Clinical Section

President Harold Ellis MCh

Meeting March 10 1972

Cases

Cephalic Tetanus

R G Farquharson MB (for S Karani FRCP)
(*Brook General Hospital, Shooters Hill, London SE 18*)

Miss J K, aged 28

History: Seen ten days after receiving a kick in the face from a horse, producing laceration of left upper lip and nose, which had been sutured. One megaunit of penicillin had been given, but no tetanus antitoxin or toxoid, and she had never previously received toxoid. Seven days later, when the wound had healed and the swelling subsided, she noticed difficulty in opening her mouth, associated with facial stiffness predominantly on the left. Within forty-eight hours the facial spasm had reduced her to a diet of fluids, early tetanus was diagnosed, and she was transferred to our care twenty-four hours later.

On examination: She was apyrexial, with a well-healed 3.5 cm laceration of the left upper lip and nose. Marked trismus and incomplete left VII lower motor neurone palsy (Fig 1). No other abnormal physical signs.

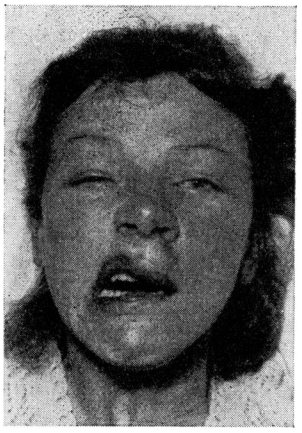

Fig 1 *Nine days after onset of symptoms, by then improving. Shows healed laceration of left lip, marked trismus, left VII cranial nerve palsy*

Investigations: Full blood count and ESR normal. Electrodiagnostic studies (Dr E H Innes) four weeks after the onset of symptoms, when clinically she was much improved, demonstrated positive potentials but no fibrillation at rest in the left orbicularis oris, with a response to 4 mA compared with 3 mA on the right. On volition there was a complete interference pattern of a mixture of normal units and bursts of rapid high frequency discharges consistent with tetanic spasm.

Treatment and progress: A course of penicillin and tetanus toxoid was begun and spasms were controlled with intramuscular diazepam. She received one dose of heterologous antiserum 50 000 u i.m. Tetanus remained localized to the head, with no generalized spasm. She began to improve one week after the onset of symptoms and two months later there were no signs of spasm or cranial nerve palsy.

Comment

Cephalic tetanus may be defined as tetanus, either localized or generalized, with an associated cranial nerve palsy. The severity of tetanus is usually inversely proportional to the duration of the incubation period, periods of seven days or less carrying a grave prognosis. In this case, however, the incubation period was seven days and the disease ran a very mild course. This case also confirms that a cranial nerve palsy associated with tetanus is not necessarily of bad prognostic significance (Patel *et al.* 1960, Patel & Mehta 1963, Vakil *et al.* 1964, Jaffari 1966). Recovery of the cranial nerve palsy within about two months is usual if the patient survives the early illness (Park 1970).

The site of the lesion and the nature of the pathology in cephalic tetanus is still not established. It is generally held that the cranial nerve

nuclei are damaged (Elischer 1876, quoted by Baker 1943; Park 1970) and the electrodiagnostic study in this case, whilst not characteristic of nuclear damage, is compatible with this.

REFERENCES
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Dr F B Gibberd (*Westminster Hospital, London SW1*) said that this patient was described as having tetanic spasms in a muscle paralysed by lower motor neurone palsy. This was difficult to explain if the tetanic spasm arose from a central action of the toxin. In these circumstances the tetanus would arise as a result of nerve impulses passing down the lower motor neurone. The EMG showed no evidence of lower motor neurone palsy. Could it be that the apparent palsy was not due to a peripheral motor neurone lesion?

Dr Farquharson replied that the lesion was clinically peripheral and trismus persisted, therefore the palsy was taken to be partial. EMG was performed too late to clarify this.

Apparently Benign Left Bundle Branch Block

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and Edgar Sowton MD FRCP
(*Guy's Hospital, London SE1*)

Mr J P, aged 40

History: Presented three years previously with epigastric pain relieved by food. There was no chest pain and no past history of rheumatic fever or heart disease.

On examination: No abnormal physical findings.

Investigations: Urine analysis negative, serum cholesterol and triglycerides normal. A barium meal revealed a duodenal ulcer. Chest X-ray normal. Resting ECG was normal but after an effort test the pattern of left bundle branch block with left axis deviation developed.

He was subsequently seen elsewhere and advised that he had coronary artery disease. Coronary arteriography was therefore performed in order to resolve the question but during the procedure intense sinus bradycardia developed, leading to complete heart block and ventricular asystole; pacing was therefore necessary. Coronary arteriograms and a left ventricular angiogram were normal. A His bundle electrogram during sinus rhythm showed a His-Q time of 95 msec; this represents considerable delay in conduction down the right bundle branch as well as persistent left bundle branch block (Fig 1). A permanent pacemaker has been required.

Discussion

Left bundle branch block is generally considered to be due to organic heart disease and to carry a serious prognosis (Wood 1968). This patient was thought to have benign left bundle branch block, but subsequently developed complete heart block and required a permanent pacemaker.

Krikler & Lefevre (1970) described this case and one other as representing left bundle branch block occurring in the absence of obvious organic heart disease, and considered not necessarily to have a serious prognosis; careful follow up was advised to verify this.

Left bundle branch block occurring in the presence of a normal QRS axis is due to block occurring in the left main bundle before it divides (Rosenbaum 1969). Left bundle branch block and left axis deviation occur when there is block in the left main bundle and additional block in the anterior division of the left main bundle (Pryor & Blount 1966, Rosenbaum 1969), implying that there is more extensive disease of the left bundle branch system when left bundle branch block and left axis deviation are seen on the external ECG than when left bundle branch block occurs with a normal axis.

Spurrell *et al.* (1972) have investigated this abnormality by recording His bundle electrograms in patients with left bundle branch block and a normal axis, and with left bundle branch block and left axis deviation. In the presence of

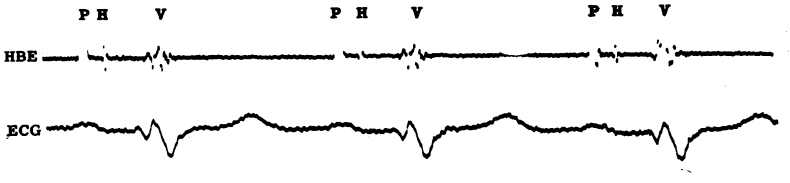

Fig 1 *His bundle electrogram recorded from J P in sinus rhythm. Rate 84/min. P-His 75 msec (representing conduction time through atria and atrioventricular node; normal 80–140 msec). His-Q 95 msec (measured from His spike to Q wave on external ECG, representing intraventricular conduction time; normal 42–60 msec)*