I agree with Dr. Pincus that prison studies are a reasonable way of investigating any association between fits and violent behavior because it is possible to match epileptics and nonepileptics in terms of many other important social and psychological variables. Therefore, I am surprised that he did not emphasize that when this was done in my small study the epileptics did not prove to have more violence in their histories.

Dr. Berger and co-workers⁶ allude to the possible political overtones of giving men medication to control their violence. Here, I also agree with Dr. Pincus that they misrepresent his case, because he is only talking about ordinary voluntary medication, not compulsory treatment. However, it does seem to me that Dr. Pincus is again guilty of loose thinking. Benefits gained from taking anticonvulsants do not prove that the patient suffers from epilepsy. Most anticonvulsants are also sedatives; maybe the patients benefit from sedation. Double-blind control studies are required.

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Reply from the Author: Dr. Gunn and I agree that "automatism" is difficult to define. As he explains above, it was for this reason that he examined his "data in terms of the time relationship between the violence and any epileptic phenomena." This is fair enough, except that one is not entirely clear about what he means by "any epileptic phenomena." Undefined phrases like "automatic crimes in the sense usually implied in forensic textbooks" are used in his paper as well. At no time does he state how many of the classical features of automatisms must be present to justify labeling particular behavior "automatism."

The fact that he has not defined what is, perhaps, undefinable is not as serious as his persistent failure to realize that he has not done so.

What of the "time relationship?" Having properly used this as one criterion for identifying automatic violent behavior, he accuses me of using it. He seems to claim that the time relationship between antisocial behavior and ictus is unimportant. He cannot have it both

ways. He feels that I have been too loose in quoting him, as only one of his criminals demonstrated violence that could have been ictal. Dr. Gunn provided synopses of the behavior of 14 patients in his article:¹ assault, rape, a raid, violent armed robbery, hitting a man with a jack, strangling a wife, arson, attempted murder and rape, dangerously aggressive behavior, "a series of dangerously aggressive behaviors," and threatening with a gun were acts encountered in 10. The first five acts mentioned were committed by men within 12 hours of a "fit."

Perhaps I have misunderstood his article, but these acts are considered violent in New England, and their chronological association with "a fit" is certainly suspicious and was considered so by him; that, presumably, is why *he* drew attention to the association.

If a violent act or a series of such acts is committed by an epileptic during a period of confusion, is followed by lassitude, is associated with EEG abnormalities, is associated with a generalized seizure, and does not recur during a trial with anticonvulsant therapy, the evidence of its ictal nature would be quite strong, though perhaps not totally conclusive. If such cases exist, it would be difficult to support the contention that ictal violence never occurs. Perhaps the behavior in such a case is more a function of the confusional state, akin to intoxication, caused by the seizure rather than a direct result of the epileptic firing of a specific aggregate of neurons. Still, the association of epilepsy and violence would clearly exist.

Dr. Gunn seems to confuse this ictal violence with nonictal violence in epileptics that could relate to an "interictal" state of reduced responsibility. To determine if such a phenomenon could exist, one can begin by attempting to discover if the prevalence of seizures, especially psychomotor seizures, is great in a violent population. Obviously other theories could explain such an association, as Dr. Gunn suggests with his illuminating parable concerning the English water troubles. Before explaining the association, though, it would be proper to determine if it exists at all. As I pointed out in my article,² there is disagreement on this point. Dr. Gunn's finding of only 6 epileptics in 153 (4%) men imprisoned for violence is low and suggests that his criteria for diagnosing epilepsy or his data base are different from those of others addressing the same issue. Mark and Ervin³ diagnosed epilepsy in 10% of 400 violent prisoners, 50% of whom had some historical evidence suggesting psychomotor seizures and one-third of whom had EEG abnormalities. In our series, 10% of 78 violent prisoners had experienced generalized motor seizures and 24% were considered to have had psychomotor spells. Dr. Gunn's "small study" in a prison population may have thus compared epileptics with other epileptics.

I am gratified that Dr. Gunn agrees with the specific statement in my article about the need for double-blind studies with blood levels to determine if benefits can be derived from anticonvulsants. If benefits are found, a comparison with nonspecific sedatives would surely be worthwhile, but at conventional therapeutic blood levels, phenytoin and carbamazepine are not very sedating and the experience with other sedating drugs,

such as alcohol, has suggested that sedatives precipitate violence rather than the reverse.

Ultimately, I believe we will find that repeated violence can be related to many factors; epilepsy, psychosis, and confusional states associated with epilepsy will account for some, not all. It seems as foolish at this time to pretend that epilepsy cannot ever give rise to violence as to suggest that I indicated that all violence is caused by epilepsy. If I am not mistaken, Dr. Gunn did not make the former claim. Our basic disagreement, if there be one, quibbling aside, is in how frequently epilepsy and violence are associated. Further studies may throw light on this, and to these both of us seem committed.

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Correction

"A clinical and electrophysiological evaluation of myoclonus" by John J. Kelly, Jr., Frank W. Sharbrough, and Jasper R. Daube, May 1981. The illustration on page 586 is figure 5 and should carry the caption printed as part of the figure in page 587. The illustration on page 587 is figure 4 and should carry the caption from page 586.

Correction

"Interferon responses of leukocytes in multiple sclerosis" by P.A. Neighbour, A.E., Miller, and B.R. Bloom, May 1981, pp. 561-6. The correct version of table 3 follows:

Table 3. Sequential measles-induced leukocyte interferon responses of normal and MS patient donors.

Donor	Normal			Donor	MS patients		
	0	Months 1	6		0	Months 1	6
1	2.0*	2.0	2.0	1 (CP)†	1.0	1.0	1.5
2	2.0	2.5	1.5	2 (CP)	1.0	1.25	1.0
3	1.5	2.0	2.0	3 (CP)	0.5	0.5	0
4	3.0	2.0	2.25	4 (CP)	1.0	1.5	1.5
5	2.25	2.25	2.75	5 (R)	2.0	2.5	2.0
6	2.0	2.5	3.0	6 (R)	0	1.0	1.0
Mean	2.13 ± 0.5‡	2.20 ± 0.2	2.25 ± 0.5	Mean	0.92 ± 0.7	1.29 ± 0.7	1.17 ± 0.7

* = IFN response expressed in log₁₀ units.
† = Clinical stage; CP = chronic progressive; R = remission.
‡ = Geometric mean IFN response ± standard deviation.

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