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.

¹ Scott McDougal, W, Izant, R J, jun, and Zollinger, R M, jun, *Annals of Surgery*, 1975, **181**, 310.

² Targan, S R, Chow, A W, and Guze, L B, *American Journal of Medicine*, 1977, **62**, 397.

³ Willis, A T, *Anaerobic Bacteriology*, 3rd edn, p 247. London, Butterworths, 1977.

⁴ McCartney, J E, and Fraser, J, *British Journal of Surgery*, 1922, **9**, 479.

⁵ Cross, R E, *Surgery of Infancy and Childhood*, p 384. Philadelphia, Saunders, 1953.

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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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¹ Morton, T G, *American Journal of Medical Science*, 1876, **71**, 87.

² Blom, S, *Lancet*, 1962, **1**, 839.

³ Heyck, H, in *Trigeminal Neuralgia. Pathogenesis and Pathophysiology*, ed R Hassler and A E Walker, p 115. Stuttgart, Georg Thieme Verlag, 1970.

⁴ Cambier, J, and Dehen, H, in *Epileptic Seizures—Behaviour—Pain*, ed W Birkmayer, p 317. Berne, Hans Huber Publishers, 1976.

⁵ Jacobson, R R, and Ross Russell, R W, *British Medical Journal*, 1979, **1**, 379.

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