that less starch reaches their colon. It confirms the report that people vary considerably in their malabsorption of starch.² These findings and our recent, similar results in patients with colonic adenomas¹⁴ are the first published evidence of a difference in starch malabsorption in any disease.

The factors underlying this difference are unclear. Mouth to caecum transit time was similar for unabsorbed potato starch in the two groups. As this measurement encompasses both gastric and small bowel transit we do not know whether the time the potatoes remained in the small bowel was an important determinant of starch absorption. In a recent study, however, even considerable prolongation of the time a meal resided in the jejunum was associated with only a small increase in carbohydrate absorption.¹⁵ Thus it seems unlikely that differences in small bowel transit time could be the only reason for the variability of starch absorption among individuals.

Our controls were healthy volunteers. We did not think it ethical to ask them to undergo colonic investigations, so we cannot exclude

the possibility that a few may have had symptomless diverticular disease, which occurs in roughly a quarter of this age group.¹⁶ If, however, we had chosen as controls patients whose large bowel had been proved to be structurally normal we believe that many would have had conditions which are characterised by abnormal intestinal function, such as constipation and the irritable bowel syndrome. Furthermore, if any of the controls had diverticular disease it is likely to have been milder than in the patients, all of whom had relatively severe, symptomatic disease.

Percentage of unabsorbed starch in eight patients with symptomatic diverticular disease and eight controls. Bars are means.

We have found previously in similar subjects that potatoes and wheat products usually provide more than 90% of the starch eaten.¹⁴ Since the propensity for starch malabsorption is similar for these two foods¹⁴ the measured values should provide a good approximation in these subjects of the unabsorbed starch from all food sources. Given an average British person's daily consumption of 150 g starch¹⁷ and 22 g fibre¹⁸ our results suggest that in the controls 18.6 g starch would escape absorption in the small bowel each day compared with only 5.0 g in the patients. Thus the difference between the two groups of 13.6 g unabsorbed starch represents an amount of carbohydrate entering the colon equivalent to two thirds of that provided by the average daily intake of fibre. This difference may prove to be less in people whose diverticular disease is asymptomatic.

Our findings confirm that unabsorbed starch is an important source of carbohydrate reaching the colon. While more patients clearly need to be studied before firm conclusions can be drawn, the data suggest that a diet rich in foods containing starch may exert a protective action against diverticular disease by virtue both of its starch as well as its fibre content. In populations having a comparatively low intake of fibre super efficient starch absorption, by reducing the provision of carbohydrate to the colon, may be an important determinant of a person's susceptibility to diverticular disease.

We thank Mrs S Shires for technical work, Dr D W W Bullimore for compiling a computer program to calculate the area under the "curve" of the breath hydrogen responses, and the Friends of St James's Hospital, some of whose members served as controls.

References

- Anderson IH, Levine AS, Levitt MD. Incomplete absorption of the carbohydrate in all-purpose wheat flour. *N Engl J Med* 1981;304:891-2.
- Stephen AM, Haddad AC, Phillips SF. Passage of carbohydrate into the colon. Direct measurements in humans. *Gastroenterology* 1983;85:589-95.
- Levine AS, Levitt MD. Malabsorption of the starch moiety of oats, corn and potatoes. *Gastroenterology* 1981;80:1209.
- Painter NS, Burkitt DP. Diverticular disease of the colon; a 20th century problem. *Clin Gastroenterol* 1975;4:3-21.
- Brodrick AJM, Humphreys DM. Diverticular disease: three studies. I. Relation to other disorders and fibre intake. *Br Med J* 1976;i:424-5.
- Gear JSS, Ware A, Fursdon P, et al. Symptomless diverticular disease and intake of dietary fibre. *Lancet* 1979;ii:511-4.
- Tadesse K, Eastwood M. Breath hydrogen test and smoking. *Lancet* 1977;iii:91.
- Levitt MD. Production and excretion of hydrogen gas in man. *N Engl J Med* 1969;281:122-7.
- Bond JH, Levitt MD. Use of pulmonary hydrogen measurements to quantitate carbohydrate absorption. *J Clin Invest* 1972;51:1219-25.
- Flourie B, Florent C, Jouany J-P, Thivend P, Etanchaud F, Rambaud J-C. Colonic metabolism of wheat starch in healthy humans. Effects on fecal outputs and clinical symptoms. *Gastroenterology* 1986;90:111-9.
- Tadesse K, Eastwood MA. Metabolism of dietary fibre components in man assessed by breath hydrogen and methane. *Br J Nutr* 1978;40:393-6.
- Bond JH, Levitt MD. Investigation of small bowel transit time in man utilising pulmonary hydrogen measurements. *J Lab Clin Med* 1975;85:546-55.
- Perman JA, Modler S, Barr RG, Rosenthal P. Fasting breath hydrogen concentration: normal values and clinical application. *Gastroenterology* 1984;87:1358-63.
- Thornton JR, Dryden A, Kelleher J, Losowsky MS. Does super-efficient starch absorption promote colonic neoplasia? *Gut* 1985;26:A554.
- Holgate AM, Read NW. Effect of ileal infusion of intralipid on gastrointestinal transit, ileal flow rate, and carbohydrate absorption in humans after ingestion of a liquid meal. *Gastroenterology* 1985;88:1005-11.
- Manousos ON, Truelove SC, Lumsden K. Prevalence of colonic diverticulosis in general population of Oxford area. *Br Med J* 1967;iii:762-3.
- Bingham S, McNeill NI, Cummings JH. The diet of individuals: a study of a randomly-chosen cross-section of British adults in a Cambridgeshire village. *Br J Nutr* 1981;45:23-35.
- Wenlock RW, Buss DH, Agater IB. New estimates of fibre intake in the diet in Britain. *Br Med J* 1984;288:1873.

(Accepted 24 April 1986)

SHORT REPORTS

Causalgia as a complication of meningococcal meningitis

We report this case for three reasons. Firstly, as a reminder that a normal cerebrospinal fluid does not exclude meningococcal infection; secondly, to report causalgia as a complication of meningococcal meningitis; and, thirdly, as an example of the efficacy of guanethidine blocks in the management of causalgia.

Case report

A 19 year old student, who had developed a cramp like pain in both calves two days previously, was admitted with a sore throat, headache, and fever. On admission his temperature was 39.2°C. He had a widespread macular rash and mild neck stiffness. Cerebrospinal fluid pressure measured at lumbar puncture was 21 cm H₂O, with a total protein concentration of 0.44 g/l, and glucose concentration of 4.4 mmol/l (79.3 mg/100 ml). Microscopic examination showed 25 red blood cells and five lymphocytes; no organisms were seen. He was treated with intravenous penicillin. Blood cultures and cerebrospinal fluid which had been taken on admission grew *Neisseria meningitidis*. He improved rapidly but complained of increasing burning pain in his left foot and lower leg associated with paraesthesiae. The pain, which was over the lateral aspect of his calf and foot, was not exacerbated by coughing or performing Kernig's manoeuvre and was accompanied by a deep pain in his calf. He had decreased power of dorsiflexion and plantarflexion at the left ankle, and the left ankle jerk was absent. There was hypersensitivity to light touch and pinprick over the lateral aspect of the left foot. The rest of his neurological examination was normal. Eight days after admission his cerebrospinal fluid had a protein concentration of 1.78 g/l, a normal glucose concentration, and a polymorphonuclear leucocytosis. It was sterile. A radiograph showed osteoporosis of the bones of the left foot. A lumbar myelogram and computed tomogram of the head were normal. Electromyographic studies of the left leg showed denervation of the left gastrocnemius and tibialis anterior muscles.

During the next three weeks the pain increased. All the muscles in his left foot became wasted and the skin over the foot was vasodilated and sweated excessively. He was treated with five guanethidine blocks using the method described by Hannington-Kiff,¹ and the pain decreased progressively after each block. The technique is similar to that of a Bier's block except that guanethidine rather than lignocaine is used. Seven weeks after admission he was discharged, no longer requiring analgesia and with only a slight limp.

Comment

This case is a reminder that in early meningococcal infection the cerebrospinal fluid may be normal biochemically and cytologically despite the presence of the organism.

In bacterial meningitis the spinal cord is rarely affected; we found reports of only four cases, two of which occurred before antibiotics were available.^{2,4} Our patient had weakness in the distribution of L5 and S1 with electromyographic evidence of denervation, but the exact site of the lesion is uncertain.

Hyperpathia was described by Loh and Nathan⁵ as a burning pain resulting from any stimulus, which radiates from the stimulus and lasts an abnormally long time. The area of abnormal sensation characteristically spreads beyond the original territory and is associated with a deep pain, increased sweating and vasodilatation, and a good response to guanethidine

blocks, as in our patient. The underlying mechanism behind the efficacy of guanethidine blockade in the management of patients with causalgia is disputed,⁶ but it probably operates by modifying the large afferent input to the spinal cord.

We thank Dr P Verril and Dr G M Stern for their advice and permission to report this case.

- Hannington-Kiff J. Intravenous regional sympathetic block with guanethidine. *Lancet* 1974;ii:1019-20.
- Turner JWA. Spinal cord lesions in cerebrospinal fever. *Lancet* 1948;ii:398-402.
- Graus F, Arbizu T, Rufi G. Partial Brown-Sequard's syndrome and meningococcal meningitis. *Arch Neurol* 1981;38:602.
- Seay AR. Spinal cord dysfunction complicating bacterial meningitis. *Arch Neurol* 1984;41:545-6.
- Loh L, Nathan PW. Painful peripheral states and sympathetic blocks. *J Neurol Neurosurg Psychiatr* 1978;41:664-71.

(Accepted 1 April 1986)

Department of Neurology, University College Hospital, London WC1E 6AU
J McLELLAND, MB, MRCP, registrar
S J ELLIS, MA, MB, senior house officer

Correspondence to: Dr McLelland, Department of Dermatology, Hammersmith Hospital, London W12 0HS.

Clinical and financial implications of a district scheme to provide plastic insulin syringes to diabetics

Despite the manufacturers' instructions to discard plastic insulin syringes after single use, multiple reuse is safe and acceptable to patients.^{1,2} If multiple use of plastic syringes can be ensured they cost less than glass syringes. In March 1983 the change to U100 insulin in the United Kingdom provided an opportunity to offer plastic syringes to patients instead of glass ones, to encourage their reuse, and to evaluate costs, problems, and patient preferences. The Southampton district accepted this policy in anticipation that it would be cost effective.

Methods and results

A doctor and state registered nurse gave detailed instruction in the use of U100 insulin at a special clinic. Sixty Becton-Dickinson 0.5 ml or 1.0 ml plastic syringes were supplied for the first year for all patients except those who requested glass replacements. It was suggested that each syringe should be used for at least one week. Patients were instructed before injection to clean the skin with soapy water and tissue rather than surgical spirit, which by transfer on the fingers to the plastic syringes causes the markings to rub off. Despite the fact that the plastic syringes used contained no "dead space," the insulin dosage was not reduced and there were no appreciable problems in diabetic control as a result of this policy.³