# Retinal haemorrhages and convulsions

S Sandramouli, R Robinson, M Tsaloumas, H E Willshaw

**Abstract**

*Aims*—To evaluate the incidence of retinal haemorrhages after convulsions in children.

*Patients and methods*—All children who required hospital admission after an episode of convulsions were included in the study. Complete neurological and ocular examinations, including ophthalmoscopy, were undertaken within 48 hours of hospital admission.

*Results*—Thirty three children were examined according to the protocol and their seizures were classified by a paediatric neurologist. Despite the fact that some of the children also vomited or underwent cardiopulmonary resuscitation, none of the 33 children developed retinal haemorrhages.

*Conclusions*—Convulsions rarely (if ever) give rise to retinal haemorrhages. The finding of retinal haemorrhages should stimulate a detailed assessment to exclude non-accidental injury, whatever the nature of the associated or antecedent events.

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Retinal haemorrhages in children are an important clinical finding. They are associated with both accidental and non-accidental injuries as well as with a variety of systemic illnesses.[1][2] In the absence of a bleeding disorder, retinal haemorrhages most often result from non-accidental injury.[2][3] Different forms of child abuse may cause retinal haemorrhages, including direct striking of the head, severe shaking causing an acceleration or deceleration injury, and thoracic compression, as may be seen in sexual abuse.[2][4][5]

Events around the time of discovery of the fundus haemorrhages, such as cardiopulmonary resuscitation, or any mechanism involving forced contracture of the chest such as convulsions, crying, or vomiting may be held responsible for the haemorrhages.[1] It is therefore important to determine the likelihood of these events giving rise to the fundus findings in an affected child. Recent publications have addressed the problem of whether cardiopulmonary resuscitation is likely to cause retinal haemorrhages in children,[6–9] but we know of no similar prospective study which has assessed the likelihood that convulsions might cause retinal haemorrhages. We conducted a prospective study to evaluate the incidence, nature, and distribution of retinal haemorrhages in children after a convulsion which required hospital admission.

## Patients and methods

All children admitted to our hospital with a diagnosis of convulsions over a period of four months were included in the study. One child with non-convulsive seizure was also seen, but not included in the analysis. All the children underwent detailed examination within 48 hours of admission after informed consent was obtained from their parents and the supervising medical team. Each child had a detailed history taken to categorise the nature of the seizure and any associated vomiting, cardiopulmonary resuscitation, or injury. A complete neurological examination was performed and the fundi were examined by both direct and indirect ophthalmoscopy. Where the pupil size was inadequate to allow full examination and when neurological observations allowed, pupillary dilatation was carried out using 0.5 or 1% tropicamide eye drops and the fundus was re-examined.

Seizure classification was based on the nature of the seizure and the type of epilepsy. The study was designed to evaluate the type, pattern, distribution, and rate of resolution of haemorrhages, with appropriate follow up in the outpatient department of those children who were noted to have retinal haemorrhages.

## Results

Thirty three children with seizures were examined, including 20 boys (61%) and 13 girls (39%). One child was excluded from the study, having been admitted for investigation of an absence attack and not convulsions. Their ages ranged from 4 months to 14 years with a mean of 46.9 months and a median of 30 months. Thirteen children (41%) were less than 2 years old. Seventeen children (53%) had generalised seizures, 14 (44%) had febrile seizures, and one child (3%) had complex partial seizures (table 1). Among the children with epilepsy, seven had primary epilepsy of varying seizure types, whereas symptomatic epilepsy was noted in 10 (table 2). Three children were admitted in status epilepticus. Associated causes of convulsion are given in table 3.

A history of at least one bout of vomiting was recorded in seven (22%) children, whereas cardiopulmonary resuscitation had to be given in two (6%) children. None of the children with febrile seizures required cardiopulmonary resuscitation. No child examined during the

**Birmingham
Children's Hospital
NHS Trust, Ladywood,
Birmingham**
S Sandramouli
R Robinson
M Tsaloumas
H E Willshaw

Correspondence to:
Mr H E Willshaw,
Birmingham Children's
Hospital NHS Trust,
Ladywood Middleway,
Ladywood, Birmingham
B16 8ET.

*Table 1  Seizure classification*

| Seizure type | No of children (n = 33) |
| --- | --- |
| Tonic-clonic | 12 |
| Generalised | |
|   Tonic | 1 |
|   Clonic | 2 |
|   Absence | 1 |
|   Myoclonic | 2 |
|   Atonic | 0 |
| Complex partial | |
|   Tonic-clonic | 1 |
| Total | 19 |

*Table 2  Type of epilepsy*

| Type of epilepsy | No of children (n = 33) |
| --- | --- |
| Primary | 8 |
| Symptomatic | 10 |
| Cryptogenic | 1 |
| Febrile | 14 |

*Table 3  Associated causes of convulsions*

| Cause of convulsion | No of children (n = 33) |
| --- | --- |
| Infection | 1 |
| Operated neuroblastoma | 1 |
| Lissencephaly | 1 |
| Central nervous system haemangioma | 1 |
| Progressive encephalitic optic atrophy | 1 |
| Cerebral palsy | 1 |
| Developmental delay | 3 |
| Hemiplegia | 1 |
| Road traffic accident | 1 |
| Total | 11 |

study was observed to have retinal haemorrhages within 48 hours of an episode of convulsions.

## Discussion

Forceful contraction of the thoracoabdominal muscles may occur from trauma, cardiopulmonary resuscitation, vomiting, seizures, or spells of coughing.[1] It causes a sudden increase in intrathoracic pressure, with a consequent rise in intracranial pressure, and leads to an increase in retinal venous pressure. These sudden, transient changes in pressure may result in cerebral and retinal haemorrhages.[1 10]

Such a mechanism is theoretically possible after both cardiopulmonary resuscitation and convulsions.[1 2 10 11] (The sagittal venous pressure measured during tonic seizures in status epilepticus has been noted to be three to four times normal.[12]) There have been a number of case reports incriminating both in the generation of retinal haemorrhages.[6 7 11] Evidence for the role of cardiopulmonary resuscitation in the genesis of retinal haemorrhages is sparse and Gayle *et al* concluded that even if cardiopulmonary resuscitation has been administered, retinal haemorrhage should be considered secondary to craniocerebral trauma.[2] This conclusion is supported by necropsy and experimental studies in humans and pigs, respectively.[6–9]

Our study of 32 children with convulsions showed none with retinal haemorrhages. This observation was valid irrespective of the age and presence of secondary causes of epilepsy such as central nervous system hemangioma.

This group includes two children who underwent cardiopulmonary resuscitation and seven children who had vomited.

Statistical analysis of this group of patients is best performed using Hanley's rule of three.[13] If none of *n* patients showed the event about which we are concerned, we can be 95% confident that the chance of this event is at most three in *n* (that is, $3/n$). In other words, the upper confidence limit of a $0/n$ rate is approximately $3/n$ (for $n > 30$). Hence the results of our study are statistically significant, with $p < 0.05$. Therefore, as seems to be the case with cardiopulmonary resuscitation, the chance of a child having retinal haemorrhages solely on the basis of having had a convulsion is unlikely. If fundus haemorrhages are found in such a child, other causes should be sought, including infection, a bleeding disorder or, particularly, non-accidental injury.

After this study was completed, SSM saw an infant admitted to the sick baby unit of the Walsall Manor Hospital after convulsions. The infant was premature, born at 33 weeks by normal vaginal delivery weighing 2100 g. On the 10th day the infant was noted to have recurrent tonic seizures with rapid eye movements. Investigations showed no evidence of septicaemia or metabolic disorder. On the 23rd day the infant was examined with dilated pupils within 24 hours of an attack of convulsive seizure. On indirect ophthalmoscopy, the right fundus was noted to have a pale centred, superficial retinal haemorrhage, close to the upper temporal artery. The haemorrhage was of 1.5–2.0 disc diameter in size and was located about two disc diameters above the disc. The left retina was entirely normal. This haemorrhage cleared within 10 days.

This child may have had a simple neonatal haemorrhage or may have had the haemorrhage as a result of the convulsions. The clinical appearance was more suggestive of a resolving haemorrhage. If the cause was a neonatal haemorrhage, it had taken an unusually long time to clear. If, however, the convulsions were to be implicated as the cause of the retinal haemorrhage, this would confirm earlier suggestions that the retinal venous system in neonates and very young infants is more vulnerable to increased intrathoracic pressure than in older children. In our study the age of the youngest child was 4 months and 41% of the children were less than 2 years old (13 of 32 children). This possibility is currently being examined in a further prospective study.

## Conclusions

In children with retinal haemorrhages, investigation into the possibility of non-accidental injury is essential to safeguard the patient and siblings. When careful assessment does not support a traumatic event, other medical causes of retinal haemorrhage should be considered. Our study suggests that retinal haemorrhages in children are rarely associated with convulsions and that appropriate investigations to exclude non-accidental injury should be undertaken. As the study series did

not include any neonates, a separate study of this high risk group is currently being undertaken.

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