

## SHORT REPORT

## Low incidence of seizures in patients with chronic subdural haematoma

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### Abstract

**A total of 129 patients treated for chronic subdural haematoma were studied retrospectively to evaluate the incidence of seizures. None of 73 patients who were given prophylactic antiepileptic drug treatment developed seizures. Only two of 56 patients not given prophylaxis, developed early postoperative seizures. In these two, surgical technique was thought to be responsible. One patient developed complex partial seizures preoperatively. The incidence of seizures was therefore low, and similar to that previously reported for minor head injury. This study suggests that routine use of antiepileptic prophylaxis is not justified in patients with chronic subdural haematoma caused by minor head injuries, or other causes when there are no additional lesions present on CT scans.**

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Chronic subdural haematoma is caused by head injury in more than 80% of patients,<sup>1-3</sup> and is often preceded by traumatic, low density, subdural fluid collection, or less commonly by a high density acute subdural haematoma.<sup>4</sup> Thus, because patients with chronic subdural haematoma may often have subclinical or apparent cerebral lesions at the time of head injury in addition to the chronic condition itself,<sup>2,3</sup> the possibility of seizures exists. The use of anticonvulsant therapy in these patients has been variable, and dependent upon individual neurosurgeons and hospitals.

We report on the incidence of seizure in our series of 129 patients and discuss the use of prophylactic anticonvulsant treatment.

### Patients and methods

We studied 132 consecutive patients with chronic subdural haematoma who were treated between August 1980 and March 1992. Two patients died of myocardial infarction or intracerebral haemorrhage during early follow up and one patient left the area without treatment. These patients were excluded from the analysis. The remaining

129 patients were analysed retrospectively. For these patients, medical records were checked for the follow up. Patients who had been discharged from outpatient treatment within a year after leaving hospital, were contacted by letter or telephone. A total of 118 patients were followed for a minimum of one year up to five years, whereas follow up of the remaining 11 patients ranged from seven to 11 months after complete resolution of the haematoma.

These included 97 male and 32 female patients, with ages ranging from 12 to 91 years. Approximately half (48.8%) of them were more than 70 years old. One hundred and four patients had a definite past history of head injury, and 13 also had a history of possible head injury. Thirty-four patients were followed, using CT, from the time of injury to the development of chronic subdural haematoma. Half of the 34 head injuries were caused by traffic accidents, and these patients were brought to our hospital because most of their injuries were more severe. CT performed within several days after head injury showed an acute subdural haematoma in five patients and a low density, subdural fluid collection in 29 patients. In these 34 patients, other abnormal lesions present on CT scans included cerebral contusion (four patients), intracerebral haemorrhage (two patients), and acute epidural haematoma (two patients). Lumbar puncture revealed bloody cerebrospinal fluid in two patients. The other 95 patients showed chronic subdural haematoma on their first CT scans without any additional abnormal lesions attributable to the injury.

Treatment in 116 patients was by burr hole and irrigation with warm saline under local anaesthesia, followed by external drainage for 20-48 hours postoperatively. The remaining 13 patients were treated nonsurgically because of mild symptoms, or the absence of symptoms.

Patients were usually given 80-100 mg of intramuscular phenobarbital about 30 minutes before surgery. Until December 1987, 56 of 59 patients received oral anticonvulsant treatment postoperatively. Prophylactic use of anticonvulsant drugs was subsequently discontinued. Seventeen of 70 subsequent patients received anticonvulsant treatment, however, mainly according to the relative severity of the injury. A total of 56 patients

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did not receive oral anticonvulsant medication throughout the whole period of treatment, whereas 49 patients were treated postoperatively or from the time of diagnosis. These 49 patients received anticonvulsant medication for 4–12 months after surgery or after the disappearance of the haematoma space. Twenty-four of the 34 patients who had CT after the injury were given anticonvulsant treatment soon after their injuries, because they were found to have intracranial pathological lesions caused by the injury. Approximately 2 mg/kg phenobarbital was given in 88% of the 73 patients, and the dose was decreased according to age in the elderly.

### Results

Four patients suffered seizures in the present study, none of whom had received anticonvulsant drugs. One of these four patients developed frequent episodes of facial twitching 15 months after surgery, evidently caused by intracerebral haemorrhage due to aplastic anaemia. Three (2.3%) of 129 patients developed seizures associated with chronic subdural haematoma, irrespective of the use of anticonvulsant medication.

Only one patient presented with several episodes of complex partial seizures before surgery, but these symptoms were not seen after evacuation of the haematoma. The incidence of seizures in 105 patients who were not given anticonvulsant prophylaxis before treating the haematoma was 1.0%. Two other patients developed focal seizures during the early postoperative period. These seizures occurred on the side contralateral to the haematoma soon after removal of the catheter used for external drainage. CT scans in these two patients showed no lesion that might have been responsible. In contrast to the other patients, thicker catheters were used. These transient seizures were therefore thought to be associated with the use of this type of catheter, although the mechanisms for the development of seizures remain unclear. In the two cases, no further seizures were

noted during follow up. The incidence of postoperative seizures was therefore 1.7% as a whole, and 3.8% of 53 patients not given postoperative anticonvulsant prophylaxis.

### Discussion

The overall incidence of seizures in patients with chronic subdural haematoma has been previously reported to vary from 9.3% to 17.0% (table).<sup>5-7</sup> This is considerably higher than the numbers we have observed in daily clinical practice. In addition, the reported incidence of postoperative seizures varies widely from 1.0% to 23.4%.<sup>1,5,6,8,9</sup> The reasons why some studies report such high incidences, and the numbers vary so widely, are not clear. One possible explanation may be the use of different operative procedures. Hirakawa *et al*<sup>1</sup> studied 309 patients with chronic subdural haematoma, and found that postoperative seizures were seen in 5.3% of patients treated with burr hole and irrigation, whereas 23.4% of patients treated with craniotomy and capsulectomy had seizures. Their study also showed that the second procedure resulted in more postoperative complications than the first. The reported seizure incidence was also lower in other studies where patients were treated by burr hole and irrigation, or trephine craniotomy,<sup>6,8,9</sup> and these numbers are quite similar to ours, for patients in whom there was no prophylactic anticonvulsant treatment. Operative procedures were miscellaneous in the study of McKissock *et al*<sup>5</sup> and were not known in the study of Luxon and Harrison.<sup>7</sup> In the report, 4% of patients had had epilepsy as a cause of head injury, and loss of consciousness was included as a symptom of epilepsy. Thus, postoperative seizures may not always reflect seizures due to chronic subdural haematoma itself. In either case, the risk of seizures after burr hole removal of haematoma is usually less than 5%.

The incidence of preoperative seizures in the study of Kotwica and Brzezinski<sup>6</sup> was much higher (6.9%), compared with 0.3% in the previous study of Hirakawa *et al*<sup>1</sup> and 1% in our study. Another possible reason for the variation between these reports is that there may be differences in the distribution of head injury severity. A multicentre cooperative study by the Japan Follow up Group of Post-traumatic Epilepsy<sup>10</sup> has shown that the risk of post-traumatic epilepsy is related to the severity of cerebral injuries, and is much higher in patients with intracerebral haemorrhage than with subdural haematoma. D'Alessandro *et al*<sup>11</sup> reported that only an intracerebral haemorrhage and intracerebral plus satellite extracerebral haematoma proved to be significantly associated with post-traumatic epilepsy. The incidence of cerebral contusion or intracerebral haemorrhage among patients with chronic subdural haematoma would therefore have an effect on the frequency of seizures in these patients.

The risk of early seizures after only minor, closed head injury in adults has been reported to be 0.4% to 2.4%.<sup>12-14</sup> Most of the 56

Table Summary of the incidence of seizures in chronic subdural haematoma in previously reported series

Series	No. of patients with seizures/ total cases treated (%)			Operative procedure
	Preoperative	Postoperative	Total	
McKissock <i>et al</i> (1960) <sup>5</sup>			20/216 (9.3)	Miscellaneous
Hirakawa <i>et al</i> (1972) <sup>1</sup>	1/309 (0.3)	7/133 (5.3%) 32/137 (23.4%)		Burr hole Capsulectomy
Luxon & Harrison (1979) <sup>7</sup>			33/194 (17.0)	?
Robinson (1984) <sup>9</sup>		3/133 (2.3%)		Burr hole
Grisoli <i>et al</i> (1988) <sup>8</sup>		1/100 (1.0%)		Trephine
Kotwica and Brzezinski (1991) <sup>6</sup>	9/130 (6.9)	7/130 (5.4%)		Burr hole
Present authors (1992)	1/105 (1.0)*	2/53 (3.8%)† 0/63 (0.0%)‡	16/130 (12.3)	Burr hole Burr hole
			3/129 (2.3)§	

\*Preoperative incidence of seizures in patients without prophylactic medication before treatment of the haematoma.

†Postoperative incidence of seizures in patients without prophylactic medication.

‡Postoperative incidence of seizures in patients with prophylactic medication.

§Thirteen patients who did not undergo surgery are included.

patients who had no prophylactic medication in the current study had minor head injuries. Only two patients in this group developed postoperative early seizures, which was probably caused by the surgical technique. It therefore seems likely that the risk of seizure activity is very low in patients with chronic subdural haematoma caused by minor head injuries. Although it has been stated that the haematoma or the capsule of the haematoma may play an important role in the incidence of epilepsy,<sup>6</sup> we consider that seizures in patients with chronic subdural haematoma are rarely related to the encapsulated haematoma itself, but rather to accompanying cerebral injuries<sup>15</sup> or surgical technique.

None of the 73 patients who were given prophylactic anticonvulsant drugs, including 24 patients with relatively severe head injuries who had serial CT, developed seizures during the period of this study. It is possible that prophylactic medication was beneficial for the prevention of seizures in some patients, especially in those with cerebral contusion or parenchymal haemorrhage. We could not draw firm statistical conclusions from our data. It appears to be inappropriate, however, to give prophylactic anticonvulsant treatment to every patient with chronic subdural haematoma, as the incidence of seizures in chronic subdural haematoma caused by minor head injuries is similar to that seen in minor head injuries. Furthermore, most in chronic subdural haematomas caused by minor head injuries tend to occur in the elderly, who are more vulnerable to the potential side effects of anticonvulsant drugs.

We conclude that the prophylactic use of anticonvulsant medication is unnecessary in patients with chronic subdural haematoma related to minor head injuries, or causes other than head injury that show no additional lesions on CT.

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