Figure 3 Full field Ganzfeld electroretinogram (ERG). Upper traces show scotopic ERG elicited by a single white flash in the dark adapted eye: A-wave latency 25.5 ms RE, 25.7 ms LE (normal range 24.4–28.1 ms), amplitude 121.0 μ V RE, 131.0 μ V LE (normal range 56.1–185.2 μ V). B-wave latency 48.0 ms RE, 47.8 ms LE (43.8–53.1 ms) and amplitude 548.0 μ V RE, 616.0 μ V LE (348.0–679.6 μ V). Lower traces show photopic ERG response to a single white flash: A-wave latency 16.6 ms in each eye (normal range 16.5–18.9 ms) and amplitude 27.0 μ V RE, 28.0 μ V LE. B-wave latency 30.7 ms RE, 31.0 ms LE (27.5–31.8 ms) and amplitude 121.0 μ V RE, 151.0 μ V LE (39.1–207.1 μ V). All normal range values represent mean (2 SD).

neither a 'window defect' suggesting depigmentation, nor hypofluorescence, which might suggest an abnormal accumulation of material within retinal pigment epithelium cells. Instead, the mild generalised irregular hyperfluorescence suggests merely a diffuse abnormality of retinal pigment epithelium.

The occurrence of a marbelised fundus in asymptomatic patients is rare. Aish and Dajani have described an Arab Palestinian family with clinical features which appear to closely resemble those of our patient.¹ In this pedigree, the parents were phenotypically normal first cousins. Seven out of 10 of their offspring showed massive invasion of both fundi by bright white or yellow fleck lesions situated behind the retinal blood vessels, and always sparing the macula. Visual findings were normal in all cases. The probable mode of inheritance within this family was autosomal recessive, since both sexes were involved, and the consanguineous parents were unaffected. Krogh *et al* have described an asymptomatic 31-year-old woman with normal visual acuity, with bilateral retinal flecks in the mid periphery of both eyes.² The flecks became more dense in the periphery, where they formed a palisade pattern quite unlike that of our case. Functional testing revealed an absent EOG light rise in one eye but was otherwise normal. More recently, a case of bilateral 'breadcrumb' flecked retinopathy with normal fluorescein angiography and normal electrophysiological findings has been reported in a 9-year-old girl. However, this child also had an idiopathic seizure disorder which had been controlled medically for 6 years, subnormal intelligence, gross motor and developmental delay, and esotropia.³ The size and shape of the retinal flecks in this case are not described in detail, but the published photographs appear to demonstrate a more uniform size and more irregular margins to the flecks than in our patient, with a more linear distribution of flecks and a greater area of normal appearing retina between the flecks.

A marbelised fundus appearance has also been reported as a rare finding in Leber's congenital amaurosis.^{4–6} In this variant,

yellowish lesions are seen deep to the retinal vessels in a periarterial distribution, and there may be associated systemic abnormalities, including medullary cystic renal disease (juvenile nephronophthisis).⁷ The other clinical features and absent ERG response of Leber's amaurosis make confusion with our case unlikely. However, it is interesting to note in such cases that a marbelised fundus may be incidental to visual functional abnormalities.

We suggest that our case represents either a new mutation of the condition described, or possibly an autosomal recessive disorder, since both parents are phenotypically normal.

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Brown's syndrome as a complication of cardiopulmonary resuscitation

EDITOR,—Brown's syndrome is a well recognised ocular motility disorder which may be congenital or acquired. Regardless of aetiology it manifests itself clinically with restriction to both active and passive elevation in adduction, minimal or only slight limitation to elevation in abduction, occasionally a downshoot of the affected eye in adduction and, in more severely affected cases, a primary position hypotropia with an associated abnormal head posture. The head posture consists of a chin-up head position with a face turned away from the affected side or a variable head tilt. Other features less commonly seen are a 'V' pattern resulting from divergence in upgaze and widening of the palpebral fissure on adduction.¹

CASE REPORT

We report a case of acquired Brown's syndrome in a 2-year-old girl without a history of ocular motility defects, who accidentally fell into the family swimming pool and was found cyanotic, face down in the water. Her mother, trained in cardiopulmonary resuscitation, rescued her from the pool and successfully resuscitated her using nasal compression, mouth to mouth ventilation, and cardiac massage. The patient was transferred to the Children's Hospital of Pittsburgh and, after a short period of artificial ventilation, made a full recovery. A computed tomography brain scan did not reveal any abnormality.

After hospitalisation it was noted that the child had developed a mild chin-up head posture. One week later her vision was 20/30 in each eye using Allen figures, she was orthophoric in the primary position of gaze, had a chin-up head posture without head tilt or face turn, and ocular versions revealed limitation of elevation in adduction (Fig 1). There was no evidence of superior oblique muscle overaction or downshoot in adduction. In addition, the right superior rectus muscle did not demonstrate overaction and there was some divergence in upgaze which helped to differentiate this entity from an isolated left inferior oblique paresis. She demonstrated 100 seconds of arc stereoacuity and had normal fusion for both distance and near using the Worth 4 dot test in the primary position of gaze. Magnetic resonance imaging of the orbits was normal and did not reveal any evidence of trochlear disinsertion or swelling. The orbital floor was intact. When the patient was considered sufficiently mature we performed forced duction testing under local anaesthesia which confirmed the diagnosis. The patient was 3 years old at this time. We did not feel it necessary to subject the patient to general anaesthesia when she first presented in order to confirm the diagnosis, particularly in light of her near drowning event.

This patient has been followed for 18 months and the restriction to elevation in adduction has improved significantly. As the patient did not have a significant head tilt and was orthophoric in primary position surgical intervention was not required.

COMMENT

Acquired Brown's syndrome has been reported following traumatic events occurring in the region of the trochlea; these include peribulbar anaesthesia,² orbital surgery,³ orbital roof fracture with superior oblique

Figure 1 Extraocular movements demonstrate restriction of elevation in adduction of the left eye.

tendon entrapment,⁴ and blunt orbital trauma.⁵ We feel that during the resuscitation procedure, the child's mother may have inad-

vertently traumatised the trochlear region while occluding the nasal passages to facilitate the oral passage of air. This is plausible given

the short distance between the nose and trochlear region in a 2-year-old child and the urgency of the situation. To our knowledge this is the first reported case of Brown's syndrome acquired after cardiopulmonary resuscitation and mouth to mouth ventilation.

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