improvement was seen in our patient. Echocardiography and phonocardiography after 48 hours of therapy showed a normal-functioning prosthetic valve. The opening angle measured by cinefluoroscopy was more than 60°.

The potential advantage of thrombolytic therapy includes the avoidance of reoperation in most patients. If total success is not achieved by fibrinolytic therapy, the patient's haemodynamic status stabilises and he stands a better chance of tolerating open-heart surgery. Overall mortality with this approach appears to be much less as compared with emergency operation [3,4].

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References

Varicella myocarditis is frequently associated with rhythm disturbances [1–4]. We present here a case complicated by permanent complete heart block. We have been unable to find a previous description of this association during life in the English language literature.

Case Report

A 17-month-old boy with no previous evidence of heart disease presented to the local hospital when his mother, a Health Visitor, noted a pulse rate of 60/minute 5 days after he had developed a varicella skin rash. Two weeks prior to the onset of the skin lesions his brother had also had varicella. An electrocardiogram showed complete heart block and the diagnosis of varicella myocarditis was made (Fig. 1). A 7-day course of 5 mg/kg of intravenous Acyclovir every 8 hours was started. The following day he was transferred to Guy’s Hospital because the heart rate had dropped to 40/minute. On admission his temperature was 37°C, respiratory rate 34/minute, pulse 44/minute and blood pressure 100/50 mm Hg. The typical cutaneous lesions of varicella were present (Fig. 2). The apex beat was displaced to the anterior axillary line in the fifth intercostal space and there were no signs of heart failure. There was moderate cardiomegaly on the chest radiograph with normal lungfields. An echocardiogram revealed left atrial and left ventricular dilatation; there was a small pericardial effusion with no signs of tamponade. The only abnormal laboratory finding was a white blood cell count of $2.4 \times 10^9$/litre with 26% neutrophils that returned to normal.

Fig. 1. Twelve-lead electrocardiogram showing complete heart block with an atrial rate of 115/minute and a ventricular rate of 44/minute.
after 3 days. A temporary transvenous pacing system was positioned in the right ventricle at a rate of 120/minute. A first pass and gated radionuclide angiogram performed the following day showed an enlarged left ventricle with septal and apical akinesia. The left ventricular ejection fraction was 35% (normal 55–75%); right ventricular function was normal. Repeat echocardiograms showed resolution of the pericardial effusion. Serial radionuclide angiograms demonstrated complete recovery of left ventricular function. Despite not having symptoms attributable to the slow endogenous heart rate, an escape rhythm of 45/minute with wide QRS complexes was considered an indication for permanent pacing. An epicardial electrode connected to a programmable pacemaker in an extrapleural pocket within the left chest was surgically introduced. After 18 months he was still in complete heart block and entirely pacemaker dependent. He remains free of cardiac symptoms.

Discussion

Complete heart block is a serious arrhythmia. The patient presented here required permanent pacing. The most probable pathological mechanism leading to heart block in
varicella is inflammatory cell infiltration in the atrioventricular node and the bundle of His. This was the histological finding in one case report of sudden death complicating varicella [3]. Post-mortem results on another child with fulminating varicella associated with supraventricular tachycardias showed extensive myocarditis; involvement of the specialized conducting tissue was not mentioned [1]. Our patient remained in complete heart block despite the use of the antiviral agent Acyclovir from the moment of presentation. This suggests that irreversible damage to the specialized conducting tissue had already occurred.

Spontaneous recovery of myocardial dysfunction in varicella myocarditis has been described [2], but a residual congestive cardiomyopathy is a recognized complication [5]. It is possible that treatment with Acyclovir induced complete recovery of left ventricular function in our patient.

Cardiac complications in the course of varicella in childhood can be severe. The role of Acyclovir in the treatment of varicella myocarditis is not clear.

References