which were nitrazepam 30-40 mg (patient 3), digoxin, paracetamol, and aspirin. Eight patients were unconscious (grade 2, 3, or 4¹) on arrival, and on another five occasions consciousness was lost after admission. Patients 1, 2, and 3 had severe respiratory depression and required endotracheal intubation and artificial ventilation for 3, 4, and 18 hours respectively. Patient 4 breathed satisfactorily after intubation. In a further six an oropharyngeal airway was used. Bronchoscopy was performed in two patients (1, 6a) who had apparently aspirated vomit before admission. Three patients (1, 2, 3) were hypothermic and hypotensive (rectal temperatures 31.5-33.5°C, systolic blood pressures 50-70 mm Hg). Another three had systolic blood pressures of only 85-90 mm Hg. The pulse rates on admission ranged from 68-150/min (mean 99). Electrocardiograms in eight unconscious patients showed no unexpected features. The muscle tone and limb reflexes were reduced or absent in deeply unconscious patients. The pupils showed no consistent changes. Bowel sounds were present in all patients. Increased salivation was observed during seven admissions in six patients. Bronchial secretions were increased in patients with chronic bronchitis but appeared normal in the others.

Intensive supportive treatment was given, as for barbiturate poisoning.¹ All patients recovered, although five were unconscious for more than 20 hours and one was in grade 4 coma for 41 hours and unconscious for 92 hours after reaching hospital. Of the six patients who developed pneumonia, four had chronic bronchitis and two had aspirated vomit before admission. Plasma was assayed for alcohol and chlormethiazole by gas-liquid chromatography² and screened for other drugs. The highest plasma chlormethiazole concentration recorded in a conscious patient was 11.5 mg/l. Concentrations in unconscious patients ranged from 7 to 36 mg/l. In 10 cases in which serial concentrations were measured the plasma half-lives ranged from 4 to 15 hours, but in one elderly patient (1) the plasma concentration was about 18 mg/l for 36 hours before falling with a half-life of 17 hours.

Comment

These and other cases^{3,4} show that chlormethiazole can cause severe and sometimes fatal poisoning. Except for increased salivation the clinical features are similar to those of barbiturate overdose,¹ with deep coma, respiratory depression, hypotension, and hypothermia in severe cases. The cause of the increased salivation is not known. Our three hypothermic patients had been lying unconscious for many hours before admission and so were at particular risk of hypothermia. But hypothermia has also been described⁵ after intravenous administration of chlormethiazole, which may have a direct effect on thermoregulatory centres. All our patients survived but the incidence of pneumonia was high. Aspirations of secretions may be more likely in patients unconscious from chlormethiazole overdose who have increased salivation. We did not use charcoal haemoperfusion. Its efficacy in removing chlormethiazole is not known, but it might be helpful in patients with deep, prolonged coma and respiratory complications.

Self-poisoning is common in alcoholics, so the long-term prescription of chlormethiazole for the "treatment" of alcoholism is dangerous⁴ as well as ineffective. The safety of chlormethiazole in elderly patients and in chronic bronchitis is also questionable since it may cause hypothermia and respiratory depression.

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Primary anaerobic peritonitis

Spontaneous peritonitis is a rare condition usually occurring in children and accounting for about 2% of abdominal emergencies in childhood.¹

The incidence of primary peritonitis is increased in patients with ascites due to hepatic or renal disease and primary anaerobic peritonitis may occur in these patients.² There have been no reports of primary anaerobic peritonitis occurring in a patient without ascites. The following case report describes a previously healthy 16-year-old boy who presented with primary anaerobic peritonitis.

Case report

A 16-year-old Negro schoolboy, who had lived all his life in Britain, was admitted to St James's Hospital, Balham, with a 24-hour history of the sudden onset of severe abdominal pain and vomiting. Except for an influenza-like illness one month earlier he had had no relevant medical history. He was dehydrated and toxic, with a pulse rate of 120/min and a temperature of 38.5°C. There was pronounced tenderness, guarding, and rebound tenderness over the whole abdomen and no bowel sounds were heard. The haemoglobin concentration was 11.9 g/dl, the white blood cell count was $27.6 \times 10^9/l$ and the sickle cell test was negative. Serum electrolytes and amylase concentrations were normal, and the serum urea concentration was 15.9 mmol/l (95.4 mg/100 ml). Urine microscopy and subsequent culture showed no evidence of a urinary tract infection.

At laparotomy, performed after resuscitation with intravenous fluids, 650 ml of odourless yellowish pus was found throughout the whole peritoneal cavity. Careful examination of the entire length of the bowel failed to show any abnormalities and the only unusual finding was a slightly enlarged spleen. Peritoneal toilet was performed and the abdomen closed without drainage. An immediate Gram stain performed on a sample of pus showed Gram-negative coccobacilli. A course of gentamicin and penicillin was started and when subsequent culture produced a profuse anaerobic growth metronidazole was added.

The patient made a slow but uneventful recovery. Postoperative liver function tests, upper and lower gastrointestinal barium studies, and an intravenous urogram all gave normal results. Ultrasonography showed a normal liver and enlargement of the spleen by a posterior splenic cyst. The causative organism was eventually identified by the Anaerobic Reference

Clinical features and highest recorded plasma chlormethiazole and alcohol concentrations in 16 cases of chlormethiazole overdose in 13 patients

	Patient No:												
	1	2	3	4	5a	5b	5c	6a	6b	7	8	9	10
Age (years)	84	77	29	22	40			46		35	43	53	70
Sex	F	M	F	M	M			M		M	M	M	M
Stated amount of chlormethiazole*													
Tablets		18	30	25	47	50	35	50		25	40-60	20-40	10-20
Capsules	90								50				
Ingestion-admission interval (hours)	c18	12-15	14	13	2	4	2-4	20	2	c12	1	9	2
Coma grade on admission	4	4	4	4	0	0	0	3	0	3	1	2	2
Deepest coma grade	4	4	4	4	4	2	0	3	2	3	3	2	2
Duration of coma in hospital (hours)	92	34	22	28	29			4	0.5	4	6	6	0.5
Increased salivation	—	+	+	—	+	+	—	+	—	—	—	+	—
Pneumonia	+	+	—	+	—	+	—	+	—	—	—	—	+
Highest plasma chlormethiazole concentration (mg/l)	20.5	36	19	19	16	19	9	(3)†	10.5	12	(10.5)†	15.5	7
Highest plasma alcohol concentration (mg/100 ml)	0	0	0	0	(10)†	100	50	0†	245	190	0†	380	0

*Heminevrin tablets each contain chlormethiazole edisylate 500 mg. Heminevrin capsules contain chlormethiazole base 192 mg in arachis oil.

†Not measured before recovery of consciousness.

Laboratory at Luton as *Fusobacterium necrophorum*, which is an anaerobic gut commensal with a pleomorphic tendency, which normally but not always produces a foul odour.³ This organism differs from *Bacteroides fragilis* in that it is sensitive to penicillin.

Comment

In 1922 McCartney⁴ described primary peritonitis as being caused by either pneumococci or streptococci. Twenty-five years later Gross,⁵ reporting a series of 158 patients with primary peritonitis, found that in only five patients (3%) was the causative organism Gram negative. Twenty years later Scott McDougal¹ found that in 17 (65%) out of 26 patients with primary peritonitis the causative organism was Gram negative. These reports seem to indicate that Gram-negative bacteria are playing an increasingly important part in primary peritonitis.

The route by which bacteria reach the peritoneal cavity is not certain. McCartney⁴ suggested that ascending infection in young girls was carried via the fallopian tubes, but most workers now believe that either haematogenous spread⁵ or transmural migration of bacteria occurs.

Primary peritonitis is only rarely diagnosed before operation and the experience in this case suggests that when a purulent peritonitis is found at laparotomy, without any intra-abdominal cause, a policy of peritoneal toilet and an immediate Gram stain on the pus is advisable. If this shows Gram-negative bacteria then the possibility of an anaerobic infection should be considered and treatment with a suitable antimicrobial drug started.

I thank Mr J S Kirkham for permission to publish this case report, and Dr B C Stratford for this help with the bacteriology.

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Carbamazepine in Morton's neuralgia

Morton¹ reported 16 cases of paroxysmal pain in the region of the fourth metatarsophalangeal joint. On case 6 he commented, "the paroxysms of suffering . . . forcibly reminded me of those cases of severe facial neuralgia . . . seen in the second branch of the fifth pair of nerves." Trigeminal^{2,3} and other neuralgias^{4,5} may be relieved by carbamazepine. I describe two patients with Morton's "metatarsalgia" who seemed to benefit from this drug. In one surgery had been unsuccessful. The other was given a double-blind crossover trial against placebo.

Case reports

Case 1—A 79-year-old woman had had intermittent burning pain and parasthesiae in the dorsal surface of the left fourth toe since 1965, usually at night in bed. It was relieved by pressure over the top of the toe, walking around, stamping the foot on the floor, and pressing the lateral side of the thigh or leg, but not by common analgesics. The middle phalanx of this toe had been excised in 1965 with no relief. The pain was eased by pressure on the site of tenotomy, where there was a positive Tinel's sign. When she was given inositol nicotinate the pain cleared for two weeks but then returned. At reoperation in 1969 both dorsal digital nerves were "involved with scar tissue on the dorsum of the operated area" and were cut proximally. The pain continued, severely disturbing her sleep. Local anaesthesia and acupuncture did not stop it. Examination in September 1976 showed that all sensation in the left fourth toe was lost; Tinel's sign was absent; and nerve conduction

in the posterior tibial and sural nerves was normal. When given carbamazepine, 200 mg at night, she reported "great improvement." When this was discontinued her pain returned and the drug was restarted. A course of dextropropoxyphene and paracetamol was of no help.

Case 2—A 46-year-old man had a ten-year history of intermittent, repetitive, lancinating pain and paraesthesiae in the left fourth and fifth toes, with free intervals of one to six weeks. It had started while he was sitting in a cinema. He had had to go back home "in agony," walking on bare feet as this

Double-blind cross-over trial of carbamazepine and placebo in case 2

Week	Drug	No bouts of pain	No intense paroxysms	Duration of pain (min)
1	Placebo	7	6	255
2	Carbamazepine	5	2	130
3	Carbamazepine	2	2	105
4	Placebo	6	3	210
5	Placebo	7	0	270
6	Carbamazepine	5	0	125
7	Placebo	3	0	15
8	Carbamazepine	1	0	5
9	Carbamazepine	3	0	60
10	Placebo	5	0	90
11	Placebo	2	0	35
12	Carbamazepine	1	0	5
13	Carbamazepine	0	0	0
14	Placebo	4	0	70
15	Carbamazepine	3	0	40
16	Placebo	0	0	0

gave him some relief. There were no constant precipitant factors; it occurred after prolonged walking, while sitting down, or in bed at night. In July 1978 he had localised pain on pressing the intermetatarsal space at the base of the third and fourth toes. X-ray films of the foot were normal. Nerve conduction in the posterior tibial nerves was normal. He was given a sixteen-week double-blind cross-over trial of carbamazepine and placebo, and made daily notes of the number, duration, and intensity of painful episodes. The first eight weeks he was given 200 mg thrice daily by mouth, and the second eight weeks 200 mg four times daily, both against placebo (see table). He remained on carbamazepine after the end of the trial with only occasional, trivial pain.

Comment

Pain occurring at night, or without apparent precipitant factors, was described by Morton.¹ Both patients appeared to benefit from carbamazepine but controlled studies in large series are required before accepting this clinical impression. In particular, the results of the double-blind cross-over trial are not conclusive. Assessment of pain depended on the patient's opinion. The design of the trial and lack of monitoring of the plasma concentration of carbamazepine make interpretation of the results uncertain.

In case 2 a "carry over" effect may have occurred. Intense paroxysms of pain stopped after the third week, whether the patient was taking carbamazepine or placebo. Nevertheless, on all five occasions in which there was a change from placebo to carbamazepine there was a reduction in the duration of pain. On three out of five occasions the change from carbamazepine to placebo was associated with an increase in the total duration of pain.

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