medullary tumours are said rarely to produce pain as compared to the frequency with which extra-
medullary tumours present with pain as an early symptom. The reason for this being that the nerve roots are not involved so frequently in the case of intramedullary tumours. Sensory and motor disturbances are a more common mode of presenta-
tion and are steadily progressive until a definite transverse cord lesion with a sensory level is pro-
duced. The upper sensory level in intramedullary tumours is less distinct than in extramedullary form. Loss of sphincter control below the level of the lesion occurs early in the intramedullary type (Rasmussen et al., 1940).

The prognosis of an intramedullary oligoden-
drolioma is probably better than in other forms of intramedullary spinal tumours since it is so slow-growing. An intramedullary tumour dis-
covered at operation need not be considered inoperable (Woods & Pimenta, 1944). It is often impossible to differentiate extramedullary from intramedullary tumours prior to operation.

Summary

A case of a primary spinal oligodendrolioma is re-
ported. This is a rare tumour and in our search of literature we found only eleven previously reported cases. The patient presented as a case of attempted suicide.

Acknowledgments

We should like to express our thanks to Dr J. D. Williams and Mr G. B. Northcroft for allowing us to report this case.

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Phenacetin nephropathy in a patient with an ileostomy

N. H. DYER*
M.A., M.B., M.R.C.P.
Medical Registrar

The London Hospital, London, E.1

F. I. LEE†
M.B., M.R.C.P.
Senior Medical Registrar

An association between excessive ingestion of
analgesic compounds containing phenacetin and
renal damage is established (Spühler & Zollinger,
1953). Moeschlin (1958) estimated that there were
20,000 analgesic abusers in Switzerland and in
Denmark 29% of hospital in-patients take
phenacetin each day (Larsen & Moller, 1959). In
Finland, 20% of in-patients and 10% of factory

workers ingest analgesic compounds daily (Kasanen,
Forström & Salmi, 1962). The extent of the problem in the United Kingdom has prob-
ably been underestimated. Prescott (1966) found
that abuse of analgesics was common in north-east
Scotland, and that it was the cause of considerable
morbidity and mortality from renal disease. He
also commented on some unusual reasons for this
abuse. We report here a patient who took an
analgesic compound for the constipating effect of
its codeine content, and in whom phenacetin
nephropathy developed.

*Present post: Research Registrar, St Bartholomew's
Hospital, London, E.C.I.
†Present post: Physician, Victoria Hospital, Blackpool,
Lancs.
Case report

The patient was a man aged 40 years. At the age of 16 he developed ulcerative colitis which slowly worsened with intermittent attacks. At the age of 26 a total colectomy with spout ileostomy was performed at another hospital. The rectum was not removed. At this time his blood pressure was 175/80, his blood urea 29 mg/100 ml, and a mid-stream specimen of urine contained no pus cells and was sterile on culture. The ileostomy motions were lose at first but no measures to control them were needed. After 2 years the ileostomy was closed and an ileo-rectal anastomosis constructed. The disease recurred and an ileostomy was refashioned through the scar tissue of the old operation. Faeces tended to leak and were much more troublesome than previously. He found that codeine made the ileostomy easier to manage, and he took three to five compound codeine tablets daily. He bought his supply from his chemist as he did not wish to trouble his family doctor for a prescription for codeine phosphate. He suffered episodes of suprapubic pain, dysuria and frequency of micturition approximately twice a year. The symptoms improved with antibiotic therapy but no bacteriological studies were performed. There was no haematuria or renal colic and no necrotic papillae were voided. At the age of 38 he developed nocturia, once per night, without diurnal frequency. Three months before admission he suffered the first of three episodes of loin pain, malaise and dysuria. Albuminuria was found during a routine medical examination and he was referred to hospital. He admitted to a little mucus discharge from his rectal stump. On examination he appeared healthy with no signs of uraemia, anaemia or methaemoglobinemia. His blood pressure varied from 120/80 to 170/105. The liver and spleen were just palpable. Apart from the operation scars there were no other abnormal findings.

The urine contained 2.2 g protein/24 hr. The specific gravity varied between 1.003 and 1.008. The pH was 6. The deposit contained a few epithelial cells and hyaline casts and, on two out of six occasions, a few leucocytes. Culture was repeatedly sterile, and a colony count revealed less than 10⁴ organisms/mm³. There was no evidence of haemolysis or methaemoglobinemia. Blood film was normal and no Heinz bodies were seen. Blood urea varied from 67 to 120 mg/100 ml, and plasma creatinine from 4.2 to 5.7 mg/100 ml. Serum bicarbonate was 11 mEq/l and the other electrolytes were normal. Creatinine clearance was 24 ml/min, and maximum urinary urea concentration 7.5 g/l. An infusion intravenous pyelogram showed that both kidneys were reduced in size with irregular outlines and an increase in peripelvic fat. The pelvocalyceal systems were not well outlined. Renal biopsy included twelve glomeruli, all of which were shrunken and fibrotic. Periglomerular fibrosis was absent. There was diffuse interstitial fibrosis and tubular atrophy with focal lymphocytic infiltration. The vessels showed mild hypertensive changes. A separate fragment of medulla contained an extensive infarct of a renal papilla, which was diffusely calcified. Liver biopsy revealed an increased amount of lipofuscin, but was otherwise normal. Ulcerative proctitis of the rectal stump was found on sigmoidoscopy, but a barium enema revealed no adverse features.

He felt better on withdrawal of the phenacetin and commencement of oral sodium bicarbonate, but there was no immediate improvement in renal function.

Discussion

Compound codeine tablets contain 250 mg of aspirin, 250 mg of phenacetin and 8 mg of codeine. His phenacetin intake was therefore approximately 1 g daily for 10 years, giving a total consumption of more than 3.5 kg. This is sufficient to reduce renal function and cause renal damage in most cases (Larsen & Moller, 1959; Burry, de Jersey & Weendon, 1966). The renal biopsy appearances of the healed papillary infarct are almost pathognomonic of phenacetin nephropathy. The severe cortical damage is not against the diagnosis especially as it is out of proportion to the vascular damage. The hepatic lipofuscin deposition is further evidence of a tissue response to phenacetin (Abrahams et al., 1964; Rubinstein et al., 1964). Renal lesions have been described in ulcerative colitis by Jensen, Baggenstoss & Bargen (1950), but none of their cases developed papillary necrosis or chronic pyelonephritis.

Nordenfelt & Ringertz (1961), Grimmelndt (1963) and Prescott (1966) noted that most of their cases commenced the analgesic habit on the advice of other laymen and unknown to their doctors. Our patient did not wish to trouble his doctor and could easily obtain compound codeine tablets from his chemist. In most countries the popular analgesics do not contain codeine, but Prescott (1966) found that codeine-phenacetin combinations were favoured in twenty-two of his thirty-six cases and had been taken in ten of the eleven previously reported cases of analgesic nephropathy in the United Kingdom.

Reasons for analgesic abuse vary. Some patients have chronic painful organic disease (Clausen & Pederson, 1961) but these are in a minority.
Case reports


Summary
A case of phenacetin nephropathy is reported in a patient with an ileostomy, following a colectomy for ulcerative colitis. Renal damage occurred as an unwanted effect of the phenacetin content of compound codeine tablets, which were taken for the constipating action of their codeine component. It is hoped that the publication of this case may prevent similar occurrences.

Acknowledgements
We are grateful to Dr Kenneth Perry for permission to publish this case.

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Phenacetin nephropathy in a patient with an ileostomy.

N. H. Dyer and F. I. Lee

Postgrad Med J 1967 43: 791-793
doi: 10.1136/pgmj.43.506.791

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