Short report

'Oblongata' crises in tabes dorsalis

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SYNOPSIS A patient in the pre-ataxic stage of tabes dorsalis suffered from gastric crises, but in addition had numerous episodes of apnoea and coma which in the older literature have been described as 'oblongata crises'—the presumption being that the crises are due to a brain stem disturbance.

Visceral crises in tabes dorsalis are well known, though uncommon. Central manifestations occurring in tabes have received little attention. The following case presented with gastric crises which in addition were sometimes associated with episodes of apnoea and coma.

CASE REPORT

A 44 year old clerical officer presented at another hospital in December 1974 with sudden severe epigastric pain which disappeared suddenly four hours after onset. No cause was found for the pain and he was discharged after three weeks.

Six months later he was admitted to this hospital as an emergency with a recurrence of the same pain. It was a severe and constant central abdominal pain which made him roll around in agony. There was associated retching, vomiting, and diarrhoea. Abdominal examination was normal as were chest and abdominal radiographs and the serum amylase.

The next day he again complained of abdominal pain and was found soon afterwards cyanosed, unconscious, and with absent respirations. He recovered spontaneously within two minutes. An ECG, chest radiographs, blood sugar, urea, and electrolytes were all normal. Continuous cardiac monitoring was begun. Over the next 36 hours he had three further episodes of apnoea and unconsciousness followed once by a mild grand mal fit. The cardiac monitor showed a sinus tachycardia during these episodes.

The patient admitted that he was a passive homosexual. He denied previous syphilis. On further examination he was noted to have bilateral ptosis

with overaction of the frontalis muscle. There were Argyll-Robertson pupils and blunted pain sensation over the mask area of the face, lower anterior chest, ulnar borders of forearms, and lateral aspects of the legs. Deep pain sensation was absent. On one occasion only, the bladder was palpable up to the level of the umbilicus without the desire to micturate, requiring catheterisation. All tendon reflexes were normal and plantar responses flexor. There was no loss of posterior column sensation and no ataxia. Romberg's sign was negative. The fundi were normal and the patient was of above average intelligence. There were no cardiovascular signs of syphilis.

Serological tests showed the *Treponema pallidum* haemagglutination test (TPHA), Reiter protein complement fixation test (RPCFT), VDRL slide test, fluorescent treponemal antibody test (FTA), and Wassermann reaction (WR) to be positive in the blood. The CSF contained protein 6.8 g/l and 31 lymphocytes per mm³. The Lange curve was 12222000 and the TPHA test was positive. The EEG and brain scan were normal.

A diagnosis of tabes dorsalis with gastric crises was made, but the cause of the apnoeic episodes was unexplained. On the fourth day after admission four apnoeic attacks occurred during a day of continuous abdominal pain and retching. The usual sequence was that the apnoeic attacks began suddenly and without warning. He would stop breathing, lose consciousness, become pale and cyanosed, and then have mild generalised twitching. On one occasion there was conjugate deviation of the eyes upwards and to the right. The pulse rose to 180/min and became thready. After about two minutes he would breathe when exhorted to do so, but not otherwise. After five minutes he was breathing spontaneously, fully conscious, and the abdominal pain had gone. He could only remember that he had suddenly 'blacked out' without warning. He was treated with a

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21-day course of procaine penicillin with initial steroid cover against a possible Herxheimer reaction. Both phenytoin and carbamazepine were used to prevent the apnoeic attacks, but seemed ineffective. The pain responded poorly to non-narcotic analgesics and seemed to subside spontaneously. During the penicillin therapy he still had occasional days of abdominal pain but no further apnoeic attacks. In the two months after discharge from hospital he developed two episodes of severe abdominal pain requiring readmission.

DISCUSSION

Although gastric crises are well known in *tabes dorsalis*, central manifestations associated with visceral crises are not mentioned in modern text books on neurology though reviewed by Kinnier Wilson (1940). The apnoeic episodes experienced by our patient are rare. Dusser de Barenne (1913) described a patient with taboparesis in which 'the nurse reported that the patient had suddenly died. He had suddenly become pale and unconscious, had stopped breathing and the pulse could not be felt. After two and a half minutes he began with slow, deep breathing. He called these episodes 'oblongata crises', believing that the site of dysfunction causing these attacks was the brain stem. At postmortem examination, however, no specific pathological changes were found in this region. This patient did not suffer from gastric crises, but de Barenne quotes two other cases from the literature in which the patients did have gastric crises and in whom morphine injections precipitated apnoeic episodes.

The only other similar situation was described by Guillain (1922) as an apnoeic variety of 'bulbar crisis'. His tabetic patient had various forms of visceral crises and 'developed great slowing of respiratory movements lasting several hours, necessari-

tating stimuli to the nasal passages to induce respiration'.

The cause of such brain stem disturbances remains unexplained, as does the cause of visceral crises. The various theories are reviewed by Catterall *et al.* (1961) and include suggestions that the crises are (1) of sympathetic origin, (2) due to recurrent posterior ganglionitis, (3) a result of vagal involvement, or (4) irritation and later paralysis of the autonomic nervous system. It is generally agreed that most crises begin in the pre-ataxic stage of *tabes*, as in our case.

Opinion is also divided as to the prognosis of crises after penicillin therapy but most feel that they do become less frequent with treatment—a view supported by Brain and Walton (1969).

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