LETTERS TO THE EDITOR

Permanent cardiac pacing for reflex anoxic seizure

EDITOR,—Reflex anoxic seizure (pallid syncope) is defined as vagally mediated cardiac arrest producing cerebral ischaemia.^{1,2} Recommended treatment consists of parental reassurance that the attacks are benign, with atropinisation in occasional cases.³

Case report

A 3 year old neurodevelopmentally intact girl presented with a history of recurrent anoxic seizures from infancy, which were typically precipitated by minor injury or upset. During the attacks, which could be provoked by eyeball compression, she exhibited a combination of breath holding ('blue attacks') in expiration, with up to 24 seconds of cardiac asystole. On occasion, there was a marked clonic seizure during the recovery phase, and on ambulatory electroencephalographic (EEG) recordings this was associated with bilateral symmetrical cortical discharges. The attacks occurred between 10 and 40 times a day. A trial of atropine had failed to abolish her events, and had produced unacceptable side effects including blurred vision and dry mouth. In February 1995 a temporary ventricular pacing lead was introduced via the femoral vein. Subsequent provocation by eyeball compression was undertaken with continuous video EEG and electrocardiographic (ECG) monitoring, with and without cardiac pacing via an external generator. Without pacing, asystole for 12 seconds occurred, associated with syncope and a 'flat' EEG recording. With pacing (VVI, rate 70 bpm), all components of the attack and EEG changes were abolished. On this evidence, a permanent transvenous ventricular lead was implanted (VVI), with a subpectoral generator. Over the next 12 months, her pallid syncope attacks have been virtually abolished, and she is able to attend nursery school full time. She continues however to have minor episodes of uncertain nature, but without loss of consciousness. Twelve months after permanent pacemaker implant, she attended the cardiac outpatient department for three consecutive days, during which the generator was randomly switched either off or on with the family, patient, and physicians 'blinded'. Several minor events were recorded by the parents in a diary, but without EEG or ECG accompaniments. However, within 36 hours of the pacemaker being turned off, she had a typical reflex asystolic/anoxic event with 23 seconds of asystole accompanied by 'flattening' of the EEG recording followed by high amplitude slow waves. This was the first typical attack after the pacemaker implant.

While typical reflex anoxic seizures are generally benign, fatal and near fatal cases have been reported.⁴ Treatment was required in this case because of failed medical treatment in the face of the severe social and emotional disability caused by the high frequency and severity of attacks. The long term effects of frequent episodes of moderately prolonged cerebral anoxia are unknown.

There are few studies documenting the effectiveness of atropine^{3,4}; however, it may well be that our patient was not completely atropinised despite exhibiting side effects. By eliminating asystole, cardiac pacing has prevented this child from having her usual reflex asystolic/anoxic seizures and has produced a significant improvement in the quality of life for the child and her family.

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Oral anticoagulation nephrotic syndrome

EDITOR,—Tait *et al* highlighted the lack of experience with oral anticoagulation in children.¹ We report the case of a child with congenital nephrotic syndrome complicated by pulmonary embolism where anticoagulation with warfarin proved difficult to control and was abandoned. Variations in the pharmacokinetics of warfarin are postulated that may explain the difficulties we experienced in achieving adequate anticoagulation.

The diagnosis of congenital nephrotic syndrome was confirmed histologically in an infant boy with oedema, proteinuria, and low serum albumin concentration. He was treated with albumin infusions (2 g/kg/day) via a central venous line three times a week and oral frusemide. At 3 months of age he developed an *Enterococcus faecalis* line infection which was treated with intravenous vancomycin. A subsequent episode of tachypnoea, after a blood transfusion, was attributed to multiple pulmonary emboli after an abnormal ventilation-perfusion lung scan. Treatment was initiated with intravenous heparin (110 units/kg/day) for one week then subsequently oral warfarin (100 µg/kg/day). During the second week when three doses of albumin were administered it was not possible to achieve an international normalised ratio (INR) within the target range (2.0-3.0). This was despite increasing the dose of warfarin to 500 µg/kg/day.

An INR within the target range was achieved only during the second week when albumin was not required. Albumin infusions were recommenced during the third week and it then proved impossible to raise the INR above 1.4. Anticoagulation was abandoned after 35 days because the INR could not be stabilised despite large doses of warfarin (100-500 µg/kg/day).

Pulmonary embolism is a well recognised complication of nephrotic syndrome. However, in this case thromboemboli from the infected central line is likely as a thrombus was present on the tip of the line when it was removed.

Warfarin is 99% protein bound and acts by inhibiting the synthesis of vitamin K-dependent clotting factors. Inactive meta-

bolic products from the liver and kidney are excreted in the urine and faeces.²

The variations in the INR after albumin infusions may be a result of: (1) an increase in protein bound warfarin excreted in the urine as part of the on-going proteinuria, (2) a reduction in the unbound fraction of warfarin available to the target sites, (3) a larger circulating blood volume, after the infusion of albumin, leading to an increase in liver and kidney perfusion enhancing drug extraction. However, the latter seems unlikely as warfarin has a low hepatic extraction which is independent of flow.³

This case illustrates some of the problems surrounding warfarin treatment that have to be considered when prescribing the drug in nephrotic syndrome.

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'Tarantula eyes'

EDITOR,—Eye injuries caused by tarantula hairs have been reported in at least six American patients (aged 13-24) in ophthalmological journals.¹⁻³ However, the condition is still not widely recognised. We describe a case of eye injury caused by tarantula hairs in a 9 year old boy.

The boy was admitted with itchy generalised fine macular rash, conjunctivitis, and periorbital oedema. He also had severe itching and a gritty sensation in his eyes. The discomfort caused him to rub his eyes vigorously. An allergic aetiology was assumed and he was treated with topical calamine lotion and oral chlorpheniramine. His skin rash largely subsided overnight and the itching in his eyes improved slightly, but he continued to rub his eyes because of the discomfort.

Ophthalmological examination showed injection of the conjunctivas. Several small tarantula hairs were embedding in his cornea at different levels within the stroma of the right eye and in the mid-stroma of the left eye. His visual acuity was 6/18 on the right and 6/6 on the left. On further questioning, he admitted annoying his Chile Rose tarantula before he developed the painful eyes.

The patient was treated with chloramphenicol eye drops four times a day and oral terfenadine twice a day. His visual acuity was back to normal after one week. However, he was still having considerable discomfort in his right eye associated with an anterior uveitis. A course of prednisolone 1% and cyclopentolate 1% eye drops was given. The topical corticosteroid dose was tapered down over the ensuing four months. Subsequent review showed that his uveitis was controlled with the steroid drops, but the tarantula hairs were still embedding in his cornea.

Tarantulas of the family Theraphosidae are becoming increasingly popular as pets. Con-