We did not attempt to measure concentrations of diazepam in the blood, but previous studies (*7*) have shown that a single 10-mg oral dose will, after 1 to 2 hours, produce concentrations similar to those in patients following a typical dosage regimen of 5 mg, three times daily. This does not imply that short- and long-term diazepam effects will necessarily be the same because of equivalent blood concentrations, nor that healthy volunteers will react like patients in any case. We cannot conclude from our results that diazepam will render all drivers, and particularly habitual diazepam users, unable to operate a motor vehicle safely. Patients possessing less driving skill than our subjects, however, might be expected to react even more adversely when beginning diazepam therapy. If they eventually adapt to long-term treatment in ways reducing the diazepam effect upon driving performance, their impairment would pass. Until this can be demonstrated, however, it would be prudent to assume that many diazepam users are impaired to some degree.

The measured impairment was confined to a loss of the subjects' ability to control the lateral position of the vehicle during high-speed travel on straight roads. It was apparent for most subjects in conditions D-10, but to widely different degrees. In two subjects, and possibly three, impairment reached levels that might rightfully be called dangerous. Their standard deviation of lateral position exceeded that associated with the containment of lateral movement within lane boundaries [about 35 cm (*8*)], and their movement extended into adjacent lane and shoulder areas. Because these excursions appeared involuntary, one might assume that their control ability had diminished below that required to operate safely on normal roads. Volitional effort, which allowed the subjects to comply with speed instructions, was apparently insufficient to overcome the effect of diazepam on lateral variability.

The correlation between changes in lateral variability from control conditions to D-10 and M may provide some clue about the mechanism of the diazepam effect. Performance changes in both cases were accompanied by a corresponding drop in subjective arousal. Those individuals whose performance deteriorated with the normal loss of arousal that accompanies prolonged wakefulness showed even greater impairment accompanying loss of arousal after the 10-mg diazepam treatment. The lability of the arousal process might therefore be the individual mitigating factor that determines the drug's effect on driving performance. If so, one would expect to find similarly adverse effects of diazepam on driving performance in all situations characterized by low task demands and monotony but perhaps not under more challenging and stimulating circumstances.

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6. This third subject allowed the vehicle's standard deviation of lateral position to reach 45 cm once near the end of his D-10 condition. He thereupon chose to terminate the experiment, saying that he could no longer operate safely. Although informed beforehand of this possibility and encouraged to exercise the same option, both of the other extreme subjects continued driving until the scheduled end of D-10, in spite of poorer performance.
7. S. F. Garattini *et al.*, in *Biological Effects of Drugs in Relation to Their Plasma Concentrations*, D. S. Davies and B. N. C. Prichard, Eds. (Macmillan, London, 1973).
8. Assuming a Gaussian lateral position distribution, nearly all variability would be contained within a range of 6 S.D. (plus vehicle width of 150 cm). Thus, when S.D. = 35 cm, full lateral range would be about 360 cm, the lane width.
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Electroencephalogram Tests for Brain Dysfunction: A Question of Validity

Ahn *et al.* reported (*1*) that abnormal electroencephalogram (EEG) patterns are much more common in abnormal children than in normal children, and concluded that "Measurement of these EEG parameters may offer a brief, reliable, and economic method for rapid examination of children who, because of consistent behavioral problems or learning difficulties, are considered at risk for brain dysfunction or disorder." However, the data reported by Ahn *et al.* do not support their proposed clinical application of EEG technology.

First, it is necessary to examine the normal and abnormal groups compared by Ahn *et al.* (*1*). Group 1 was composed of U.S. children of normal intelligence and school achievement. Group 2 was composed of Barbados children, also of normal intelligence and achievement. Group 3 was composed of children examined in a pediatric neurology service, with intelligence and school achievement information unavailable. Group 4 was composed of children with IQ's between 65 and 84 and Wide Range Achievement Test scores below 90 in language or arithmetic skills or both. Group 5 was composed of children with IQ's above 85 but with below-normal school achievement by the same definition as group 4. Thus EEG data were examined for two groups of normal children, one group of neurological patients, one group that could as well be called dull-normal as learning disabled, and one group of learning-disabled children.

By their more stringent criterion of abnormality (two or more EEG parameters different at $P < .01$ from the developmental norm), Ahn *et al.* (*1*) find that 4 percent of group 1, 2 percent of group 2, 48 percent of group 3, 46 percent of group 4, and 47 percent of group 5 children have abnormal EEG records.

These data do not indicate the validity of the EEG test for discriminating between children with and without brain dysfunction or disorder. Validation of a test of brain dysfunction would require showing a higher proportion of abnormal EEG's in a group known to have a higher rate of brain dysfunction than in a group known to have a lower rate of brain damage (*2*). But Ahn *et al.* (*1*) do not present any independent evidence that rates of brain damage are higher in their three abnormal groups than in their two normal groups. For example, it would be helpful to know what proportion of the children in group 3 were having neurological problems, and what the reason is for believing that the children in group 4 have brain dysfunction of any kind.

More troublesome still is that the three abnormal groups show almost exactly the same percentage of abnormal EEG's

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(48, 46, and 47 percent). It seems unlikely that the rate of brain dysfunction would be the same in neurological patients, dull-normal, and learning disabled; indeed it seems likely that brain disorder would be more common in the neurological patients than in the other two abnormal groups. But if groups differ in the base rate of some condition, then a valid test for that condition must show different rates in these groups (*2*). In other words, the constant proportion of abnormal EEG's in the three different abnormal groups leaves us with two possibilities: either the rate of brain disorder is equal in these three groups or the EEG test has no validity for detecting brain disorder.

It might be argued that the data do at least support the validity of the EEG test for distinguishing normal from abnormal children, where *abnormal* now designates a behavioral category broad enough to comprehend neurological patients, dull-normals, and the learning disabled. The meaning and usefulness of this new combined category remain to be established.

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McCauley and Ciesielski (*1*) question the validity of neurometric evaluation of the EEG for discriminating between children with and without brain dysfunction. Their reservation is based on the lack of evidence for differential incidence among children at risk for brain dysfunction because of specific learning disabilities (SLD), learning disabilities (LD), or the presence of various neurological symptoms (NEURO).

The purpose of our report (*2*) was simply to show that significant (abnormal) values of the 32 univariate features were more common among at-risk than normally functioning healthy children. Although the incidence of significant univariate features was almost identical in our three at-risk groups, clear differences among them can be demonstrated by multivariate techniques. One way to show this is to compute the Mahalanobis distance (*3*) across various subsets of the 32 features. This yields a multivariate estimate of abnormality which corrects for intercorrelations among the selected univariate features. Such multivariate features consistently yield an incidence of abnormality at chance levels for normal children, somewhat higher (two to three times chance) for SLD children, substantially higher (four to six times chance) for LD children, and very much higher (eight to twelve times chance) for the NEURO group.

Table 1. Classification of groups of normal and at-risk children by a multiple discriminant function using neurometric EEG features.

Group	*N*	Percentage classified as	
		Normal	Abnormal
	Training set		
Normal	153	82	18
At risk*	286	37	63
SLD	79	48	52
LD	69	39	61
NEURO	138	23	77
	Independent replication		
Normal	153	89	11
At risk*	286	38	62
SLD	79	54	46
LD	69	39	61
NEURO	138	30	70

*"At risk" is the sum of the children in the SLD, LD, and NEURO groups.

Further, one can see the differences among the three groups by computing a multiple discriminant function using such features. We did this computation with a split-half "training set" consisting of 153 members of the group of normal children, 138 members of the neurological at-risk patients, 69 members of the "dull-normal" learning disabled group (LD), and 79 members of the learning disabled group with normal intelligence (SLD). The accuracy of classifying the children according to the discriminant function constructed on the training set was then independently replicated by using the second split-half of each group. The results of these computations are shown in Table 1.

In the training set, most of the normal children were indentified as such on the basis of the classification rules derived from neurometric EEG features. The proportion of children classified as abnormal increased steadily from SLD to LD to NEURO. The independent replication of these results in the second split-half test set was excellent.

Most of the children in the NEURO group in the previous report (*2*) and the two NEURO subgroups referred to in Table 1 were diagnosed as suffering from neurological disorders or systemic diseases affecting brain function. It is of interest that the percentage of patients in the NEURO group classified as abnormal varied greatly in subgroups with different neurological diagnoses (*4*).

These findings thus support the validity of neurometric evaluations as an aid to identification of children with consistent behavior or cognitive problems who have brain dysfunction. Positive neurometric findings in normally functioning asymptomatic children should be regarded as probable false positives. However, if a child with consistent behavioral or cognitive problems displays positive neurometric findings, it seems reasonable to suggest that brain dysfunctions should be considered a more plausible explanation for those problems than psychosocial factors.

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