growth and maturity in growth hormone deficiency treated with human growth hormone. *Acta Paediatr Scand* 1984;73:120-6.
26 Prader A, Zachmann M, Poley JR, Illig R, Szeky J. Long-term treatment with human growth hormone (Raben) in small doses: evaluation of 18 hypopituitary patients. *Helv Paediatr Acta* 1967;22:423-40.
27 Finkelstein JW, Roffwarg HP, Boyar RM, Kream J, Hellman L. Age-related change in the 24 hour spontaneous secretion of growth hormone. *J Clin Endocrinol Metab* 1972;35:665-70.
28 Zadik Z, Chalew SA, McCarter RJ, Meistas M, Kowarski A. The influence of age on the 24-hour integrated concentration of growth hormone in normal individuals. *J Clin Endocrinol Metab* 1985;60:513-6.
29 Hindmarsh PC, Stanhope R, Kendall BE, Brook CGD. Tall stature: a clinical, endocrinological and radiological study. *Clin Endocrinol (Oxf)* 1986;25:223-31.
30 Rosenfeld RG, Kemp SF, Hintz RL. Constancy of somatomedin response to growth hormone treatment of hypopituitary dwarfism and lack of correlation with growth rate. *J Clin Endocrinol Metab* 1981;53:611-7.
31 Nilsson A, Isgaard J, Lindahl A, Dahlstrom A, Skottner A, Isaksson OGP. Regulation by growth hormone of number of chondrocytes containing IGF-1 in rat growth plate. *Science* 1986;233:571-4.
32 Schlecter NL, Russell SM, Spencer EM, Nicoll CS. Evidence suggesting that the direct growth-promoting effect of growth hormone on cartilage in vivo is mediated by local production of somatomedin. *Proc Natl Acad Sci USA* 1986;83:7932-4.
33 Trippel SB, Van Wyk JJ, Mankin HJ. Localization of somatomedin-C binding to bovine growth-plate chondrocytes in situ. *J Bone Joint Surg (Am)* 1986;68:897-903.
34 Lippe BM, Van Herle AJ, La Franchi SH, Uller RP, Lavin N, Kaplan SA. Reversible hypothyroidism in growth hormone-deficient children treated with human growth hormone. *J Clin Endocrinol Metab* 1975;40:612-8.
35 Root AW, Snyder PJ, Revzani I, Digforce AM, Utiger RD. Inhibition of thyrotropin-releasing hormone-mediated secretion of thyrotropin by human growth hormone. *J Clin Endocrinol Metab* 1973;36:103-7.
36 Porter BA, Refetoff S, Rosenfeld RL, De Groot LT, Fang VS, Stark V. Abnormal thyroxine metabolism in hyposomatotropic dwarfism and inhibition of responsiveness to TRH during hGH therapy. *Pediatrics* 1973;51:668-74.
37 Conway S, McCann SM, Krulich L. On the mechanism of growth hormone autofeedback regulation: possible role of somatostatin and growth hormone-releasing factor. *Endocrinology* 1985;117:2284-92.
38 Nakamoto JM, Gertner JM, Press CM, Hintz RL, Rosenfeld RG, Genel M. Suppression of the growth hormone (GH) response to clonidine and GH-releasing hormone by exogenous GH. *J Clin Endocrinol Metab* 1986;62:822-6.
39 Rosenthal SM, Hulse JA, Kaplan SL, Grumbach MM. Exogenous growth hormone inhibits growth hormone-releasing factor induced growth hormone secretion in normal men. *J Clin Invest* 1986;77:176-80.
40 Ross RJM, Borges F, Grossman A, *et al*. Growth hormone pretreatment in man blocks the response to growth hormone-releasing hormone; evidence for a direct effect of growth hormone. *Clin Endocrinol (Oxf)* 1987;26:117-23.
41 Underwood LE. Report of the conference on uses and possible abuses of biosynthetic HGH. *N Engl J Med* 1984;311:606-8.
42 Rosenfeld RG, Wilson DM, Dollar LA, Bennett A, Hintz RL. Both human pituitary growth hormone and recombinant DNA derived human growth hormone cause insulin resistance at a postreceptor site. *J Clin Endocrinol Metab* 1982;54:1033-8.
43 Scheiwiller E, Guler HP, Merryweather J, *et al*. Growth restoration of insulin-deficient diabetic rats by recombinant human insulin-like growth factor I. *Nature* 1986;323:169-71.
44 Binoux M, Lassarre C, Hardouin N. Somatomedin production by rat liver in organ culture. III. Studies on the release of insulin-like growth factor and its carrier protein measured by radiological assays. *Acta Endocrinol (Copenh)* 1982;99:422-32.
45 Bucher H, Zapf J, Torresani T, Prader A, Froesch ER, Illig R. Insulin-like growth factor I and II. Prolactin and insulin in 19 growth hormone deficient children with excessive, normal or decreased longitudinal growth after operation for craniopharyngioma. *N Engl J Med* 1983;309:1142-6.
46 Milner RDG. Clinical experience of somatrem: UK preliminary report. *Acta Paediatr Scand* 1986;325(suppl):25-8.
47 Takano K, Shizume K. Clinical experience with somatrem in Japan. *Acta Paediatr Scand* 1986;325(suppl):19-24.
48 Preece MA. Experience of treatment with pituitary derived human growth hormone with special reference to immunological aspects. In: Milner RDG, Flodh H, eds. *Immunological aspects of human growth hormone*. Oxford: Medical Education Services, 1985:9-17.
49 Holder AT, Aston R, Preece MA, Ivanyi J. Monoclonal antibody-mediated enhancement of growth hormone activity in vivo. *J Endocrinol* 1985;107:R9-12.

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SHORT REPORTS

Severe sexual dysfunction in women with the irritable bowel syndrome: comparison with inflammatory bowel disease and duodenal ulceration

Good evidence now exists that the irritable bowel syndrome is a much more diffuse gut disorder than was originally appreciated,[1] and we reported recently that among other symptoms women with the syndrome commonly suffer from dyspareunia.[2] Relatively little attention has been paid to the problem of sexual dysfunction in patients with gastrointestinal disorders except in relation to pelvic or abdominal surgery. We undertook a more detailed evaluation of sexual function in women with the irritable bowel syndrome, using groups of women with colonic inflammatory bowel disease and duodenal ulceration as controls.

Patients, methods, and results

Fifty consecutive women outpatients with the irritable bowel syndrome (abdominal pain, abdominal distension, and an abnormal bowel habit) were studied, with no refusals. Patients with painless diarrhoea were excluded. The control group consisted of 30 patients with active duodenal ulceration and 30 with active inflammatory bowel disease affecting the colon with no history of surgery. Five patients with inflammatory bowel disease and six with duodenal ulceration had symptoms suggesting coexisting irritable bowel syndrome and were not included in the control group because we would not have been able to ascertain which disorder was contributing to any sexual problem reported.

Forty two, 27, and 25 of the patients with, respectively, the irritable bowel syndrome, inflammatory bowel disease, and duodenal ulceration were sexually active. The distribution of social class in the groups was similar, but the mean age of the patients with duodenal ulcers was higher by nine years. Subjects were interviewed by a woman doctor (a trained psychiatrist) in their own homes. As part of a wider assessment of psychosocial state patients completed a self report questionnaire about sexual function in relation to their bowel disorder; only those with a score indicating severe or very severe disturbance (4 or 5 on a five point scale) were considered positive for the purposes of this analysis. Psychiatric state was measured with the psychiatric assessment schedule, a score of 11 or more indicating possible psychiatric illness.[3] Results were analysed with contingency tables (χ^2).

The table shows that the irritable bowel syndrome was associated with a profound impairment of sexual function, with 83% of patients reporting problems compared with 30% of women with inflammatory bowel disease and 16% of those with duodenal ulcers. When patients with psychiatric disorder were excluded from the analysis the same significant trend emerged, with 77%, 29%, and 14%, respectively, of women showing sexual dysfunction.

Comment

This study showed that sexual dysfunction is common in women with the irritable bowel syndrome. The presence of abdominal symptoms cannot be the sole explanation as the controls were specifically chosen because they had abdominal disease. In addition, the explanation cannot simply be the presence of psychopathology[4] because when women with psychiatric

Number (%) of sexually active women with the irritable bowel syndrome, inflammatory bowel disease, and duodenal ulceration with sexual dysfunction, and its relative significance

	Irritable bowel syndrome	Inflammatory bowel disease	Duodenal ulceration	Irritable bowel syndrome *v* inflammatory bowel disease *v* duodenal ulceration		Inflammatory bowel disease *v* duodenal ulceration		Irritable bowel syndrome *v* inflammatory bowel disease and duodenal ulceration	
				χ^2	p	χ^2	p	χ^2	p
Sexual function affected by bowel disorder									
All	35/42 (83)	8/27 (30)	4/25 (16)	34·7	<0·001	0·96	0·33	33·7	<0·001
Without psychiatric disorder	17/22 (77)	6/21 (29)	2/14 (14)						
Abdominal pain on sexual intercourse									
All	29/42 (69)	2/27 (7)	0/25	45·0	<0·001	0·32	0·57	44·7	<0·001
Without psychiatric disorder	13/22 (59)								
Vaginal pain on sexual intercourse									
All	7/42 (17)	2/27 (7)	3/25 (12)	1·28	0·26	0·24	0·62	1·04	0·31
Without psychiatric disorder	3/22 (14)	1/21 (5)	1/14 (7)						

disorder were excluded the same significant differences remained. Thus sexual dysfunction and the irritable bowel syndrome are specifically related, but the mechanism remains speculative. A particular feature was that sexual intercourse often induced abdominal pain, which often had a delayed onset. This may reflect secondary colonic spasm, although it may have originated within the genitourinary tract. More subtle psychological mechanisms must also be considered.

The women in our study probably represent the severe end of the range of the irritable bowel syndrome. Thus extrapolation of these results to the syndrome as a whole is not justified. Our findings suggest, however, that sexual dysfunction in the irritable bowel syndrome is a hitherto completely unrecognised aspect of this condition that needs to be addressed.

1 Cann PA, Read NW. A disease of the whole gut? In: Read NW, ed. *Irritable bowel syndrome*. New York: Grune and Stratton, 1985:53-63.
2 Whorwell PJ, McCallum M, Creed FH, Roberts CT. Non-colonic features of irritable bowel syndrome. *Gut* 1986;27:37-40.
3 Wing JK, Mann SA, Leff JP, Nixon JM. The concept of a 'case' in psychiatric population surveys. *Psychol Med* 1978;8:203-17.
4 Swan M, Wilson LJ. Sexual and marital problems in a psychiatric out-patient population. *Br J Psychiatry* 1979;135:310-14.

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^{99m}Tc-Sucralfate scintigraphy and colonic disease

Sucralfate is an aluminium substituted disaccharide, which at low pH binds to sites of ulceration in the gastrointestinal tract. Dawson *et al* have recently reported that radionuclide abdominal scans taken 12-24 hours after ingestion of ^{99m}Tc-sucralfate provide a range of normal appearances in healthy subjects and the pattern of ileal or colonic abnormalities in inflammatory bowel disease.[1] This is surprising in view of the low binding affinity of sucralfate at ileocolonic pH.[2] We have studied a series of consecutive patients undergoing routine colonoscopy immediately preceded by ^{99m}Tc-sucralfate scintigraphy.

Patients, methods, and results

We studied 18 patients. Bowel preparation comprised clear fluids for five days preceding colonoscopy and Picolax taken over the last 36 hours of this period. Seventeen to 20 hours before colonoscopy, at least two hours after the last dose of Picolax, each subject drank 100 MBq of ^{99m}Tc labelled sucralfate in 10 ml fluid.

Comparison of colonoscopic findings and interpretation of ^{99m}Tc-sucralfate scintiscans (figures are numbers of cases)

Colonoscopic diagnosis	Areas of uptake
Normal	Caecum (2), whole colon (3), transverse colon (1), hepatic flexure (1)
Left sided colonic neoplasms (6) (6 benign polyps, 1 carcinoma)	Caecum (4), whole colon (1), hepatic flexure, ileum, and rectum (1)
Inflammatory bowel disease (5)	
Left sided (3)	Caecum and ascending colon (3)
Pancolitis (2)	Ascending and transverse colon (1), caecum (1)

This was prepared with a 1 g tablet of sucralfate according to the method of Vasquez *et al*.[3] Labelling efficiency was 99·5% (SD 0·6%; n=10). Patients underwent abdominal scintigraphy next morning, roughly one hour before colonoscopy. Scans were reported without knowledge of the colonoscopic findings. The study was approved by the local ethical committee.

The quality of bowel preparation assessed at colonoscopy was good in all but one patient, who had adult Hirschsprung's disease. Pancolonoscopy was successful in all patients. The table shows the final diagnoses and scintigraphic findings.

Comment

We were unable to define a range of normal scintigraphic appearances and found no relation between areas of increased radioactivity and the distribution of inflammatory bowel disease. In addition, there was no relation between the magnitude of retained abdominal radioactivity and the extent or activity of disease, some normal subjects having a higher retention of radioactivity than those with extensive inflammatory bowel disease.

One explanation for the difference between our results and those of Dawson *et al* may be related to bowel preparation. Unlike us, Dawson *et al* used mannitol; this disaccharide undergoes bacterial fermentation in the colon, tending to produce an acid environment favouring sucralfate binding to ulcerated mucosa.[2] After ingestion of the undigested carbohydrates lactitol and lactulose intraluminal pH of the ascending colon falls from 6·5 to 5·2-5·6, whereas the pH in the descending colon and rectum is unaltered[4]; probably similar changes occur after mannitol. Binding of ^{99m}Tc-sucralfate occurs only when the exudative mucosal proteins are positively charged[2] and, as their isoelective points range from 4·8 to 7·2, the relatively modest change in pH that occurs after mannitol is unlikely appreciably to enhance ^{99m}Tc-sucralfate binding.

In vitro tests of adherence of the isotope to the parent molecule and the absence of gastric mucosal uptake show that our disappointing findings were not due to disruption of the ^{99m}Tc-sucralfate complex. We therefore suggest that labelled sucralfate attaches to luminal contents rather than adhering to the mucosa. This is supported by the caecal "hot spots" found in those patients shown to have caecal pooling at colonoscopy and by the observation that the one poorly prepared patient who had pronounced faecal retention at the time of endoscopy also had increased retention of radioactivity throughout the colon in the absence of any mucosal abnormality.

1 Dawson DJ, Khan AN, Miller V, Ratcliffe JF, Shreeve DR. Detection of inflammatory bowel disease in adults and children: evaluation of a new isotopic technique. *Br Med J* 1985;291: 1227-30.
2 Scopinaro F, Signori C, Di Rocco E, Liberatore M. Diagnostic possibility of 99m Tc sucralfate in reflux oesophagitis. In: Malaguti P, Sciarretta G, Abbati A, Furno A, eds. *New radioisotope tests in gastroenterology*. Milan: Masson Itali Editori, 1986:102-4.
3 Vasquez TE, Bridges RL, Braunstein P, Jansholt AL, Meshkinpour H. Gastro-intestinal ulcerations: detection using technetium 99m-labelled ulcer-avid agents. *Radiology* 1983;148: 227-31.
4 Patil DH, Westaby D, Mahida YR, *et al*. Comparative modes of action of lactitol and lactulose in the treatment of hepatic encephalopathy. *Gut* 1987;28:252-9.

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Mechanisms responsible for thirst and polyuria associated with primary hyperaldosteronism

Renal resistance to the antidiuretic action of vasopressin is the recognised cause of the polyuria and thirst that are associated with primary aldosteronism and is believed to be due to chronic hypokalaemia, which leads to renal tubular dysfunction and nephrogenic diabetes insipidus.[1] We describe a patient with this disorder whose thirst and polyuria were principally due to an unusual form of hypothalamic diabetes insipidus; nephrogenic diabetes insipidus, although present, was minimal.

Case report

A 43 year old woman with no medical history presented complaining of thirst, polyuria, lethargy, and weakness that had persisted for six months. She was not taking any drugs and did not eat liquorice. Blood pressure was persistently raised at 170-180/100 mm Hg. She was otherwise normal on physical examination. Urine volume ranged from 4·0 to 5·7 l/24 hours. Preliminary investigations showed mild hypokalaemia (plasma potassium concentration 2·8-3·0 mmol/l); plasma sodium, urea, creatinine, and calcium blood glucose concentrations were all normal. Resting supine plasma renin activity was low at 3·0 pmol/l/min (normal range 0·8-14·5), but plasma aldosterone concentration was raised at 879