Epilepsy and acute traumatic intracranial haematoma

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SYNOPSIS Acute intracranial haematoma significantly increases the incidence of traumatic epilepsy, both early (first week) and late. Early epilepsy can cause diagnostic and management problems, while late epilepsy may produce persisting disability.

A pilot study of traumatic epilepsy after non-missile injuries indicated that acute intracranial haematoma was an important predisposing factor (Jennett and Lewin, 1960). The numbers were small but a much larger series has now been accumulated from various sources, as described in detail elsewhere (Jennett, 1973, 1975). These additional patients were shown not to differ significantly from the original series, which came from an unselected series of head injuries (Table 1). It seems reasonable, therefore, to regard the series as a whole as a valid sample of patients with acute intracranial haematoma. This term was applied if operative drainage was performed within 14 days of injury. Chronic subdural haematoma was excluded from the study, as were birth injuries; there were few infantile haematomas (less than 5% of the series were under 2 years of age). Traumatic epilepsy was diagnosed if the patient had even one fit, but in the event it

TABLE 1

FREQUENCY OF FEATURES IN PATIENTS WITH HAEMATOMA IN ORIGINAL SERIES OF UNSELECTED INJURIES AND IN ADDITIONAL CASES

Feature	Original series 58		Additional cases 362		P
	(No.)	(%)	(No.)	(%)	
< 16 years	9	16	59	16	NS
Extradural	23	40	123	34	NS
PTA > 24 hours	24/42	57	168/346	49	NS
Early epilepsy	13	23	99	27	NS

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TABLE 2
EFFECT OF HAEMATOMA ON INCIDENCE
OF EARLY EPILEPSY

	(No.)	(%)
No haematoma	33/928	4
Intracranial haematoma	112/420	27
P	< 0.001	

was unusual for a patient to have only a single fit. Epilepsy which occurred in the first week was termed early, and if after that late; some patients had both early and late epilepsy. If any of the seizures suffered by a patient had any evidence of focal signs, even at the beginning of a fit, then that patient was regarded as having focal epilepsy; others were classified as suffering from non-focal epilepsy.

EARLY EPILEPSY This was significantly more common after injuries associated with acute intracranial haematoma (Table 2). The incidence was similar in adults and children (including those under five years of age), and whether or not post-traumatic amnesia (PTA) was prolonged or there were focal neurological signs (Table 3). Patients with only an extradural haematoma had a significantly lower epilepsy rate than those with intradural haematomas (Table 4); subdural and intracerebral haematomas were similar. Rather more than half the patients had some fits with a focal component, and about one patient in 10 developed status epilepticus. Early epilepsy associated

TABLE 3

INCIDENCE OF EARLY EPILEPSY AFTER ACUTE INTRACRANIAL HAEMATOMA WITH VARIOUS FEATURES

	Feature				P
	Present		Absent		
	(No.)	(%)	(No)	(%)	
< 5 years	9/28	32	103/392	26	NS
≥ 16 years	95/352	27	17/68	25	NS
PTA > 24 hours	62/210	30	36/136	26	NS
Focal signs	50/175	29	62/245	25	NS

TABLE 4

INCIDENCE OF EARLY EPILEPSY WITH ACUTE HAEMATOMA IN DIFFERENT SITES*

	(No.)	(%)
Extradural only	15/146	10
Intradural	76/219	35
P	< 0.001	
Subdural	58/159	36
Intracerebral	18/60	30
P	NS	

* Excluding patients with haematoma at more than one site.

TABLE 5

TIME AFTER INJURY OF FIRST EARLY FIT WITH ACUTE HAEMATOMA

	First hour		1-24 hours		> 24 hours	
	(No.)	(%)	(No.)	(%)	(No.)	(%)
Haematoma	108	11	23	21	74	69
No haematoma	321	107	119	37	95	30
P	< 0.001		< 0.01		< 0.001	

with haematoma was often delayed for more than 24 hours after injury and this was significantly different from the time of onset of early epilepsy after other types of injury (Table 5). This suggests that the epilepsy is often associated with the development of the haematoma; but in a third of cases epilepsy first appeared only after the haematoma had been evacuated (Table 6).

SIGNIFICANCE OF EARLY EPILEPSY AS SIGN OF

DEVELOPING HAEMATOMA The question is often asked whether the development of epilepsy is in itself important evidence in favour of this complication. Although acute intracranial haematoma is much more consistently associated with other features—in particular, deterioration of the level of consciousness—epilepsy is such a dramatic event that not infrequently this is what draws attention to this complication. In the unselected Oxford series 28% of patients with early epilepsy proved to have an intracranial haematoma; two-thirds of these patients had acute subdural clots, a half of whom developed early

TABLE 6

EARLY EPILEPSY IN RELATION TO OPERATION FOR ACUTE HAEMATOMA

	(No.)	(%)
Only before	50	56
Only after	30	34
Both	9	10
All before	59	66
All after	39	44

epilepsy. Such patients are usually already seriously ill, with focal signs and altered consciousness; the occurrence of epilepsy is therefore an incident of limited diagnostic significance. It is more important to discover how often epilepsy heralds an extradural haematoma, which more often develops after a lucid interval or even when there has been no initial loss of consciousness. Only 10% of 146 patients with extradural haematoma had early epilepsy and in a third this did not occur until after surgical evacuation of the clot. Of more than 400 patients with early epilepsy less than 2% proved to be developing an *extradural* haematoma at the time of their first fit; moreover there were always other signs pointing to this complication. Even among patients who develop early epilepsy after injuries associated with neither initial loss of consciousness nor PTA, a haematoma was found in only a small proportion (8%). It seems reasonable to conclude that the occurrence of a fit is in itself an insufficient reason for strongly suspecting an intracranial haematoma; if there

are no other features pointing to this complication epilepsy does not justify further investigation and certainly not exploration.

LATE EPILEPSY Only patients followed-up for more than a year were considered for this part of the study; they were shown not to differ significantly from the original series (Table 7). Late epilepsy was significantly more common than after other types of injury (Table 8), and the incidence was higher after intradural than extradural haematoma (Table 9). The epilepsy rate

focal component in almost 50% of patients. In cases followed for two years or more after the development of late epilepsy, fits continued at a rate of more than one a month in rather more than a quarter; somewhat more than a third had at least two years free of attacks. However, epilepsy frequently recurred even after such a remission.

TABLE 7
FEATURES OF ORIGINAL HAEMATOMA SERIES
AND OF THOSE FOLLOWED

	Feature				P
	Original series 420		Followed-up 128		
	(No.)	(%)	(No.)	(%)	
< 5 yr	28	7	9	7	NS
> 16 yr	352	84	100	78	NS
PTA > 24 hr	210/382	55	79/126	63	NS
Extradural	146	35	59	48	NS
Focal signs	175	42	73	57	< 0.01
Early epilepsy	112	27	35	27	NS

TABLE 8
EFFECT OF ACUTE HAEMATOMA ON INCIDENCE
OF LATE EPILEPSY

	(No.)	(%)
No haematoma	27/854	3
Acute haematoma	45/128	35
P	< 0.001	

did not differ significantly with various features of the injury (Table 10).

Two-thirds of patients with late epilepsy after haematoma had their first fit in the first year after injury. Subdural haematoma was significantly more common in patients whose post-traumatic fit occurred in the second, third, and fourth weeks after injury than when it happened in the first week or after the end of the first month (Table 11). Late epilepsy after haematoma had a

TABLE 9
INCIDENCE OF LATE EPILEPSY AFTER HAEMATOMAS
IN DIFFERENT SITES*

	(No.)	(%)
Extradural	13/59	22
Intradural	25/56	45
P	< 0.01	
Subdural (acute)	14/33	42
Intracerebral	11/23	48
P	NS	

* Excluding patients with haematoma in more than one site.

TABLE 10
INCIDENCE OF LATE EPILEPSY AFTER HAEMATOMA
WITH DIFFERENT FEATURES

	Feature				P
	Present		Absent		
	(No.)	(%)	(No.)	(%)	
Age < 16 yr	7/28	25	38/100	38	NS
PTA > 24 hr	33/79	42	12/47	26	NS
Focal signs	29/73	39	16/55	29	NS
After early epilepsy	9/32	28	36/96	38	NS

TABLE 11
FREQUENCY OF SUBDURAL HAEMATOMA
IN PATIENTS WITH TRAUMATIC EPILEPSY

Time of first fit (week)	% with subdural haematoma		
	(No.)	(%)	
1	40/273	15	P < 0.001
2-4	13/33	39	
5-8	4/33	12	P < 0.02

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DISCUSSION

Few reports about intracranial haematoma include any reference to epilepsy; in these a clear distinction is seldom drawn between acute and chronic haematoma, and never between early and late epilepsy. Chronic subdural haematoma, evacuated more than two weeks after injury, is such a different clinical and pathological entity that it was judged wise to exclude it from the present analysis. It is late epilepsy which is of significance for the patient himself, because it tends to persist and can cause appreciable disability even when recovery is otherwise satisfactory.

In the wider study of traumatic epilepsy after non-missile injuries (of which this report is a part) patients with acute intracranial haematoma proved to have the highest risk of developing late epilepsy—considerably higher than those with depressed fracture (Jennett, 1973). The incidence of late epilepsy after acute intradural haematoma is strikingly similar to that reported after penetrating missile injuries of the skull, a rate which has remained constant in spite of improved early management of such injuries in successive wars (Jennett, 1975). The brain damage in the two instances is somewhat similar, consisting of cortical laceration; the 40–50%

risk attaching to this presumably reflects the susceptibility or threshold of the population. The importance of knowing that patients who have survived the evacuation of an acute intracranial haematoma have a high risk of late epilepsy is that prophylactic anticonvulsant medication can be considered. Attention has recently been drawn to the possibility of preventing post-traumatic epilepsy by the early institution of such treatment (Rapport and Penry, 1972, 1973).

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