with congenital heart disease,⁴ the youngest previously reported case of Q fever endocarditis was in a 15-year-old boy with congenital subaortic stenosis,⁹ and 7 adults with involvement of congenital bicuspid aortic valves have also been described.² The present case emphasises that chronic Q fever infection must be considered in the differential diagnosis of infective endocarditis in childhood, particularly if there is a history of contact with farm animals and if blood cultures are negative.

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Facial palsy in an infant with coarctation of the aorta and hypertension

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SUMMARY We report the unusual association of facial palsy and severe hypertension in an infant with coarctation of the aorta. The facial palsy resolved before the hypertension was cured.

Case report

A 14-week-old boy was admitted because of failure to thrive and for recoarctation after a left subclavian flap aortoplasty which had been performed for a severe isolated coarctation at age 2 weeks. The femoral pulses which had been present after surgery were absent at 10 weeks, and repeat cardiac catheterisation and angiography at 11 weeks had demonstrated the site of recoarctation and shown that left ventricular function was poor and there was a thickened left ventricular wall. On admission the infant was in mild cardiac failure and his systolic blood pressure was 200 mmHg in the right arm and 80 mmHg in the legs, measured by the flush method. The next day a right lower motor neurone facial palsy was noted (this was particularly obvious when he cried), with facial asymmetry and inability to close the eye. No other neurological abnormality

was found and fundoscopy was normal. Despite little success in controlling blood pressure with β -receptor blockade, the facial palsy improved after 5 days and had resolved after 3 weeks. The only other treatment given was methylcellulose eye drops and a pad to close the eye. Six weeks later the coarctation was successfully repaired by inserting a Dacron bypass graft at the site of the previous aortoplasty. The blood pressure fell to 140 mmHg (flush method) in the right arm in 4 days. Three weeks later there was a further fall to 120 mmHg. He remains well and is normotensive 6 months later.

Discussion

Lower motor neurone facial palsy associated with hypertension was first noted by Moxon in 1869.¹ Since then the association has been described in adults and children.²⁻⁵ An incidence of between 3 and 17% has been reported,³⁻⁵ although it was not noted in a review of 100 patients.⁶ All the reported cases have been associated with hypertension of a renal or idiopathic aetiology and the overall prognosis has been poor. The present case appears to be the first to be caused by hypertension

due to coarctation of the aorta. This is probably because coarctation of the aorta accounts only for between 11 and 15% of severe childhood hypertension.⁴⁻⁶ However, the outlook with this form of hypertension is excellent, as in our patient. The pathogenesis of the facial palsy is unknown but haemorrhage¹⁻³ or oedema in the facial canal may be important factors. As in this case the prognosis for the palsy is good.

In conclusion, lower motor neurone facial palsy in childhood may be associated with significant hypertension and the blood pressure should be recorded in any child with a facial palsy.

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