glucose concentration, the glomerular filtration rate, and the capacity of the renal tubules to reabsorb glucose.² Glycosuria develops in the diabetic because raised blood glucose concentration leads to quantities of glucose in the glomerular filtrate that cannot be reabsorbed. When the ureter has been diverted into the bowel an additional factor operates. The enterocytes tend to reabsorb glucose from urine traversing the loop in proportion to the urinary glucose concentration, the length of time urine remains in contact with the absorptive surface, and the area of intestinal mucosa exposed to urine—the same factors that govern the trading of bicarbonate for chloride ions in the genesis of hyperchloraemic acidosis.

These variable factors may help to explain why glucose reabsorption is complete in some ileal loops but not in others. Furthermore, ileal glucose transport that is carrier-mediated and sodium-dependent³ is significantly enhanced in diabetic patients⁴ and by high luminal glucose concentrations in the rat.⁵

I thank Dr A R Harrison for permission to report this case.

¹ McGouran RCM. Glucose reabsorption from ileal loops. *Br Med J* 1977; ii:932.

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³ Schultz SG, Fuisz RE, Curran PF. Amino acid and sugar transport in rabbit ileum. *J Gen Physiol* 1966;49:849.

⁴ Vinnik IE, Kern F, Sussman K. The effect of diabetes mellitus and insulin on glucose absorption by the small intestine in man. *J Lab Clin Med* 1965;66:131-6.

⁵ Sund RB. Glucose and cation transport in rat jejunum, ileum and colon in vivo. *Acta Pharmacol Toxicol (Kbh)* 1978;42:117-24.

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Strangulated hernias through Tenckhoff cannula sites

Continuous ambulatory peritoneal dialysis has become a well-established method of treating end-stage renal failure since 1978.¹ Though the commonest gastrointestinal problem is the development of peritonitis, abdominal hernias are a well-recognised complication.² We report here two cases of patients on continuous ambulatory peritoneal dialysis who developed strangulated hernias through Tenckhoff cannula sites, a complication we believe not to have been recorded.

Case reports

Case 1—A 64-year-old woman with chronic glomerulonephritis was started on continuous ambulatory peritoneal dialysis on 29 July 1980 and discharged on 30 August 1980 with dialysis running satisfactorily. She was admitted as an emergency on 22 November 1980 with a "blocked catheter," 3.2 l of dialysate having run in and only 300 ml drained; otherwise she was asymptomatic. On examination there was a small, firm, tender subumbilical swelling at the site of peritoneal entry of the Tenckhoff cannula. Bowel sounds were normal. Her catheter was flushed with heparinised saline and urokinase inserted but to no avail. Two days later she started vomiting and developed abdominal distension. By this time the subumbilical mass was tender and erythematous and bowel sounds were obstructive. Abdominal films showed dilated small-bowel loops with fluid levels. Laparotomy was carried out that day, and the small bowel and the cannula were found to be herniating through the peritoneal entry site. Bowel and the intraperitoneal portion of the cannula were lying in the proximal end of the subcutaneous catheter tunnel. The small bowel was necrotic. The hernia was reduced, small-bowel resection carried out, and the catheter resited in the pelvis. Her postoperative course was uneventful and she was discharged on 9 December 1980. She was, however, readmitted four days later with a candida peritoneal infection but failed to respond to treatment. She also developed a chest infection, deteriorated despite treatment, and died on 27 December 1980.

Case 2—A 61-year-old man was started on continuous ambulatory peritoneal dialysis on 14 April 1980 after having rejected a transplant and

being found incompatible with haemodialysis. After multiple peritoneal infections and having his cannula changed twice he was admitted as an emergency on 26 November 1981 with an eight-hour history of lower abdominal pain, nausea, and vomiting. On examination the abdomen was soft and not distended. At the lower end of a small infraumbilical scar was a tense swelling with a questionable cough impulse. Bowel sounds were normal. Abdominal films showed a few dilated small-bowel loops with fluid levels. At laparotomy a necrotic loop of small bowel was found to have herniated through the peritoneum 2 cm below the entry site of his present cannula, presumably through the entry site of a previous one. The hernia was reduced, small-bowel resection was carried out, and the cannula tip resited in the pelvis. His postoperative course was uneventful. He was discharged on 11 December 1981 and has remained well since.

Comment

Though the most common gastrointestinal complication in continuous ambulatory peritoneal dialysis is peritonitis, an appreciable number of patients develop abdominal hernias, of which the most common seem to be incisional.² The two patients discussed here had hernias through Tenckhoff cannula entry sites. The hernial orifices were small and thus not surprisingly strangulation of bowel was a feature in both cases. The only other strangulated hernias reported in patients undergoing continuous ambulatory peritoneal dialysis were umbilical³ and epigastric.²

In our experience of 28 patients undergoing a combined total of 237 months of dialysis, four have developed abdominal hernias—one inguinal, two incisional (in the same patient), and the two strangulated hernias reported here. Both our patients had had their cannulas implanted using the standard introducer for penetrating the peritoneum. We suggest that a non-absorbable purse-string suture should be placed around the cannula on insertion and that an attempt should be made to suture the peritoneum when a cannula is removed.

Though the signs and symptoms may be minimal obstructed hernia should be considered as a cause of abdominal pain in patients undergoing continuous ambulatory peritoneal dialysis, especially if a tender lump is present in a scar.

¹ Popovich RP, Moncrief JW, Nolph KD, Ghods AJ, Twardowski ZT, Pyle WK. Continuous ambulatory peritoneal dialysis. *Ann Intern Med* 1978;88:449-56.

² Khanna R, Oreopoulos DG, Dombros N, et al. Continuous ambulatory peritoneal dialysis after three years: still a promising treatment. *Peritoneal Dialysis Bulletin* 1981;1:24.

³ Chan MK, Bailod RA, Tanner A, et al. Abdominal hernias in patients receiving continuous ambulatory peritoneal dialysis. *Br Med J* 1981; 283:826.

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Neonatal convulsions caused by withdrawal from maternal clomipramine

Clomipramine is a tricyclic antidepressant particularly useful in treating depression with obsessive or phobic features. Although its use is not recommended in the first trimester, there has been no contraindication to its use in later pregnancy. We describe two infants who developed convulsions due to withdrawal from maternal clomipramine, the second of whom was treated with reducing doses of clomipramine.

Case reports

Case 1—A 22-year-old primigravida who had a depressive illness with obsessional features was treated with clomipramine for the last seven weeks of pregnancy. A male infant was born by spontaneous vertex delivery at term weighing 3420 g with Apgar scores of 7 and 8 at one and five minutes respectively. He had a convulsion at eight hours of age, and intermittent convulsions persisted until he was 53 hours of age despite treatment with parenteral phenobarbitone and paraldehyde. He remained hypertonic and

jittery with ankle clonus until the 11th day of life. Anticonvulsant treatment was discontinued on day 14 and no abnormality was present when he was discharged two days later. Biochemical and infective causes for the fits were excluded. Convulsions secondary to withdrawal of clomipramine was diagnosed, supported by estimations¹ of concentrations of clomipramine and its metabolite desmethylclomipramine in his serum (figure).

Serum concentrations of clomipramine and desmethylclomipramine in cases 1 (top) and 2 (bottom)

Case 2—A 38-year-old para 0+1 was treated with clomipramine and flurazepam throughout pregnancy. A male infant weighing 2360 g was delivered by Haig Ferguson forceps at a gestational age of 33 weeks and had Apgar scores of 9 and 9 at one and five minutes respectively. Convulsions started at seven hours of age and continued in the form of myoclonic jerks despite parenteral phenobarbitone. After our experience with the first case

and after exclusion of other causes of convulsions treatment with intravenous clomipramine was started at 24 hours of age. An initial bolus of 0.4 mg over two hours resulted in complete cessation of convulsions for 11 hours. Twitching movements of all limbs occurred at this time and were controlled by a further bolus of 0.5 mg over two hours, followed by a continuous infusion of clomipramine in reducing doses and then oral treatment on day 12. Convulsions were controlled but he remained jittery. Clomipramine was discontinued on the 17th day of life with no ill effect.

Comment

Both infants presented with convulsions on the first day of life. In the first case the timing of the convulsions coincided with the initial steep decline in concentrations of clomipramine and its metabolite desmethylclomipramine in the infant's serum. In the second case convulsions were controlled by administration of clomipramine, thus permitting a more gradual decrease in serum concentrations of clomipramine and its metabolite (see figure). In both cases neurological signs were present until the concentration of clomipramine was less than 10 µg/l. Musa and Smith² described an infant who became jittery on the second day of life after withdrawal from maternal clomipramine. Plasma concentrations of clomipramine in their case were less than 20 µg/l on the first and third days of life. Our infants had much higher serum clomipramine concentrations, while the combined clomipramine and desmethylclomipramine concentrations in the mothers after delivery were 610 µg/l at two and a half days in case 1 and 549 µg/l at 24 hours in case 2. This is greater than the suggested therapeutic range of 200–500 µg/l.

Our experiences of neonatal convulsions after maternal treatment with clomipramine suggest that this drug is best avoided during pregnancy. If tricyclic antidepressants must be prescribed we suggest that the maternal dose is regulated by monitoring serum concentrations. Should withdrawal convulsions occur in the neonatal period they may be controlled by administration of clomipramine.

¹ Braithwaite R. Measurement of antidepressant drugs. *Proceedings of the Analytical Division of the Chemical Society* 1979;16:69-72.

² Musa BA, Smith CS. Neonatal effects of maternal clomipramine therapy. *Arch Dis Child* 1979;54:405.

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MEDICINES APPROPRIATED TO THE HEART—These are they which are generally given under the notion of Cordials; take them under that name here. The heart is the seat of the vital spirit, the fountain of life, the original of infused heat, and of the natural affections of man. So then these two things are proper to the heart. 1. By its heat to cherish life throughout the body. 2. To add vigour to the affections. And if these be proper to the heart, you will easily grant me, that it is the property of cordials to administer to the heart in these particulars. Of Cordials, some cheer the mind, some strengthen the heart, and refresh the spirits thereof, being decayed. Those which cheer the mind, are not one and the same; for as the heart is variously disturbed, either by anger, love, fear, hatred, sadness, &c. So such things as flatter lovers or appease the angry, or comfort the fearful, or please the hateful, may well be called cordials: for the heart, seeing it is placed in the middle between the brain and the liver, is wrought upon by reason, as well as by digestion, yet these, because they are not medicines, are beside my present scope. And although it is true, that mirth, love, &c. are actions, or motions of the mind, not of the body; yet many have been induced to think such affections may be wrought in the body by medicines.

The heart is chiefly afflicted by too much heat, by poison, and by stinking vapours, and these are remedied by the second sort of cordials, and indeed chiefly belong to our present scope. According to these three afflictions, viz 1. *Excessive heat*. 2. *Poison*. 3. *Melancholy vapours*. Are three kinds of remedies which succour the afflicted heart. Such as

1. *By their cooling nature mitigate the heat of fevers*. 2. *Resist poison*. 3. *Cherish the vital spirits when they languish*. All these are called Cordials. 1. Such as cool the heart in fevers, yet is not every thing that cooleth cordial, for lead is colder than gold, yet is not lead cordial as gold is, some hold it cordial by a hidden quality, others by reason. 2. Such as resist poison; there is a twofold resisting of poison. 1. *By an antipathy between the medicine and poison*. 2. *By a sympathy between the medicine and the heart*. Of the first we shall speak anon, in a chapter by itself. The latter belongs to this chapter, and they are such medicines, whose nature is to strengthen the heart, and fortify it against the poison, as Rue, Angelica, &c. For as the operation of the former is upon the poison, which afflicteth the heart, so the operation of the latter is upon the heart afflicted by the poison. To this class may be referred all such medicines as strengthen the heart either by astral influence, or by likeness of substance, if there be such a likeness in medicines, for a Bullock's heart is of like substance to man's, yet I question whether it be cordial or not. 3. And lastly, Such as refresh the spirits, and make them lively and active, both because they are appropriated to the office, and also because they drive stinking and melancholy vapours from the heart, for as the animal spirit be refreshed by fragrant smells, and the natural spirits by spices, so are the vital spirits refreshed by all such medicines as keep back melancholy vapours from the heart, as Borage, Bugloss, Rosemary, Citron Pills, the compositions of them, and many others, which this treatise will amply furnish you with. (Nicholas Culpeper (1616-54) *The Complete Herbal*, 1850.)