

Fig. 2 Five weeks after tuberculostatic treatment. (a) Lesion appears unchanged. but (b) periosteal reaction has disappeared.

Discussion

Pauker et al. (1972) described 2 cases of BCG osteomyelitis and osteoarthritis of the knee joint with positive pathologico-anatomical findings but negative bacteriological findings. In the present case the infection seemed to be of low virulence, no regression was seen after several weeks' treatment with antibiotics effective against Gram-positive cocci, and fewer than 4 years had elapsed since the BCG vaccination. In accordance with the criteria of Foucard and Hielmstedt (1971) we therefore concluded the lesion was probably BCG osteomyelitis. Since our experience and that of others (Paisseau et al., 1941; Backman and Wallgren, 1954; Felländer, 1963; Foucard and Hjelmstedt, 1971) was that even biopsy may not give the diagnosis with certainty we decided to try conservative treatment. The prompt healing with tuberculostatic treatment was in our opinion sufficient evidence that the diagnosis and management were correct.

On the basis of this case a 2-month-old baby girl with an osteolytic focus of the humerus near the site of the BCG inoculation was recently treated elsewhere in the same conservative way with good results.

Summary

A 3-year-old boy presented with an osteolytic lesion and periosteal reaction in the distal metaphysis of the right femur which failed to respond to immobilization and intensive antibiotic treatment. Since the infection seemed to be of low virulence and fewer than 4 years had elapsed since BCG vaccination BCG osteomyelitis was suspected. Tuberculostatic treatment led to rapid recovery and surgical measures were unnecessary.

References

Backman, A., and Wallgren, E. I. (1954). On tuberculosis of the bones and joints of BCG vaccinated children. Acta Paediatrica, 43, 252-258.

Felländer, M. (1963). Tuberculous osteitis following BCG vaccination. Acta Orthopaedica Scandinavica, 33, 116-126. Foucard, T., and Hjelmstedt, A. (1971). BCG-osteomyelitis and -osteoarthritis as a complication following BCG vaccination. Acta Orthopaedica Scandinavica, 42, 142-151.

Marks, J., Jenkins, P. A., Kilpatrick, G. S., Engback, H. C., and Vergmann, B. (1971). Pulmonary tuberculosis due to BCG. British Medical Journal, 3, 229-230.

Paisseau, G., Sorrel, E., and Ngugen, K. V. (1941). Mal de Pott sous-occipital chez un nourrisson vacciné au BCG. Bulletins et Mémoires de la Societé Medicale des Hôpitaux de Paris, **57.** 328-333.

Pauker, M., Lotem, M., Levy, M., and Luria, M. (1972). BCG arthritis and osteomyelitis. Harefuah, 83, 429-432.

M. PAUKER,* M. SEELENFREUND, and G. MORAIN Department of Orthopedic Surgery and Traumatology, Beilinson Medical Center, Sackler School of Medicine, Tel Aviv University, Petah Tikva, Israel.

*Correspondence to Dr M. Pauker.

Symptomatic sinus arrest in a young girl

A recent report (Scott et al., 1976) on symptomatic sinoatrial node dysfunction noted its infrequency in children and no reported occurrence of the

syndrome in girls. The following case is therefore presented.

Case report

A 13-year-old girl was first seen at 9 years of age because of an asymptomatic persistent ductus arteriosus. At this time electrocardiograms (ECG) were normal, showing rates from 72-80/min, normal P waves, and PR interval 140 ms. Ligation and division of the ductus was accomplished uneventfully. Postoperatively there were no arrythmias and an appropriate increase in heart rate to stress. One year postoperatively there was no clinical or x-ray evidence of heart disease and ECG was unchanged. She was therefore discharged.

She reappeared 3 years later because of recurrent syncopal attacks of 2 years' duration. Attacks were described as transient loss of consciousness, unrelated to activity and unaccompanied by other manifestations. These episodes had occurred two to three times a month during the first year and three to four times a month in the second year. There had been no known illness before the onset of the syncopal attacks. Drug use was denied. There was no family history of arrythmias or syncope.

Examination at this time showed no clinical evidence of heart disease. Chest x-ray was normal. ECG showed a frontal plane axis of $+70^{\circ}$ and no evidence of chamber enlargement. The rate was 45–49/min, PR interval 200 ms, QRS interval 40 ms, and QT interval 400 ms (Fig. 1a). On exercise heart rate increased to 75/min and PR interval to 260 ms (Fig. 1b).

She was hospitalized for further diagnostic study and therapy. On Holter monitoring when the sinus rate was 71/min or less, there were frequent periods of sinus arrest, maximally of 1 · 6 s duration (Fig. 1c). The sinus arrest was unrelated to activity; syncope did not occur under observation. Additional labora-

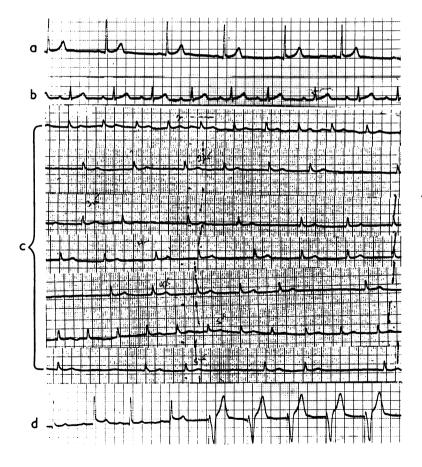


Fig. 1 (a) Resting ECG, sinus bradycardia at a rate of 45–49/min with PR interval 200 ms; (b) after exercise, with an increase in heart rate to 75/min and PR interval 260 ms; (c) representative tracings over a 3-hour period of Holter monitoring showing frequent episodes of sinus arrest, maximum 1·68 s; (d) pacemaker tracing, showing periodic appearance of sinus beats. PR interval 260 ms.



Fig. 2 His bundle electrogram with the patient paced from the high right atrium at a rate of 68/min. There is Wenckebach atrioventricular block associated with the second and third pacer stimuli. S=pacer stimulus; A=atrial depolarization; H=His bundle depolarization; L2=lead 2 of surface ECG.

tory studies ruled out other causes for syncopal attacks.

Cardiac catheterization and cineangiography showed no structural abnormalities. Cardiac dvnamics were normal; the cardiac index was 3.82 1/min per m². Atrioventricular conduction was evaluated by means of His bundle studies and atrial pacing at rest and under the influence of atropine. The resting heart rate was 51/min with PR interval 245 ms. A-H interval was 204 ms and H-V 44 ms. Atrial pacing at a rate of 68/min produced Wenckebach atrioventricular block (Fig. 2). After administration of atropine 0.4 mg IV, heart rate increased to 88/min with short periods as high as 98/min. PR interval was then 225 ms, but A-H interval decreased to 171 ms and H-V interval was essentially unchanged at 46 ms.

Because of the recurrent syncope and demonstrated abnormalities, permanent cardiac pacing seemed to be indicated. She was implanted with a transvenous ventricular-inhibited (Biotronik) pacemaker in the right retromammary position at a rate of 70/min. Over the subsequent 4 years she has been entirely asymptomatic and has pursued normal activity. Sinus rhythm with first-degree block frequently occurs when the sinoatrial rate exceeds that of the pacemaker (Fig. 1d).

Discussion

Sinus bradycardia with sinus arrest was shown in this patient on Holter monitoring. The frequent prolongation of the PR interval and production of the Wenckebach phenomenon at a pacing rate below 100 (2 SDs) is clear evidence of disturbed atrioventricular conduction (Pahlajani et al., 1975). Intra-atrial conduction delay is also suggested. inasmuch as the PR interval remained prolonged while the A-H interval shortened and the H-V interval was unchanged when a faster rate was produced on administration of atropine.

Sinus node dysfunction with atrioventricular conduction disturbance has been reported in a number of adults (Eraut and Shaw, 1971) but only once in children (Nugent et al., 1974). Complete heart block with persistent ductus arteriosus, though uncommon, is second in frequency to its occurrence with L transposition in patients with congenital heart disease (Nakamura and Nadas, 1964; Michaëlsson and Engle, 1972). Sinus node dysfunction in association with congenital heart disease (types unspecified) has been described in 4 children and attributed to a congenital defect of the sinus node or alterations on the sinus node due to right-sided volume or pressure overload (Nugent et al., 1974).

The aetiology of the sinoatrial dysfunction and disturbance in conduction in this patient is obscure. Such abnormalities have not been noted previously in patients with persistent ductus arteriosus. Haemodynamic abnormalities were not present. The time relationship of the two conditions reasonably negates a congenital origin. Evidence, however, for an acquired aetiology is also lacking.

Summary

Sinus node dysfunction, previously unreported in girls, occurred in a 13-year-old girl who required permanent pacemaker implantation because of recurrent syncopal attacks. In addition to periodic sinus arrest, the presence of significant atrioventricular conduction disturbance was also documented. Although she had had a persistent ductus arteriosus divided at an earlier age, the disturbance of rhythm and conduction cannot be ascribed to a congenitial or haemodynamic abnormality. An acquired origin can only be postulated.

References

Eraut, D., and Shaw, D. B. (1971). Sinus bradycardia. British Heart Journal, 33, 742-749.

- Michaëlsson, M., and Engle, M. A. (1972). Congenital complete heart block: an international study of the natural history. *Cardiovascular Clinics*, 4, 85-101.

 Nakamura, F. F., and Nadas, A. S. (1964). Complete heart
- Nakamura, F. F., and Nadas, A. S. (1964). Complete heart block in infants and children. New England Journal of Medicine, 270, 1261-1268.
- Medicine, 270, 1261-1268.

 Nugent, E. W., Varghese, P. J., Pieroni, D. R., and Rowe, R. D. (1974). 'Sluggish' sinus node syndrome as part of congenital heart disease. American Journal of Cardiology, 33, 160.
- Pahlajani, D. B., Miller, R. A., and Serratto, M. (1975).
 Patterns of atrioventricular conduction in children.
 American Heart Journal, 90, 165-171.
- Scott, O., Macartney, F. J., and Deverall, P. B. (1976). Sick sinus syndrome in children. Archives of Disease in Childhood, 51, 100-105.

Dennison Young* and Robert E. Eisenberg Department of Pediatrics, Montefiore Hospital and Medical Center, 111 East 210th Street, Bronx, New York 10467, USA.

*Correspondence to Dr D. Young