normal children (eg, the work of Needleman et al[3] on lead poisoning) and the retarded (as in trisomy 21, in which language skills are most impaired). The reason is that, of cognitive abilities, language skills would appear to be the most sensitive to CNS insult, and therefore, as the problem is studied more deeply, more instances of language delay as a final common pathway will be seen. In exploring possible etiologies, language delay should not call to mind specifically prenatal alcohol abuse, but rather any of the possible pre-, peri-, or postnatal CNS insults.

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In Reply.—

We thank Miller, Valente, and Farber for the interest they show in our report. All three letters raise a basic question—that of what implications can be drawn from diagnostic labels. Thus, while certain diagnostic labels do present implications about etiology, natural course and response to treatment, others, unfortunately, do not, and serve, more appropriately, as tools that only describe a set of developmental or physical findings. For example, while the term cerebral gigantism describes a certain group of children who seem to share characteristics, it would not be correct to invoke cerebral gigantism as an explanation or etiology for the overgrowth that is seen in these patients. We believe that a discussion of these issues is instructive and are pleased by the opportunity to elaborate on what we consider to be basic issues in developmental pediatrics.

First, we point out that the measurements reported for patient J.E. represent an obvious clerical error for which we apologize. Thus, for case 1, height was obtained at age 5 10/12 and was 114 cm, placing him at the 50th percentile for age; his weight at this time (21 kg) was also at the 50th percentile as we reported. These findings were confirmed at later visits (age 6 2/12, height 116 cm, weight 21 kg; age 6 8/12, height 121.4 cm, weight 22 kg; age 8 3/12, height 123 cm, weight 23.5 kg) when height and weight were each found to be at the 50th percentile for age or below. Case 2 was also seen at age 7 8/12 years when his height was measured at 125 cm (50th percentile) and weight 27.7 kg (75th percentile). Thus, while there was an error made in transcribing the measurements for case 1, the percentiles were accurate.

The two children discussed in our report represented instances of a disturbance in several areas of development, apparently related to prenatal exposure to ethanol. The aspects of development that were most strikingly impaired were the cognitive/linguistic/pragmatic systems, and these were thus given most attention in our report. Valente suggests that both boys suffer from autism secondary to fetal alcohol syndrome (FAS). Miller believes the two children's problems should be diagnosed as cerebral gigantism, and Farber suggests that the children's language difficulties be viewed as delays; he cautions that while language delay may be one of the characteristics of FAS, it is not a marker that is specific to FAS.

Cerebral gigantism is a poorly defined syndrome of unknown etiology that is characterized by excessive rapid growth, acromegalic features and mental deficiency.[1] Inasmuch as our patients did not demonstrate increased growth velocity, acromegalic features, or retardation, quite obviously this diagnosis is not tenable. However, Miller's affinity for the diagnosis of cerebral gigantism in these two patients raises an important issue and one that we might add, parenthetically, was a major impetus for our report—the need for precision in both the description and the diagnosis of children with disorders of cognition and behavior. Although we are often impeccably precise in our descriptions of chromosomal, biochemical and physical characteristics of children, the fine details of behavioral, cognitive, and linguistic features are often overlooked, ignored, or lumped together under the broad rubric of "developmental disabilities," as if this were sufficient to both characterize a child's behavioral and intellectual/psycholinguistic functioning and to make accurate developmental prognoses. Although the diagnosis of neuropsychiatric disorders in children is a fledgling area, there do exist diagnostic criteria for these disorders so that they may be distinguished, one from the other. The physician grows to recognize the distinctions among each of the developmental disorders, and to appreciate the emptiness of terms such as "developmental disabilities," especially when description and explanation are desired instead of merely searching for a label. In this connection, the main points of our paper were to provide a detailed and comprehensive picture of the language and cognitive disabilities present in our patients, and, further, to provide the primary care giver with a schema for evaluating such patients in order that a specific diagnosis rather than a vague reference to a developmental disability might be the result of the evaluation. Miller chooses to pass over this information and instead to refer to both children as having "developmental disabilities"; he then equates the resulting clinical picture with one of CNS functioning in Sotos' syndrome, a condition that is quite unlike that shown by our two patients. Our patients displayed marked hypervigilance, distractibility, and cognitive confusion manifest as anxiety and behavioral disorganization, features not prominent in cerebral gigantism. We should also like to point out, that although the children in our report had large heads, we noted that computed tomography (CT) scans were normal; this is in contrast to reported cases of Sotos' syndrome in which pneumoencephalography reveals hydrocephalus; similarly, Sotos reported widely spaced eyes, while we noted the inter inner canthal distance to be normal.[1] Thus, not only are the bahavioral and physical characteristics of these children inconsistent with Miller's suggestion, but such reliance on phenotypic characteristics belies a sci-

entific precision that simply does not exist at the present time. Such phenotypic entities as downward slanting eyes, palpebral fissures, and highly arched palate are poorly defined and lack good normative data, and judgements about their normalcy are as often subjective as they are objective. To be complete, we should also point out that both hydrocephalus and downward slanting eyes have been reported for fetal alcohol syndrome as well as for cerebral gigantism.[2] Could ethanol teratogenicity be considered an etiology for Sotos' syndrome?

Farber's comments about language delay as a common finding in several developmental disorders is, of course, quite correct. However, his utilization of the label "language delay" suffers from the same difficulties of imprecision and vagueness that we discussed in relation to the term "developmental disabilities." Indeed, the two boys we reported were not suffering from a simple language delay. Rather, both showed very unusual speech and language development that clearly sets them apart from the child with language delay; this pattern is reported by us, apparently for the first time, precisely because of its uniqueness. However, we would be remiss if we did not point out that many of the disorders heretofore described as language delay reflect more a tendency to simply classify all disturbances of language as a language delay rather than focusing on the particular details of abnormal language acquisition patterns. We cannot stress enough the need to give the same careful attention to the evaluation of the disturbance of language and behavior in children as is given to the delineation of specific chromosomal and biochemical disorders. If this were to be the case, we certainly would no longer classify large groups of children as having language delay, just as we would not feel it correct to label a child as having a "chromosomal abnormality" without then going further to describe precisely what that abnormality was.

Valente suggests a diagnosis of autism, and in fact, both boys showed some features of autism and these were noted in the article. Neither boy, however, had a developmental history marked by a pervasive lack of responsiveness to others, or a pattern of social withdrawal. Nor did either show, at the time of evaluation or since, the social aloofness of the child with autism, or the repetitive and stereotyped movements one finds so often in autism. Continued observations of both boys made since submission of the report, confirm that a diagnosis that captures the linguistic and conceptual disturbances is more appropriate than one which overemphasizes the system of social attachment and relationships alone. Our visits to their school programs, and our observations of their home behavior, provided us with a rich body of information that excluded such a diagnosis and more strongly suggested the role of linguistic/conceptual disorganization and confusion in the emergence of their disorder.

Most of the points Valente makes about these boys are relevant only if his diagnostic conclusions, based on our descriptions and the implications he drew from these descriptions, are accepted. We assure Valente that we would be very pleased to discover still another biologic cause for autism that can be added to the list (eg, phenylketonuria, maternal rubella, meningitis). It appears to us, however, that we can, instead, describe in more detail the sorts of behavioral and learning problems FAS children are likely to display; in our opinion, autism is not one of them.

Finally, Valente argues that the recommendations we made for early screening and intervention are not warranted, since the prognosis for children with autism is poor; indeed, he comments that "These children never outgrow their social and emotional handicaps." A small portion, perhaps 10% to 15% of children with infantile autism do make progress that allows them at least semi-independent living arrangements as adults.[3] Our suggestion that the prognosis for the children we reported could be altered has been confirmed by follow-up evaluations; both boys are making steady gains in all aspects of development (including social development) as a result, we think, of special educational attention.

Our response to the comments on our report can best be described as bringing to mind the parable of the blind men and the elephant. A very detailed picture of the behavioral and linguistic functioning is provided, and yet, three pediatricians offer alternate diagnoses, diagnoses that conflict both with our own and with each of the others. The utilization of the labels "developmental disability" and "language delay" as diagnostic terms, more often than not, represent diagnosis of exclusion denoting that either development or language acquisition has not been "normal." Such general and exclusionary diagnostic terms are valid only when more precise and meaningful diagnoses are lacking and this was true for many years in this area of pediatrics. However, more recently, there has evolved a large body of research, reflecting the work of the many disciplines that touch on the development of children, that provides both the theoretic and pragmatic information that serves as prerequisite to the formulation of specific and meaningful diagnoses. Indeed, the developmental pediatrician has emerged to serve as the nexus by which the most recent and relevant findings from such areas as developmental psychology, linguistics, and developmental neurobiology are incorporated into clinical practice. Thus, the thrust of our work and that of other developmental pediatricians has been to begin to provide the structure for a diagnostic process that allows for the differentiation of the multiple disorders affecting learning and behavior. While we have provided a schema for the evaluation of language and behavior in children born to alcoholic mothers, such an approach may serve as a prototype of a diagnostic process that pediatricians may use in evaluating children with other cognitive and/or behavioral disturbances. Although such a process is often tedious and time consuming, its utilization will result in a more precise and ultimately more meaningful diagnosis.

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Phenobarbital and Cognitive Function

To the Editor.—

The article by Wolf et al[1] describes the performance of children on a battery of psychometric tasks. The study is an important one since it attempts to evaluate the effects of phenobarbital on cognitive abilities of preschool aged children. This topic has been neglected even though phenobarbital has been freely administered to children for prophylaxis against febrile seizures.

The authors state that no impairment of cognitive function was observed in children who received daily phenobarbital for a period of years. It is easy for the reader to conclude, therefore, that phenobarbital given in the manner described does not affect cognitive functions. However, the authors do not discuss an alternative hypothesis, namely, that the measures utilized were not sufficiently sensitive to detect cognitive disturbances.

As the authors themselves state, phenobarbital administration often produces behavioral disturbances in children. In an earlier report from this project, they reported that 42% of all children receiving phenobarbital exhibited behavior disturbances according to the parents.[2] Continuation of the medication was based upon parental acceptance of the behavioral changes. The implication of the present study is that the behavioral effects of phenobarbital are not translated into cognitive disturbances, a hazardous assumption.

The authors utilize the Wechsler Preschool and Primary Scale of Intelligence (WPPSI) and two tests of problem solving abilities, the Matching Familiar Figures Test (MFFT) and the Children's Embedded Figures Test (CEFT), to assess cognition. It should be recognized that measures of global intelligence such as the WPPSI have not been found to be sensitive to the presence of hyperactive behavior disorders (attention deficit disorders) such as those associated with phenobarbital administration. In a study conducted by our group, the version of the Wechsler scale for older children (WISC) was not found to be sensitive to effects produced during withdrawal of phenobarbital in epileptic children.[3]

The use of the MFFT and the CEFT is uncertain in a group of children whose mean age is <5 years. Most of the normative data available and reports on the use of these tasks involve older children, usually aged 6 to 12 years or in the first six grades of school. The authors describe a testing session of 2½ hours. Even with the 20-minute break noted, this is a formidable task for any small child, especially those <5 years of age. The authors report the order of test administration to be uniform; if this is the order in which the results are reported, the subjects were carrying out the problem solving tasks at the end of the session when they were likely to be fatigued and restless.

The results on the MFFT suggest difficulties with the tasks. The mean time of 5+ seconds reported indicates that the children were responding very quickly to the test items; it is difficult for even older children to respond effectively in this short time period (normative mean time for grade 1 is 11.7 seconds). The mean number of errors reported to be made by the children to all items (2+ errors) must be in error itself; average mean errors for grade 1 children are in the order of 15 errors.[4]

We believe that the study by Wolf and co-workers should not be used to conclude that prolonged phenobarbital administration does not affect cognitive development. Failure to disprove a null hypothesis does not establish the truth of the hypothesis. The concern remains that children whose behavior is altered during their formative developmental years are at risk for cognitive disturbances.

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In Reply.—

Much anxiety has been engendered in clinicians and parents by reports of serious brain damage due to phenobarbital given to young rodents in doses far higher than those clinically used for children.[1,2] Our study, and that of Camfield et al[3] better enables parents and pediatricians to evaluate the degree of risk to cognitive development with continuous phenobarbital at conventionally used dosage. We would welcome the publication of any of Schain's data, using whichever tests he prefers, which might give further information about the effect of both phenobarbital and febrile seizures on intellectual development in children. It is possible, as Schain and Dreisbach speculate, that the tests used in our study may not have been sufficiently sensitive to detect some disturbances of intellectual function. There may indeed be other tests that would have revealed cognitive abnormalities in the treated or comparison group, or both, or neither. We chose one standard intelligence test, the Wechsler Preschool and Primary Scale of Intelligence (WPPSI), and two tests of attention and problem solving ability, the Matching Familiar Figures Test (MFFT) and the Children's Embedded Figures test. Originally this project was to have been done in collaboration with experts at a highly esteemed local university, and this

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