PARAPLEGIA FOLLOWING THE USE OF "EFOCAINE"

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"Efocaine" is a long-acting local analgesic consisting of a solution of 1% procaine base, 0.25% procaine hydrochloride, and 5% butyl-*p*-aminobenzoate in a solvent composed of 2% polyethylene glycol-300, 78% propylene glycol, and water. It also contains 0.1% sodium metabisulphite and phenylmercuric borate (1:25,000) as preservative. When diluted with body fluids the aqueous insoluble analgesic base is deposited in a micro-crystalline form. The slow absorption of this deposit is believed to be responsible for the long-acting analgesic properties of efocaine, but it is evident that such a compound might cause serious ill effects if deposited in delicate tissue.

Three examples of such complications have been reported from America by Shapiro and Norman (1953); in one patient intercostal nerve block in the thoracic region was followed by "transverse myelitis" causing permanent paraplegia. Three further examples have been reported by Brittingham *et al.* (1954); in all three, paravertebral block of several intercostal nerves with efocaine was followed by paraplegia. Two of these patients improved to some extent, but the third was permanently paralysed. Angerer *et al.* (1953) described another striking case in which paraplegia developed within a few minutes of the injection of efocaine into an intercostal nerve. This patient died, and the necropsy findings suggested that the spinal cord lesions were secondary to thrombophlebitis of an intercostal vein.

The present report deals with a patient in whom evidence of severe spinal cord damage occurred after the injection of efocaine into intercostal nerves for the prevention of post-operative pain after the reduction of a hiatal hernia.

Case Report

A married woman aged 50, who had complained of indigestion for many years, was found to have a sliding hernia of the stomach through the oesophageal hiatus of the diaphragm. In spite of medical treatment her condition deteriorated until she could take only fluids by mouth. Oesophagoscopy showed superficial ulceration of the lower oesophagus adjacent to the herniated gastric mucosa.

On January 1, 1953, left lateral thoracotomy was performed under general anaesthesia with thiopentone, D-tubocurarine, gas and oxygen. The patient lay in the right lateral position and the left seventh rib was resected. A local analgesic consisting of 1/2,000 amethocaine hydrochloride and 0.25% procaine was injected into the third, fourth, fifth, sixth, seventh, eighth, and ninth intercostal nerves, into the mediastinal tissues, and into the splanchnic nerves. The hernia was reduced and the diaphragm repaired without difficulty. Before closing the chest 1 ml. of efocaine was injected directly into each of the sixth, seventh, eighth, and

ninth intercostal nerves at the angles of the ribs, where the nerves were clearly visible under the parietal pleura.

After returning to the ward the patient complained of numbness and paralysis of the legs. On January 2 she was found to have a flaccid paralysis of the lower trunk and leg muscles with complete sensory loss from the seventh dorsal dermatome downwards. Retention of urine and incontinence of faeces were present. On January 3 the C.S.F. contained 13 polymorphonuclear leucocytes per ml. and 950 mg. of protein per 100 ml., but there was no evidence of block on jugular compression. During the next three weeks her neurological condition did not change, but the C.S.F. returned slowly towards normal. On January 9 the C.S.F. protein value was 788 mg. per 100 ml. and on January 15 it had fallen to 435 mg.

The glucose and chloride content of the C.S.F. remained normal throughout. The C.S.F. Wassermann reaction was negative.

The thoracotomy wound healed normally and the digestive symptoms disappeared. A post-operative barium swallow showed complete reduction of the hernia and a normally functioning cardia. On January 24 the patient was transferred to a neurological unit for long-term rehabilitation.

On admission to the neurological unit some three weeks after operation the patient was found to have complete loss of spinal cord function from the seventh dorsal segment downwards. As she had complained of shoulder and trunk pains prior to operation the possibility of coincidental spinal cord compression was considered. A lumbar puncture on February 3 revealed the spinal fluid to be under a pressure of 110 mm. of water, with a rapid rise and fall of pressure on bilateral jugular compression. The fluid contained 3 cells (2 mononuclear and 1 polymorphonuclear), 100 mg. of protein per 100 ml., and an increased amount of globulin (Nonne-Apelt and Pandy). Radiographs of the spine showed no abnormalities apart from a minimal degree of osteophytosis in the mid-dorsal region, and a myelographic examination was within normal limits.

The usual measures for the long-term treatment of a spinal paraplegic subject were instituted. During the succeeding nine months there was never at any time evidence of returning spinal cord function. There remained complete absence of sphincter control, necessitating the regular administration of enemata and the maintenance of an indwelling vesical catheter. At the time of admission a small sacral bedsore involving skin and subcutaneous tissue was already present. This took many months to heal, and, despite the greatest possible care, it was impossible to prevent the development of similar lesions over the femoral trochanters. These later-developing lesions proved most resistant to treatment, so that at the end of the nine months' observation period they were still incompletely healed.

The patient was much troubled by persisting sensations of burning and "tightening" in her legs and lower abdomen, in addition to hallucinations of movements of her toes. These symptoms varied considerably in intensity from time to time and proved difficult to analyse, since they appeared to be dependent upon factors of both physical and psychogenic origin. Their response to the simpler analgesics was also variable, but at no time did they seem to become intolerable to the patient. On October 7 the spinal fluid was found to be normal in all respects, but in view of the extensive motor paralysis it was not possible to rehabilitate the patient beyond the state of a wheelchair existence, despite the application of appropriate leg splints. A final neurological examination confirmed that there was no objective evidence of spinal motor, sensory, or reflex function from the seventh dorsal segment downwards, a moderate degree of wasting of the legs having occurred by this time.

Discussion

This case emphasizes the possible catastrophic effects of the injection of a long-acting analgesic such as efocaine. It is conceivable that the drug passed along the perineural

sheath of one of the intercostal nerves, but the very high C.S.F. protein figures and the complete lack of recovery of reflex function below the level of the lesion suggest that extensive necrosis of the spinal cord occurred. Such widespread damage cannot reasonably be attributed to the small amount of efocaine which could have entered the spinal cord via the perineural sheath.

The clinical picture and C.S.F. changes can be explained on the basis of necrosis through interference with the blood supply of the spinal cord. The injection of efocaine close to an intercostal vein might have produced thrombosis which spread into the spinal cord and caused intramedullary thrombosis with necrosis. This view is supported by the necropsy findings in the patient reported by Angerer *et al.* (1953). Forty-four hours after the injection of efocaine they found that the intercostal vein immediately beneath the needle puncture was acutely inflamed and filled with recent thrombus. The spinal cord itself showed extensive necrosis, with evidence of acute inflammation.

The mode of action of efocaine has been reinvestigated by Mannheimer *et al.* (1954), and they have shown that the substance produces necrosis of both nerve and muscle of similar severity to that caused by the injection of alcohol and phenol. These findings, together with the published reports of gross neurological damage, lead to the conclusion that the clinical use of efocaine is no longer justifiable.

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Medical Memoranda

Long Survival in Renal Carcinoma

There are many gaps in our knowledge of the natural life history of malignant tumours, and renal cancer seems to be particularly unpredictable. In the patient we are here reporting, at least a quarter of a century elapsed before the disease caused death, and treatment was refused for a decade after the tumour was clinically apparent.

CASE REPORT

In 1930 a Jewish business man, aged 31, began to experience pain in the left flank and haematuria. The pain was episodic until 1934, when its severity so increased that he sought admission to hospital. A mass was then discovered in relation to the left kidney, of such a size that it caused obvious deformity of the stomach on radiological examination.

Surgical treatment was strongly urged, but the patient stubbornly refused to submit to operation until 1943. The operation disclosed a rounded tumour which replaced the lower pole of the kidney and was "much larger than the renal tissue proper." There was no involvement of the renal vein, or any evidence of metastases. Nephrectomy was carried out.

Pathological examination of the tumour by the late Colonel Harvey showed the tumour to be a glandular carcinoma (hypernephroma) of clear cell type. Its structure was unusually glandular, and of the small alveolar and tubular type rather than the larger and more cellular masses more common in this tumour. There was considerable polymorphism of the cell nuclei, suggestive of degenerative forms. Though mitotic activity was not recognized, the tumour had spread into the fibrous tissue separating the growth from the kidney. In some areas haemorrhage and necrosis were marked.

The patient remained relatively well until 1949, when he began to complain of vague abdominal discomfort. Sundry investigations were negative; but in 1952, when he came under our joint care, there was a mass in the left flank and some enlargement of the liver. Barium enema revealed a filling defect suggestive of extrinsic pressure in the descending colon, and a radiograph of the chest showed a rounded opacity in the right mid-zone. As much for diagnostic as therapeutic reasons, he was advised to undergo laparotomy.

At operation extensive malignant disease was found throughout the abdominal cavity, including a large fixed mass involving the descending colon and the adjacent part of the posterior abdominal wall. Numerous secondary growths were present in the liver, including a massive growth in the left lobe. It was found practicable to remove the pericolonic tissue and so relieve the obvious obstruction.

Histological examination showed the tissue to be metastatic hypernephroma. There was a remarkably good immediate recovery, and he was able to return to the business he had conducted so successfully throughout the years; thereafter his condition deteriorated; but in spite of the extensive metastases he survived for another two years, and died in September, 1954.

COMMENT

It is notoriously difficult to forecast the expectation of life in patients with renal tumours. Herbut (1952) has stated that symptoms and signs may be prolonged up to 35 years; and Griffiths and Thackray (1949) reported a 10-year survival rate of 21% after nephrectomy. The "best" American figures—those of Priestley (1939) at the Mayo Clinic—record a 10-year survival in 27.3% of 395 cases. Nevertheless, survival for 25 years despite the well-substantiated existence of renal carcinoma, as in the present case, seems to us most exceptional. Possibly the phenomenon was thrown into sharper relief by a number of contemporary cases in which the course was very rapid.

The corollary is obvious—that great caution must be exercised in venturing a prognosis in renal tumours, both with and without operation, and that even greater caution is needed when claims are made for the efficacy of treatment.

We are indebted to Dr. Alister Matheson for letting us have access to his careful records regarding this patient.

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Case of Bilateral Calcification of the Breasts

The deposition of calcium in the soft tissues of aged subjects is not uncommon, particularly in those parts where fibrous tissue normally predominates. In the breast, fibrous tissue is laid down in varying amounts after the menopause; only very rarely, however, does it go on to calcification so extensive as to be detectable both clinically and radiologically. The following is a case discovered accidentally shortly before the patient's death.

CASE RECORD

A woman aged 85 was admitted to hospital with a three-weeks history of pain in the left groin. Her private practitioner suspected the presence of a fatigue fracture of the neck of the left femur, no history of trauma being elicited. This was confirmed radiologically. Examination of the