SHORT REPORTS

Early unrestricted oral fluids after major gastrointestinal operations

Abdominal operations are followed by a period of impaired gastrointestinal motility[1-3] during which patients are allowed to drink little.[4] Conventionally, oral fluids are started after the return of gut motility.[1-4] Commonly patients are given small volumes of fluid to drink each hour, the volumes being increased progressively over two to three days until the patients are drinking normally.[1] We have investigated the effect of allowing early unrestricted consumption of fluids after major gastrointestinal operations.

Patients, methods, and results

Seventy seven compliant patients who had undergone major gastrointestinal operations (resection of gut, or anastomosis, or both) were studied. After the return of bowel sounds patients were randomly allocated to have unrestricted fluids by mouth (n=38) or to conventional management (n=39). There were no differences between the groups in age, sex, or operations performed.

Patients having unrestricted fluids were given jugs containing 750 ml of water twice daily by nurses; they were instructed to drink freely and to regulate their own intake. The control group were given oral fluids by nurses as follows: 50 ml/hour on day 1, 100 ml/hour on day 2, and unrestricted fluids on day 3. In all other respects the postoperative management of the two groups was identical, and nasogastric tubes were removed before entry to the study. All patients received 1 litre saline (0·9% wt/vol) and 2 litres dextrose (5% wt/vol) intravenously daily,[4] which was discontinued when the oral intake equalled 1·2 litres. Oral fluid intake was recorded by nurses. The number of visits by nurses to give drinks to patients was calculated by counting the entries on the charts. Statistical analysis was by Fisher's exact probability test.

Two patients in each group developed intestinal obstruction and were withdrawn from the study. There were no differences between the groups in the incidence of nausea, vomiting, abdominal distension, or length of hospital stay.

Patients allowed free access to fluids drank more than the controls (table). Patients allowed free access to fluids had fewer visits from nurses, the mean number of visits (SD) being 2·4 (1·4) on day 1, 2·4 (0·77) on day 2, and 2·8 (0·74) on day 3; in the control group the figures were 8·4 (2·3), 7·8 (1·9), and 2·6 (1·2), respectively.

Amount of fluid consumed postoperatively

	Group treated conservatively (n=39)			Group allowed free fluids (n=38)		
	Mean (SD) volume (l)	Range	No (%) consuming ≥1·2 l day	Mean (SD) volume (l)	Range	No (%) consuming ≥1·2 l day
Day 1	0·42 (0·12)	0·25-0·7	0	1·15 (0·4)	0·46-1·82	19 (50)*
Day 2	0·77 (0·18)	0·4-1·25	2 (5)	1·33 (0·3)	0·7-2·1	26 (68)*
Day 3	1·43 (0·34)	0·5-2·07	32 (82)	1·6 (0·27)	0·95-2·0	33 (87)

*Difference between groups, $p<0·001$.

Comment

The four patients who developed intestinal obstruction had bypass operations for obstructive jaundice caused by pancreatic malignancies. Such patients have an increased incidence of postoperative complications,[5] and it is unlikely that the intestinal obstruction was related to the fluid regimen.

None of the patients experienced difficulty in managing the regimen. In a few patients the volumes of fluid consumed were disproportionate to the number of visits by nurses. Among those with free access to fluids this was caused by upsetting the jugs and in the control group by patients refusing the drink when offered. During the first two days of oral intake the mean number of visits solely to give fluids in each 24 hours was 2·5 for those with free access to fluids and roughly eight in the control group. This represents a potential saving in nurses' time and may lead to greater efficiency. In the control group the number of visits by nurses ranged widely despite the protocol of giving drinks hourly, probably because of the large variations in the work of nursing staff in surgical wards.

Postoperatively gastric contractions are diminished or absent, but propulsive activity in the small bowel continues unabated.[1-3] The return of bowel sounds signals the return of gastric contractions and emptying.[1-3] After this time selected patients can safely manage their own fluid intake. Patients do better clinically, and the saving in nurses' time is an additional potential benefit.

We thank Mr J C Postlethwaite, Barnet General Hospital, for allowing his patients to be included in this study, and the nurses of Lister and Nightingale wards, Barnet General Hospital, for their cooperation.

1 Wells C, Tinkler L, Rawlinson K, Jones H, Saunders J. Postoperative gastrointestinal motility. *Lancet* 1964;i:4-10.
2 Wilson JP. Postoperative motility of the large intestine in man. *Gut* 1975;16:689-92.
3 Anonymous. Postoperative ileus. *Lancet* 1978;ii:1186-7.
4 Randall HT. Enteral nutrition. In: Dudrick SJ, Baue AE, Eiseman B, MacLean LD, Rowe MI, Sheldon GF, (Committee on pre- and postoperative care), eds. *American College of Surgeons: manual of pre- and postoperative care*. 3rd ed. Philadelphia: W B Saunders, 1983:69-70.
5 Dixon JM, Armstrong CP, Duffy SW, Davies GC. Factors affecting mortality and morbidity after surgery for obstructive jaundice: a review of 373 patients. *Gut* 1983;24:845-52.

(*Accepted 25 February 1988*)

Barnet General Hospital, London EN5 3DJ
G G P BROWNING, FRCSED, senior surgical registrar
M J NOTARAS, FRCS, consultant surgeon

Correspondence to: Mr G G P Browning, 23 Braehead Avenue, Edinburgh EH4 6QN.

Dantrolene sodium for treatment of carbon monoxide poisoning

The efficacy of dantrolene in the treatment of muscle rigidity and hyperpyrexia associated with malignant hyperthermia and neuroleptic malignant syndrome has been shown.[1] We describe a case of carbon monoxide poisoning in which fever and hypertonicity led us to treat the patient supportively with dantrolene sodium.

Case report

A woman aged 28 was admitted elsewhere having been asphyxiated by a defective ventilating system. She was comatose and had generalised extensor rigidity. The carboxyhaemoglobin concentration three hours after her rescue was 9%, and the estimated value at the time of rescue (calculated from the Clark nomogram[2]) was about 18%. Arterial pH was 7·3, arterial oxygen pressure 11·8 kPa, and arterial carbon dioxide pressure 4·5 kPa. She was treated with 100% oxygen for 12 hours, and given 250 ml of 20% mannitol and 25 mg prednisolone intravenously twice daily.

Because her condition remained unstable she was transferred to the intensive care unit at this hospital three days later. She was unconscious but opened her eyes to painful stimuli, made incomprehensible sounds, and showed extensor motor responses. Her Glasgow coma score was 6. She also had symmetrical hyperreflexia, increased jaw jerk, and corneomandibular reflexes. She was sweating profusely and hyperventilating; there was sinus tachycardia, and her temperature was 37·5°C. Creatine phosphokinase activity was 1368 U/l, arterial pH 7·4, arterial oxygen pressure 21·5 kPa, and carbon dioxide pressure 4·6 kPa. An electroencephalogram showed bilateral synchronous slowed activity, and visual evoked potentials slowness of conduction. Later that day her temperature rose to 38·5°C and she became more excitable on touch. Muscle tone was considerably increased and she had boardlike rigidity of the abdominal muscles.

Dantrolene sodium 60 mg was given twice daily by nasogastric tube, resulting in reduction of the hypertonicity and the return of her temperature to normal (36·6°C). She continued to improve, opening her eyes and sticking out her tongue on request, and the creatinine phosphokinase concentration fell to 279 U/l. Four days later she again became comatose with extreme muscle rigidity, and her temperature rose to 39·1°C, so over two days her dose of dantrolene was gradually increased to 60 mg four times daily. Her condition improved and stabilised. Dantrolene 60 mg three times daily was given by mouth for two further weeks and then tapered off.

She made a slow recovery and subsequent electroencephalograms and visual evoked potentials showed progressive improvement. After a month she was discharged to a rehabilitation centre and went home well after a further month.

Comment

The treatment of carbon monoxide poisoning consists of removing the patient from exposure to the gas and giving 100% oxygen. This results in accelerated replacement of carbon monoxide and relief of tissue hypoxia. Treatment with hyperbaric oxygen should be considered.[3,4] The clinical effects are those of tissue hypoxia, so any symptom of increased oxygen demand—particularly hyperthermia—must be rigorously treated because of the increases in cerebral and cardiac metabolic demands. Controlled hypothermia has been recommended, but there are few reports of its use.[5] The similarities between the symptoms of malignant hyperthermia,

neuroleptic malignant syndrome, and this presentation of carbon monoxide poisoning led us to treat the patient with dantrolene,[1] with good effect. Thermogenesis was probably due to skeletal muscle rigidity, and the relaxant action of dantrolene produced a fall in temperature as a secondary benefit.

Our patient may have recovered spontaneously, but the responses to dantrolene merit notice. Further investigation is needed to see whether dantrolene is effective in treating severe cases of carbon monoxide poisoning. Moreover, its use in other hypermetabolic states could be beneficial.

1 Guze BH, Baxter LR. Neuroleptic malignant syndrome. *N Engl J Med* 1985;313:163-6.
2 Clark CJ, Campbell D, Reid WH. Blood carboxyhaemoglobin and cyanide levels in fire survivors. *Lancet* 1981;i:1332-5.
3 Dolan MC. Carbon monoxide poisoning. *Can Med Assoc J* 1985;133:392-9.
4 Myers RA, Linberg SE, Cowley RA. Carbon monoxide poisoning: the injury and its treatment. *Journal of the American College of Emergency Physicians* 1979;8:479-84.
5 Boutros AR, Hoyt JL. Management of carbon monoxide poisoning in the absence of hyperbaric oxygenation chamber. *Crit Care Med* 1976;4:144.

(*Accepted 8 February 1988*)

Department of Neurology, St Elisabeth Hospital, Postbox 90151, 5000 LC Tilburg, The Netherlands
J B M TEN HOLTER, MD, registrar
R L L A M SCHELLENS, MD, senior neurologist

Correspondence to: Dr Ten Holter.

Photosensitive epilepsy in children who set fires

The flickering light of television is recognised as epileptogenic. Some children with photosensitive epilepsy find viewing television compulsively attractive[1] and even induce seizures by viewing it (C D Binnie, Compliance in epilepsy, Salzburg, 1987). Flames may be a self induced stimulus in those children with photosensitive epilepsy who repeatedly set light to things (fire setting).

Case reports

CASE 1

A man aged 31 had his first attack of epilepsy at the age of 7, when he saw "hundreds of tiny silver dots, then blackness." Minutes later his sight returned. He had innumerable similar attacks for eight years. As his vision cleared he was overtaken by anger and violence commonly followed: he punched bystanders, broke windows, and burned sheds and hedgerows. Recognising the sequence, he tried to limit the damage, running off alone whenever he saw the silver dots.

His behaviour antagonised his parents. He was seen in child guidance clinics, put on probation, taken into care, placed in children's homes, and sent to approved schools, but his disruptive behaviour continued, often after viewing television. At age 13 his epilepsy was diagnosed. Electroencephalography without photic stimulation showed diffuse abnormalities, arrhythmia and sharp and slow components that were maximal in the posterior left temporal region. Although prescribed phenytoin, he palmed the tablets. His attacks stopped without treatment at age 15, and electroencephalographs were subsequently normal.

CASE 2

From babyhood the 6 year old daughter of the man in case 1 had thrown paper on to fires and watched it burn. She set fire to her toys, singed her rabbit's fur, posted lighted matches through the letterbox, and fused an electric circuit with burning papers. She watched television from a distance of 50 cm with maximum colour and contrast. Her mother noticed repeated short episodes, while she was viewing television when she suddenly stared at the wall, white faced and withdrawn. Tears or pranks followed; once she set fire to the sofa. Epilepsy was suspected, and while awaiting neurological investigation she stole a cigarette lighter. Her father, fearing a fire, caned her, and she was admitted to hospital. Compulsory care proceedings were started. On transfer to another hospital an electroencephalogram showed typical photoconvulsant responses during photic stimulation at flash rates above 10 cycles/second, particularly with red and orange light; the recording was otherwise normal. She returned home and complied with instructions to stay three metres from the television screen.[2] Her behaviour was exemplary. Three months later a 24 hour ambulatory electroencephalogram showed that abnormalities occurred immediately when she viewed television from her former position.

After six months of normal behaviour her parents reported renewed moodiness, then discovered caches of spent matches. Meanwhile her teachers, misled by her episodic pallor and abstraction, suspected parental cruelty. Electroencephalography showed sensitivity to fluorescent light, to which she was exposed at school, and to patterns. She was admitted to hospital for drug treatment under electroencephalographic control. Photic spikes lessened with phenytoin, returned with carbamazepine, then disappeared with sodium valproate 200 mg twice daily. She continued to take this drug, and her behaviour was normal during the next six months.

Comment

Fire setting and photogenic epilepsy might coexist by chance but were closely linked in these two cases. The aggressive conduct of the patient in case 1 followed his seizures, and he continued to set fires until he outgrew his epilepsy. His daughter (case 2) behaved normally after avoiding the photic stimulus of close viewing of the television. She began to set fires again in response to photic stimulation by fluorescent lights. Both her fire setting and electroencephalographic abnormalities resolved on treatment with sodium valproate.

Fire setting inevitably causes family disturbances, and blaming the child or the family may lead to inappropriate attempts at containment instead of treatment. Sensitivity to television as a cause of epilepsy is easily missed because the family watches the screen, not each other. Flames lit during preictal compulsion or postictal confusion may kindle further epilepsy.[3] Those who set fires, particularly younger, solitary children,[4] should undergo electroencephalography.

We thank Professor V Dubowitz for allowing publication of case 2; Oxford Medical Systems for the loan of an ambulatory electroencephalographic recorder; and Elizabeth Wallace of the British Epilepsy Association for helpful advice.

1 Jeavons PM, Harding GFA. *Photosensitive epilepsy*. London: William Heinemann, 1975.
2 Anonymous. Television-induced epilepsy and its prevention. [Editorial.] *Br Med J* 1978;i:1301-2.
3 Reynolds EH. Biological factors in psychological disorders associated with epilepsy. In: Reynolds EH, Trimble MR, eds. *Epilepsy and psychiatry*. Edinburgh: Churchill Livingstone, 1981:264-90.
4 Jacobson RR. Child fire setters: a clinical investigation. *Journal of Child Psychology and Psychiatry and Allied Disciplines* 1985;26:759-68.

(*Accepted 2 March 1988*)

Surgery, Clifton Hampden, Oxfordshire OX14 3EJ
ELIZABETH A MEINHARD, MD, FRCPATH, general practitioner

Hammersmith Hospital, London W12 0HS
ROWENA OOZEER, chief technician in electroencephalography
DUNCAN CAMERON, MRCP, DCH, registrar in paediatrics

Correspondence to: Dr Meinhard.

An offer of rheumatology training: failure to influence clinic referrals

It has been suggested that teaching of small groups of general practitioners by consultants in the general practitioners' surgeries may be a more useful approach than lectures to large audiences in hospitals.[1] Such teaching can influence the behaviour of general practitioners volunteering for educational sessions,[2] but education does not necessarily decrease the demand for specialist opinions.[3] This study reports the responses of general practitioners referring patients with soft tissue lesions of the shoulder and elbow when they were offered individual clinical tutorials by a consultant.

Patients, methods, and results

From 1 August 1984 until 31 July 1987 every general practitioner who referred a patient with an uncomplicated soft tissue lesion of the shoulder or elbow to a rheumatology clinic was sent an offer of individual teaching by a consultant with the relevant patient's clinic letter. This offer suggested that should the general practitioner have any further patients with shoulder or elbow lesions he or she should contact the consultant, who would come to the surgery and, with the general practitioner, examine, diagnose, and, where appropriate, inject the patient's soft tissue lesion. Further letters were sent to the general practitioners after each subsequent referral. These educational offers had no implications with respect to section 63 or domiciliary visit payment. In all 120 offers were made (19 in relation to elbow and 101 in relation to shoulder lesions) to a total of 41 principals and four trainees. Twenty two doctors received two or more offers, and six sessions were arranged, with nine general practitioners receiving teaching.

A lunchtime meeting entitled "shoulder pain—the GP's role," accounting for half of a section 63 session, was organised two and a half years after the start of the study at this hospital, and all general practitioners in Leeds were invited to attend. The potential effects of the educational exercise on clinic load were examined by observing how many referrals in the last six months of the study might have been avoided if general practitioners who had received two or more educational offers had responded to them and managed the patient themselves.