

the second cytotoxic treatment of a pair usually causes less vomiting than the first.

About 18 hours after treatment patients assessed nausea by marking a unipolar visual analogue scale 100 mm long at the point most closely equivalent to their experience, 0 mm being equivalent to no nausea at all and 100 mm the worst nausea imaginable. They recorded the number of vomits with a digital counter, and nausea and vomiting were also graded by a ward observer as none, mild, moderate, or severe. To assess tolerance the occurrence of any side effects was recorded.

Data were analysed by non-parametric statistical methods—namely, visual analogue scores by a split plot analysis of variance after application of an arcsine square root transformation to the raw data, and the total number of patients who did not vomit with the Mann-Whitney test.

The incidence of vomiting with the MOPP regimen was greater than that with the CHOP regimen ($p < 0.05$), but there was no significant difference in these groups between the effects of the three antiemetic treatments. The table shows that all three treatments were similarly effective in preventing vomiting.

Proportions (and %) of patients who did not vomit over 18 hours

Regimen	Antiemetic		
	Chlorpromazine 99 mg	Nonabine 15 mg	Combination
MOPP	9/15 (60)	7/16 (44)	9/16 (56)
CHOP	25/31 (81)	28/32 (88)	24/29 (83)

MOPP = Mustine, vincristine, procarbazine, and prednisolone.
CHOP = Cyclophosphamide, adriamycin, vincristine, and prednisolone.

No patients rated their vomiting as severe, and nausea was rated only mild to moderate. In the patients who received the MOPP regimen the mean visual analogue score was significantly lower on the eighth day than on the first day with all three antiemetic treatments ($p < 0.01$), confirming the impression that the second cytotoxic treatment of a pair causes less nausea than the first. In this group on day 1 nausea was subjectively less after chlorpromazine than after nonabine, but this trend was not significant.

Comment

Both nonabine and chlorpromazine appeared adequate for controlling nausea and vomiting in patients treated with the CHOP regimen but failed to control vomiting in around 40% of those receiving the MOPP regimen. Possibly this figure could be improved by giving higher or repeated doses. Combining nonabine with chlorpromazine did not improve the antiemetic effect and was so sedative that the doses had to be reduced.

Side effects were fairly minor with each antiemetic, several symptoms, including drowsiness, dizziness, dry mouth, and headache, being common to both drugs. Cannabis-type "highs" did not occur, and postural hypotension and vagotonia, which have been reported with cannabis and its analogues, were also absent.³⁻⁵

This study shows that the antiemetic effects of nonabine and chlorpromazine in the doses used are similar. Side effects were not a serious problem with either drug.

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¹ Clark JA, Clarke MSG, Cooke A. 2,2-Dimethyl-7-(3-methyl-2-octyl)-4-(4-pyridyl) 2H-chromen-5-ol (BRL 4664), a novel psychotherapeutic agent. Cited by: Pars HG, Razden RK, Howes JF. Potential therapeutic agents derived from the cannabinoid nucleus. *Adv Drug Res* 1977;11: 97-189.

² Seigel LJ, Longo DL. The control of chemotherapy-induced emesis. *Ann Intern Med* 1981;95:352-9.

³ Sallan SE, Zinberg LE, Frie E. Antiemetic effect of delta-9-tetra-hydrocannabinol in patients receiving cancer chemotherapy. *N Engl J Med* 1975;293:795-7.

⁴ Lucas VS, Laszlo J. Delta-9-tetrahydrocannabinol for refractory vomiting induced by cancer chemotherapy. *JAMA* 1980;243:1241-3.

⁵ Anonymous. Cannabinoids for nausea. (Editorial.) *Lancet* 1981;i:255-6.

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Cerebrovascular accident in a 14 year old marathon runner

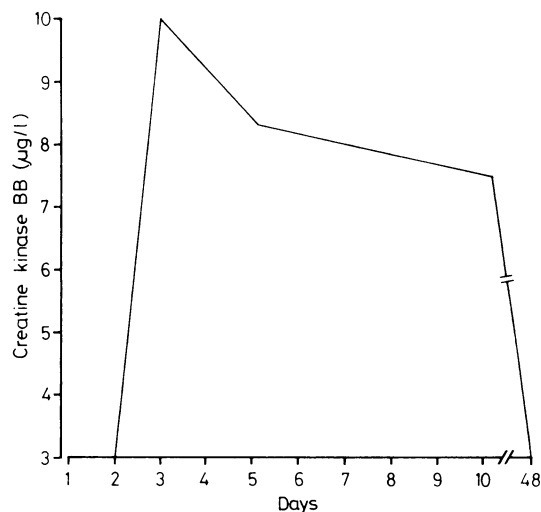
Marathon running is now extremely popular, and the large, publicised national events attract fit, well-acclimatised competitors. Medical directors dissuade ill-prepared or unhealthy people from competing and provide adequate ambulance and medical services during races. None the less, the body's complex physiology is tested by the arduous exertion associated with marathon running, and ill effects may be experienced, even by young, healthy trained competitors. We report here the onset of a cerebrovascular accident in a 14 year old boy during a 13-mile marathon.

Case report

A 14 year old healthy schoolboy, who participated regularly in sporting activities, had trained specifically for a 13-mile run through the streets of Dublin. After 10 miles he felt unwell, developed heaviness in his right leg, and after a further five strides collapsed with right-sided weakness.

On arrival at hospital he was alert but dysphasic with right hemiplegia. He was transferred to our neurosurgical unit, where a computed tomogram of the brain showed several low-density areas in the territory of the left middle cerebral artery, suggesting ischaemia or early infarction. Bilateral carotid angiography after 48 hours showed delayed filling and incomplete opacification of several ascending frontoparietal branches of the left middle cerebral artery. The cervical carotid arteries were smooth and of normal calibre. Conventional and two-dimensional echocardiography showed normal heart and valves. Results of haematological investigations, including clotting times, platelet count, and studies of platelet function, were normal. Blood viscosity, estimated from the packed cell volume and serum globulin and fibrinogen concentrations, was normal. The cerebrospinal fluid contained 11 lymphocytes. The creatine kinase BB level, measured by radioimmunoassay, was raised on days 3, 5, and 10 (figure).

He was treated with bed rest, steroids, and low-molecular-weight intravenous fluids. After five days all neurological signs had improved, and he was discharged at 12 days. There were no abnormalities at six weeks. There was no evidence of a collagen disorder and no history of migraine.



Serum creatine kinase BB measured by radioimmunoassay on days 2, 3, 5, 10, and 48. (Normal range 0-3 µg/l.)

Comment

Neurological complications may be associated with excessive dehydration and an altered packed cell volume. Marathon runners are advised to take adequate fluid replacement during races. It may be important that our patient did not do this. None of his haematological variables indicated dehydration, but most were measured four hours after the race, after oral intake of fluid. The major causes of "collapse" in the 1982 London marathon was dehydration, although most runners who retired did so because of cramp or fatigue.¹ While the physiology of red-cell deformability and serum viscosity during prolonged exertion is complex, a relation exists between increased blood viscosity, decreased cerebral blood flow, and cerebral infarction.² Changes in viscosity together with cerebral hypoxia not measurable on routine screening and influenced by inadequate fluid replacement may have contributed to this athlete's temporary neurological complication.

Interestingly, serum creatine kinase BB measured by radioimmunoassay, was abnormal in this patient (figure). Brain-type creatine kinase BB is present in astrocytes and, at a lower concentration, in other tissues. In cases of proved macroscopic brain injury very high enzyme levels may be detected within hours.³ Here, highest levels were observed after 48 hours, possibly when hypoxic cells developed structural changes due to continuing ischaemia. Previously we found transient abnormalities of creatine kinase BB and MB in marathon runners without neurological deficits.⁴ In this instance creatine kinase MB was normal while the BB isoenzyme remained high and then fell, correlating with clinical improvement.

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¹ Pedoe DST. Marathon running and creatine kinase levels. *Lancet* 1982;iii:154.

² Thomas DJ. Whole blood viscosity and cerebral blood flow. *Stroke* 1982;13:285-7.

³ Phillips JP, Jones HM, Hitchcock R, Adams N, Thompson RJ. Radioimmunoassay of serum creatine kinase BB as an index of brain damage after head injury. *Br Med J* 1980;281:777-82.

⁴ Phillips J, Horner B, Ohman M, Horgan J. Increased brain-type creatine phosphokinase in marathon runners. *Lancet* 1982;ii:1310.

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Outpatient paediatric fibreoptic proctosigmoidoscopy: possible and useful

The role of colonoscopy in diagnosis and follow-up of children with chronic inflammatory bowel disease is becoming recognised.¹⁻⁴ In adult practice flexible fibreoptic sigmoidoscopy without sedation and with minimal bowel preparation is a safe and useful investigation.⁵ In assessing adult inflammatory bowel disease conventional rigid proctosigmoidoscopy and rectal biopsy are normally the first-line procedures. In most paediatric centres, however, rigid proctosigmoidoscopy and rectal biopsy are performed only under general anaesthesia and are regarded as major investigations, rarely undertaken. We therefore decided to see if limited fibreoptic proctosigmoidoscopy using a small-diameter colonoscope would be acceptable to children in a paediatric outpatient clinic and to evaluate its role in the diagnosis and follow-up of children with inflammatory bowel disease.

Patients, methods, and results

Fibreoptic proctosigmoidoscopy was attempted in 21 children (13 boys and eight girls, age range 4-15 years, mean age 11) attending the paediatric inflammatory bowel disease clinic at St Bartholomew's Hospital between February and May 1982. Fibreoptic proctosigmoidoscopy was added without formality to the routine outpatient physical examination. No prior bowel preparation was given and no sedation was used before, during, or after the procedure. A 1 cm diameter very flexible paediatric colonoscope with tip designed for comfortable insertion (Olympus PCF) was selected. Patients were examined in the left lateral position. Only limited examination was attempted. Biopsy samples for histological assessment were normally taken at 10 cm intervals, at least one biopsy specimen being taken from the rectum during each examination.

The instrument was successfully inserted to between 20 and 30 cm in 20 of the 21 patients. The time for the whole procedure including taking biopsy specimens and teaching was 2-10 minutes (mean 5.6 minutes). One examination was impossible because of impaction of solid faeces. Fifteen of the children felt no discomfort and the remaining five tolerated the procedure, though finding it initially uncomfortable.

In four patients the tissue samples taken were considered inadequate for histological assessment, mainly because of their small size. Nine children showed histological abnormalities, including three in whom the mucosa appeared normal endoscopically. In six patients the results of the examination were thought to have aided clinical management. Five of the patients

subsequently underwent total colonoscopy with bowel preparation, in each case giving results compatible with those obtained during the limited examination.

Comment

This small study shows that fibreoptic proctosigmoidoscopy can be quickly performed in a routine paediatric outpatient clinic and is acceptable to children. It allows the paediatric gastroenterologist to follow the standard practice in adult clinics of rapid inspection of the rectal mucosa and the taking of a rectal biopsy specimen. The paediatric colonoscope is smaller in diameter than an examining finger and is considerably more comfortable than the rigid metal instrument. The colonoscope and its light source can be conveniently mounted on a standard metal dressing trolley (figure). The "teaching attachment" side arm allowed some children and parents to appreciate the nature of the clinical problem by watching during the examination, as well as being convenient for discussion with students or medical colleagues. Photographic documentation is easy either for teaching or so as to affix an instant (Polaroid) photograph to the case record.

The only drawback of the paediatric colonoscope used for this study was the small-size forceps biopsy specimens obtained. The importance of taking biopsy samples is considerable; thus multiple samples should be taken in each case.

Though fibreoptic proctosigmoidoscopy gave useful information in these cases, it cannot supplant the additional information given in some cases by total colonoscopy. The occurrence of rectal sparing in Crohn's disease means that a very limited examination with normal biopsy appearances might be misleading, though in our experience there is frequently minor abnormality in the sigmoid colon which is visible on limited examination. Small size is offset by the taking of accurate "target" specimens, which give a high percentage of successful histological diagnosis of Crohn's disease. Whereas total colonoscopy requires considerable experience which may not be available in every paediatric centre, limited examination is very easy to perform and requires little training. Our study convinced us that paediatric fibreoptic proctosigmoidoscopy without sedation or bowel preparation is practicable, well tolerated, and a useful investigation in diagnosis and management of children with possible inflammatory bowel disease.

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¹ Liebman WM. Fiberoptic endoscopy of the gastrointestinal tract in infants and children. *Am J Gastroenterol* 1977;68:452-5.

² Chong SKF, Bartram CB, Campbell CA, Williams CB, Blackshaw AJ, Walker-Smith JA. Chronic inflammatory bowel disease in childhood. *Br Med J* 1982;284:101-4.

³ Rodesch P, Cadranet S, Peeters JP, Cremer N, Cremer M. Colonic endoscopy in children. *Acta Paediatr Belg* 1976;29:181-4.

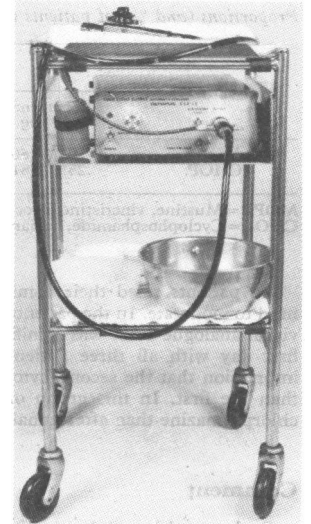
⁴ Williams CB, Laage NJ, Campbell CA, et al. Total colonoscopy in children. *Arch Dis Child* 1982;57:49-53.

⁵ Vellacott KD, Hardcastle JD. An evaluation of flexible fibreoptic sigmoidoscopy. *Br Med J* 1981;283:1583-5.

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Modified trolley for outpatient fibreoptic proctosigmoidoscopy.