Case Reports

Psychomotor Seizures and Mirror Focus Secondary to Retained Knife Blade in Temporal Lobe

Resolution of Mirror Focus after Extirpation of Primary Lesion

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One of the more difficult problems in planning the surgical management of seizures of the temporal lobe, as well as other focal epilepsies, arises from the phenomena of “mirror focus” and other “secondary spike-discharging foci.”1–7 Falconer and Kennedy4 reported 7 patients with temporal-lobe epilepsy in whom bilaterally independent spike-discharging foci were associated with unilateral lesions of the temporal lobe. The electroencephalographic abnormalities were more pronounced on the side contralateral to the eventually proven organic lesion in 4 of the 7 cases, yet unilateral lobectomy resulted in clinical and electroencephalographic improvement. Subsequently Falconer et al.7 reported 2 additional cases of psychomotor epilepsy in which a presumably epileptogenic but nonspike-discharging organic lesion was distant from the anterior portion of the temporal lobe where the spikes were located by recordings in the scalp. They treated these cases successfully by excision of the organic lesion without temporal lobectomy. These observations suggest that the precise localization of the site of the true organic epileptogenic focus and its excision are essential and that the site of the “spike-discharging focus” is of secondary importance. The following report adds further support to this important concept.

Case History

A 37-year-old right-handed Negro male was admitted to the medical service on Feb. 1, 1960 for evaluation of seizures. In 1944 he had been stabbed in the left temporal region, but suffered no immediate effects of the wound. The handle of the knife was removed later that evening in a physician’s office but, unknown to the patient, a portion of the blade remained embedded in the temporal lobe. In 1957 attacks of frank automatism and “blackouts” began. These attacks occurred on an average of 1 to 8 times per day, but might on occasion recur for as long as a week. Various anticonvulsant medications were tried without success. The attacks varied in type, some being ushered in with olfactory or gustatory hallucinations, others with nausea and abdominal cramps. Automatism and slurred speech occurred with most spells. Chewing movements, urinary incontinence and loss of consciousness were noted only when the attack progressed into a grand-mal pattern. Significant past history included an incident in 1939 when the patient sustained a heavy blow to the occiput with a brick but was not rendered unconscious.

Examination. General physical and neurological findings were normal. Roentgenograms of the skull revealed the blade of a knife projecting medially from the left temporal bone. Spinal fluid was entirely normal and the serum test for venereal disease and lues was negative.

Waking electroencephalogram (Fig. 1) revealed an intermittent slow focus and random spikes in the right temporal leads, and only a single burst of spikes in the left temporal leads. The sleep tracing (Fig. 2), induced with 2½ gr. sodium secobarbital, revealed spikes bilaterally in the temporal leads. These spikes were of higher voltage and greater frequency in the right temporal leads, and usually occurred independently of those on the left. On occasion the spike discharges occurred simultaneously, or nearly so, in the homologous temporal leads. Unlike Falconer’s cases,2 barbiturate-induced fast activity was clearly visible over all recording areas.

Course. Operation was recommended but refused. During the next 7 months the frequency and duration

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Fig. 1. Waking electroencephalogram taken 6½ months prior to operation, showing a slow-wave focus in the right temporal lead. The calibration here is the same as that in all figures of electroencephalograms to follow.

LF, left frontal. LP, left parietal. RF, right frontal. RP, right parietal. LT, left temporal. RT, right temporal. LO, left occipital. RO, right occipital. LAT, left anterior temporal. RAT, right anterior temporal.
of the psychomotor attacks increased but it was only when grand-mal attacks became a more severe problem that the patient was admitted to the neurosurgical service at Syracuse Memorial Hospital.

Examination. On readmission neurological findings and visual fields by perimetry were again normal. A pneumoencephalogram (Figs. 3 and 4) revealed atrophy of the left hippocampus and slight dilatation of that temporal horn.

Operation. On Aug. 31, 1960, under local and, later, light Pentothal anesthesia, a large flap was elevated, leaving an island of bone and dura mater attached to the blade of the knife. The blade was located in the middle and adjacent portion of the inferior temporal gyrus, about 5 cm. behind the temporal tip. Cortical recording (Fig. 5) revealed greatest spike activity anterior to the embedded blade, particularly from the superior and inferior temporal gyri. These sporadic spikes would appear in bursts whenever a small dose of barbiturate was given intravenously to sedate the patient, who by now had become quite combative. This unfortunately precluded observation of the patient’s reaction to cortical stimulation. The anterior 6½ cm. of temporal lobe was resected, including the uncus and anterior portion of the hippocampus. Following this, repeated recording from adjacent temporal, insular and frontal cortex revealed no residual spikes. The blade of the knife is shown in Fig. 6.

Pathologic report on the excised brain described gliosis and hemosiderosis adjacent to the foreign body.

Course. A moderate aphasia cleared after 1 week and the postoperative course was otherwise benign. A waking electroencephalogram 3 and 6 weeks after operation (Fig. 7) showed a low-voltage fast pattern but no spikes. The barbiturate-induced sleep record (Fig. 8) 6 weeks postoperatively, however, revealed the continued presence of random spikes in the right temporal leads. Waking and sleep records obtained 3 months (Fig. 9), 27 months and 41 months after operation revealed neither spikes nor focal slowing.

Psychomotor attacks have not occurred since operation. Single grand-mal convulsions occurred approximately 4 and 12 months postoperatively. Rare “blackouts” or “faints” have continued, usually precipitated by alcoholic intake or exposure to very warm temperature.

Fig. 2. Sleep record taken 6½ months prior to operation, showing frequent spike discharges most marked in the right temporal and right anterior temporal leads, some of which are independent of the smaller left temporal spike discharges. Note the barbiturate-induced fast activity in all leads.

Fig. 3. Left lateral view of pneumoencephalogram, demonstrating the presence of the blade of the knife in the left temporal region.

Discussion

The late development of seizures from retained metallic fragments is of itself not so rare as to warrant a report, but an unusual feature here was the finding of a contralateral dominant homologous spike and slow focus. Both the ipsilateral spike focus and the contralateral spike and slow foci had disappeared by 3 months after extirpation of the primary pathologic process.

The phenomenon of “mirror focus” was first demonstrated experimentally by Pacella et al., following application of alumina cream to the motor cortex of monkeys, and was originally interpreted as a spread from the primary focus. Pope et al. confirmed the above findings and further observed that independent spike discharges could appear in the contralateral homologous area. They postulated the development of a secondary, autonomous, epileptogenic zone resulting from persistent bombardment across the corpus callosum. Utilizing the technique of small chronic circumsections, Eidelberg et al. demonstrated not only the epileptogenesis in the contralateral homotopic area but also augmentation of the local cortical response in the mirror site.

The occasional dominance of spike discharges in the secondary foci over those in the primary focus has been recognized recently both experimentally and clinically. Wada and Corneilus studied chronic cats with superficial focal irritative lesions and demonstrated additional independent spike-discharging foci in many deep structures including basal ganglia, thalamic nuclei, hypothalamus, and limbic system. These
data strongly support the important recommendation made by Meyers et al.\(^7\) that the spike discharge should not be taken as the only index of the primary epileptogenic focus.

Morrell et al.\(^{10,11}\) have done extensive studies on the abnormal physiology of the mirror focus. Later Morrell\(^9\) stressed the importance of early surgical intervention in focal epileptic lesions, for his experimental data and those of others\(^{2,8,14}\) suggested that surgical intervention after secondary foci had been well established, whether dependent or independent, did not alter the outcome for continued paroxysmal discharges. These experimental observations are in contrast to successful results in clinical reports.\(^3,5\)

In the light of both experimental and clinical data, the presence of a predominant and independent spike-discharging focus on the side contralateral to the retained blade of the knife in our case is not entirely novel, although the presence of slow-wave activity in the mirror focus has not been well explained. It is most unlikely that this slow activity represented some undiscovered organic lesion in the mirror focus, as this also disappeared subsequent to operation. The possibility that this slow activity represented dispersion by conduction from other independent spike-discharging foci in deep structures remains entirely

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**Fig. 4.** Frontal view of pneumoencephalogram. Note presence of the blade of the knife on the left with its point immediately adjacent to the temporal horn, and the atrophic change in the left hippocampal area.

**Fig. 5.** Electroccorticogram at the time of operation showing the most active electrodes. A schematic illustration of the position of the blade of the knife as well as the recording electrodes is shown in the insert. Note the spike discharges in the electrodes 13, 14 and 3. The line of amputation is shown by the dotted line which was 6½ cm. from the tip of the temporal lobe.

**Fig. 6.** Blade of knife and attached bone.
speculative at our present state of knowledge, although, if correct, this could account for persistence of generalized convulsions even after cessation of psychomotor attacks. In any event, pneumoencephalographic demonstration of an atrophic lesion in the temporal lobe ipsilateral to the blade of the knife clearly pointed to the site of the primary epileptogenic focus. This does not exclude the possibility that some of the psychomotor attacks might have originated from the paroxysmal discharges occurring in the mirror focus. Nevertheless the successful result here together with those of Falconer et al.,3–5 justify surgical attack on the organic lesion regardless of the site of predominance or bilaterality of electrical paroxysmal activity.

Summary

A case is recorded of psychomotor and grandmal seizures occurring as a complication of the retained blade of a knife in the left temporal lobe. The electroencephalogram revealed ipsilateral spikes and a contralateral mirror and independent focus of spike and slow activity. The ipsilateral and contralateral spikes and slow abnormalities disappeared following excision of the left temporal lobe and the retained blade therein. Psychomotor seizures subsided promptly, occasional grand-mal convulsions continued for 1 year, and rare "faints" have continued. Possible mechanisms for these phenomena are discussed in relation to previously reported experimental and clinical data. Surgical intervention at the site of the organic lesion even in the presence of electrically predominant secondary foci is recommended.

Reference


