Short report

Psychosis associated with epilepsy: significance of the laterality of the epileptogenic lesion

IRA SHERWIN

From the Neurophysiology Unit, Research Service, ENRM Veterans Hospital, Bedford, Massachusetts, and the Department of Neurology, Harvard Medical School, Boston, Massachusetts

SUMMARY The psychiatric histories in a group of patients who had previously undergone temporal lobectomy for intractable epilepsy, were analysed for the occurrence of psychosis. The data suggest that epileptic patients with left temporal lobe lesions are especially disposed to develop a schizophrenic-like psychosis.

In an earlier study of patients selected for the concordance of medically intractable epilepsy and behaviour disorder, we reported that 15% of the patients demonstrated a schizophrenic-like psychosis at some point in their illness.¹ All of the psychotic patients appeared to have had left temporal lobe epileptogenic lesions (TLEL) based on pneumoencephalographic and electroencephalographic (EEG) findings. It is difficult to establish a relationship between a type of psychiatric illness and the laterality of an epileptogenic lesion, for conventionally recorded EEG, or even depth recorded *interictal* data,^{2,3} are unreliable. We have examined the relative frequency of psychosis in a group of patients with unilateral epileptogenic lesions established by depth recorded *ictal* episodes, so as to justify a temporal lobectomy.

Patients and methods

Details of the evaluation of these patients for possible "seizure-surgery" have been reported elsewhere.⁴ In those patients in whom a unilateral temporal lobe lesion was identified, Dr Paul Crandall performed an *en bloc* anterior temporal lobectomy, which extended only 1 cm more posteriorly in the minor

hemisphere. Only those patients who were diagnosed as psychotic were accepted for the present study, which was retrospective. No attempt to "validate" the psychiatric diagnosis was made.

Results

The composition of the series is shown in the table (part A). Only the 63 cases (part B) warranting temporal lobectomy, were analysed further.

Right temporal lobe lesions predominated ($\chi^2=13.65$, $p<0.001$) in contrast to the opposite finding in our earlier study. The principal selection criteria in that study was the *coexist-*

Table

<i>A: Series composition</i>			
<i>Category</i>	<i>Number of patients</i>		
Lost to follow-up or dead	13		
Cases remaining for analysis	99		
Temporal lobe epileptogenic lesions	90		
Unilateral (lobectomy)	63		
Bilateral (depth electrodes only)	27		
Other epileptogenic lesions	9		
	Total (N = 112)		
<i>B: Laterality and sex</i>			
<i>Sex</i>	<i>Left (N = 17)</i>	<i>Right (N = 46)</i>	<i>Total (N = 63)</i>
Male	13	23	
Female	4	23	
<i>C: Psychosis and laterality</i>			
<i>Lobectomy</i>	<i>Psychotic (N = 7)</i>	<i>Non-psychotic (N = 54)</i>	<i>Total (N = 61)</i>
Right	2	44	
Left	5	10	

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Address for reprint requests: Dr Ira Sherwin, ENRM Veterans Hospital, Bedford, Massachusetts 01730, USA.

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ence of severe behaviour disorder. However, in the epilepsy programme from which the current sample was drawn, a deliberate attempt has been made to exclude patients with a severe behaviour disorder or active psychosis, at the time of intake. This was necessary for the depth electrode studies.

Left and right-sided temporal lobe lesions tended to be unequally distributed amongst the sexes (Fisher Exact Probability=0.053). The Binomial Test (assuming no *a priori* laterality bias) revealed that the left/right difference was not significant for males, but for females it was ($p<0.001$). The under-representation of females with left temporal lobe lesions is what might have been anticipated when psychotic patients were excluded if, as Taylor⁵ has postulated, this is the group most at risk for developing the schizophrenic-like psychosis. Despite this attempted exclusion of patients with active psychosis from the present series, we found seven cases who were diagnosed as having been psychotic. (Two other patients (with left temporal lobectomies) had histories suggestive of a paranoid schizophrenic-like psychosis, but such a diagnosis had not been recorded, so they were not included in the subsequent analysis).

Of these seven cases (mean age at onset of seizures, 10.2 years) six were diagnosed as having had a paranoid or schizophrenic type of psychosis. Of the six, one had undergone a right and five a left temporal lobectomy. Case number seven (right temporal lobectomy) was diagnosed as having had a schizo-affective psychosis with marked depressive features. The relative frequencies of left- and right-sided temporal lobe lesions in the psychotic and non-psychotic groups are shown in part C of the table, and differ significantly (Fisher Exact Probability=0.0077).

Six of these seven cases were rendered seizure free (post-operative follow-up >5 years); and case number seven had only occasional seizures, that is less than four a year. Despite these excellent results, indicating accurate lateralisation of the epileptogenic lesions, the psychiatric picture did not improve substantially in any case. In one patient a full-blown paranoid schizophrenic-like psychosis appeared three years after his successful surgery. In two patients, the psychiatric features appeared to progress despite the relief of their seizures.

Discussion

The finding in the present study that patients with left temporal lobe epileptogenic lesions are

especially disposed to develop a schizophrenic-like psychosis reinforces our own earlier findings¹ and those of Taylor.⁵ Also it is compatible with the finding of a high percentage of sinistrals amongst *psychotic* epileptics,^{6,7} which may indicate a left hemispheric lesion. Men predominated (but not significantly so) in our sample of psychotics. However, because of possible selection factors noted above, this is not necessarily incompatible with the suggestion⁵ that women may be at greater risk.

In addition to a correlation between a schizophrenic-like psychosis and left temporal lobe lesions, Flor-Henry⁸ reported that psychotic *depressions* tended to occur in patients with right temporal lobe lesions. In the present study the only psychotic reaction characterised by depression occurred in one of the patients with a right temporal lobe lesion. Although no conclusions can be drawn from this single case, it indicates the need for additional studies.

The prevalence of psychosis in our sample may have been overestimated or underestimated as a result of our decision to accept the previously-recorded psychiatric diagnoses. However, there is no reason to believe that the accuracy of those diagnoses should have systematically varied as a function of which temporal lobe was ultimately excised. The prevalence of 11% observed in this sample, however, is in remarkable agreement with a recent report⁹ in which three out of 32 "completely lateralised" temporal lobe epileptics, that is 11%, had a schizophrenic-like psychosis (see also ref 10, 13%; ref 11, 14%).

This propensity for patients with left-sided temporal lobe lesions to develop a schizophrenic-like psychosis challenges the current popular view that the psychopathology seen in epileptics is a non-specific reaction to having a stigmatising illness,¹² to chronic anticonvulsant therapy,¹³ or to chronic illness *per se*.¹⁰ In the present study the psychosis associated with epilepsy, deliberately has been referred to as "schizophrenic-like" and not as schizophrenia, which it resembles in several ways. However, even if the two disorders should prove to share some common pathophysiological substrates, there is no reason why they should be identical. Slater *et al*¹⁴ noted that a distinguishing clinical feature in this disorder was the preservation of affect. In our cases other distinguishing features were that the psychosis often was more periodic, and that frequently it did not require the use of major tranquillisers and usually did not require chronic hospitalisation. Similar findings have been noted by others.¹⁵

Two other issues raised by these studies deserve

mention. Falconer¹¹ reported that the schizophrenic-like psychosis appeared to be associated with hamartomatous lesions. We and others¹⁶ have not been able to confirm this association. However, it may be that the more "sophisticated" mental mechanisms responsible for paranoid ideation, which are not operative in infancy may be mobilised in late-onset (hamartomatous), temporal lobe epilepsy.¹⁷ A last point requiring comment was our observation and that of others¹⁵ that the onset of psychosis might follow the surgical elimination of the seizures. It may be that the epilepsy protects against the emergence of a psychosis.¹⁸ However, such a mechanism would neither account for the majority of cases in which the psychosis appears in the course of the seizure disorder, nor for its long latency when appearing after operation. Alternatively it is possible that the psychoses might have developed, in time, had no surgery been performed in these cases.

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