# Letters

## Oil Resource Estimates

The National Academy of Sciences' Committee on Mineral Resources and the Environment has estimated (*1*) the amount of U.S. oil yet to be discovered and produced as 113 billion barrels (News and Comment, 28 Feb., p. 723), considerably below the U.S. Geological Survey's 1974 estimates of 200 to 400 billion barrels. They conclude that the Geological Survey used misleading arithmetic and also imply that the goals of Project Independence are unlikely to be met. Acceptance of these conclusions is resulting in a clamor for removing financial incentives that would lead to the production of new oil and gas, as well as for a government-financed crash program for synthetic fuels based on coal.

I believe that the committee's result is based on an uncritical acceptance of mathematical curve fitting, rather than on geologic and economic reasoning. Furthermore, their result—even if correct—should not require drastic changes in energy policy (*2*).

Their attack is focused on the Geological Survey's methodology, according to which it is assumed that unexplored sedimentary rock contains about as much oil as the same volume of average drilled-up rock, with the ratio $R$ assumed to lie between 0.5 and 1.0. M. K. Hubbert, however, claims to have derived a value for $R$ of only 0.1 (*3*). He presents a histogram (*3*; *4*, p. 2223) of U.S. oil discoveries, $dQ/dh$, in barrels of oil per foot drilled, against cumulative exploratory footage and proceeds to approximate it by means of a negative-exponential curve. Such a curve extrapolates rapidly to negligible values of $dQ/dh$. The area under the curve then gives a rather low value for the ultimate amount of oil, $Q_\infty$, to be discovered by even unlimited drilling (*5*).

A glance at the histogram, however, reveals a rather different situation: $dQ/dh$ for the period from 1860 to World War II is about 200 barrels per foot drilled, followed by a nearly constant value of 35 barrels per foot since 1950. It is likely that the histogram reflects changes in geologic techniques and economics, rather than a uniform trend. Oil exploration started with the discovery of natural seeps and with the recognition of prominent anticlines, and little exploratory drilling was required. Later, seismic techniques located other anticlines and structural traps. Presently, only extensive drilling can uncover stratigraphic traps. There is no guarantee that the curve will decrease exponentially in the future, as Hubbert assumes, and thus there is no reliable way of estimating $Q_\infty$. For example, advances in exploration technology, such as the "bright spot" seismic technique, could even drive the curve up; the accessibility of new oil provinces offshore certainly would. Economic changes which discourage unproductive exploratory drilling, such as a change in the present tax law which allows deduction of most drilling expenses, or a decrease in the current high price of oil, could also drive the curve up.

The Academy committee seems to have accepted Hubbert's analysis without question, but then simply estimates a middle value. It quotes various industry estimates, which are not further substantiated; these estimates are also quite low but probably because they are not comprehensive and address themselves mainly to commercial prospects. But the committee does not discuss or reference the two-volume study (*4*) of the American Association of Petroleum Geologists, which uses the volumetric method and arrives at the high estimate of 485 billion barrels of undisovered recoverable oil.

Fortunately, policy should not be affected by the committee's results. A large increase in the rate of U.S. oil production requires mainly the opportunity to test new oil provinces, principally offshore and in Alaska, on federal lands. No one would deny that the oil content there should be similar to that of average onshore rocks (*5*).

Pending such an exploration program, a crash program now for synthetic fuels would lock in the U.S. consumer to high-cost energy unnecessarily.

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**References and Notes**

1. *Mineral Resources and the Environment* (National Academy of Sciences, Washington, D.C., 1975.
2. This critique of the Academy committee's estimates of oil and gas resources reflects in no way on the many other conclusions of their comprehensive report.
3. M. K. Hubbert, in (*1*), appendix to section 2, p. 9.
4. ———, *Am. Assoc. Pet. Geol. Bull.* **51**, 2207 (1967). The histogram covers the period 1860 to 1965. All Alaskan and most offshore discoveries are omitted.
5. Hubbert fits the curve through the extreme points of the histogram and gives a value for $Q_\infty$ of 168 billion barrels, of which 136 billion had already been discovered by 1965. Had he applied his method 10 years earlier, $Q_\infty$ would have been only 93 billion barrels, which is less than the 115 billion barrels that had actually been discovered by 1955.
6. I. Cram, Ed., *Future Petroleum Provinces of the United States—Their Geology and Potential* (American Association of Petroleum Geologists, Tulsa, Okla., 1971).
7. It may well be greater, especially in the Southern California borderlands, which are offshore from the Los Angeles Basin. This basin has one of the highest oil contents (per rock volume) known in the world.

## Autism, Stress, and Ethology

Nikolaas Tinbergen is to be applauded for his innovative attempt (5 July 1974, p. 20) to apply the methods of ethology to the study of pathology. However, his conclusion that autism is caused by psychological stress rather than by organic factors is at such variance with that of scores of researchers from many countries that the usefulness of the approach, or at least of Tinbergen's application of it, is thrown into question.

Tinbergen appears to have misinterpreted a study in which I participated when he writes, in attempting to discredit the biogenic view, that evidence "such as that on blood platelets" leads to erroneous conclusions in which causation is supposedly inferred from correlational data. The study in question (*1*) used my diagnostic checklist to subdivide a group of psychotic children into those who exhibited Kanner's syndrome and those with an undifferentiated form of psychosis. The determination of the 5-hydroxytryptamine (5-HT) efflux from the blood platelets, which was done in a blind study, showed a

highly significant difference between the Kannerian autistics and the undifferentiated "autistic" children. This study, as well as an earlier one in which the efflux of the Kanner-type children was shown to differ from that of normal children, was described in my 1971 article (*2*) which Tinbergen quotes.

If all that our studies showed was that the 5-HT efflux from the platelets of psychotic children differed from that of normal children, Tinbergen's reiteration of the truism that "correlation does not prove causation" might be applicable. However, since our study showed that a small subgroup of children, preidentified by a symptom checklist, could be discriminated by a blind biochemical test, not only from normal children but also from other psychotic children, in 19 out of 23 attempts, the idea that emotional disturbance causes the biochemical error must be rejected in favor of the far more plausible explanation that this type of biochemical error produces a predictable pattern of aberrant behavior in children. In other words, if stress caused the 5-HT efflux to rise, the increase would be seen uniformly, or at least in a gradient, in all psychotic children. Kanner's syndrome, like the Lesch-Nyhan syndrome (*3*), thus represents a specific biochemical abnormality closely related to a unique behavioral syndrome. The assertion that the causal arrow points in the other direction is not tenable.

In discussing the concept of causation, Tinbergen states in his reference 5, "Our experimental evidence discussed on pages 21 and 22 is hard, whereas evidence on correlations . . . are scientifically useless . . . ." The error in logic Tinbergen explicitly rejects, that of assuming from correlation, is the first cousin to one which he then commits: assuming correlated effects (superficial resemblance between a stressed normal child and a psychotic one) must have an identical cause. Further, he assumes that the implied identical cause—stress—is socially induced in each child, rather than the result of a physically caused cognitive impairment in the autistic child.

Kanner's original observations, on the incidence of autism among the firstborn, based on his first 100 cases (of children with Kanner's syndrome) do not appear to have been confirmed by subsequently collected data. It is interesting that Tinbergen should assume that an elevated incidence of autism among the firstborn implies psychological causation. Much to the contrary, there are a number of biological mechanisms which could well account for a primogeniture effect (*4*).

I am less convinced than Tinbergen is of the value of the ethological approach to the study of pathology, if the approach is to require observation of healthy individuals whose behavior simulates that of the sick ones in some way. How much can one learn about polio by observing one's colleagues hobbling about on crutches after an unhappy weekend on skis? How much, in fact, can one learn about polio by observing the polio victims themselves? The danger is not only one of failing to reach correct conclusions but also one of reaching conclusions that are incorrect and possibly damaging.

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M. Lesch and W. L. Nyhan, *Am. J. Med.* **36**, 561 (1964).
4. B. Rimland, *Infantile Autism: The Syndrome and Its Implications for a Neural Theory of Behavior* (Appleton-Century-Crofts, New York, 1964), pp. 142–143.

Tinbergen's discussion of early childhood autism as a stress disease, while adding new observations and fresh insights to the field, leaves the false impressions that the disease is caused primarily by a quirk in the child's environment, that it consists primarily of social withdrawal and related symptoms, and that the establishment of adequate social interactions is a necessary and sufficient therapy for it. Many years of careful study by researchers of various persuasions have shown these ideas to be erroneous, or at least only part of the story.

Although Tinbergen states in his reference 18 that genetic predisposition is possible, the body of his article emphasizes possible environmental determinants of the disease. Several studies of concordance in twins have suggested the "possibility of a genetic determinant in at least some cases of childhood autism" (*1*), and the large number of seemingly unrelated symptoms which these children have in common argues strongly for an important component. Such characteristics as repetitive use of objects in inappropriate ways, under- or overreaction to stimuli, apparent insensitivity to pain, and characteristic body motions are not obviously related to deficits in affiliation and socialization processes. Wing (*2*) also points out that any abnormality in mother-child interaction may have resulted from the child's early lack of response to the mother's maternal behavior, so that abnormal socialization relationships may be a result rather than a cause of autism. Stress in parents, their overattention or underattention to the infant (both have been proposed as triggers of autism), or other subtle parental behaviors do not explain why some children living in families become autistic while others developing in highly stressed families or in deprived orphanage environments, with much greater social insults, develop normally. Further, autistic children who do develop language acquire it in characteristic and highly abnormal ways, having particular difficulty with synonyms, for instance (*3*, p. 68). None of these widely observed characteristics of autism can be explained by affiliation and socialization difficulties. From such facts as these, as well as our own observations, we favor the hypothesis that a child is born with a threshold level for autism, and that the level for some children is so low that it is exceeded even in near-optimal environments.

There is some evidence that autism is associated with high intelligence (*4*), perhaps as part of the "genetic load" which the human race accepts in exchange for obtaining some individuals of very high intelligence. Parents of autistic children are very frequently of above-average intelligence, and both highly intelligent and autistic children are overrepresented among firstborns, males, and Jews. With Jamesian eloquence, Rimland concludes: "We must give serious consideration to the hypothesis that an infant's road to intelligence lies along a knife-edged path, and the higher the potential intelligence, the steeper and more precarious the slope" (*4*, p. 127).

In recent years most therapists and schools of therapy have recognized the importance of establishing sincere rapport between the autistic child and the teacher-therapist. It has long been known that the autistic child's extreme aversion to strangers and strange situations can usually be overcome with patience and with techniques like those which Tinbergen describes (*3*, p. 123). We see this as a *prerequisite* to effective therapy, a starting point from which the child must be taught as much as possible about his world. Here again, special problems of the autistic child are encountered; even after the child is comfortable with the therapist and is well-motivated, learning—especially lan-

guage learning—is difficult. We have seen autistic children in operant-conditioning situations who were comfortable with the situation and were having difficulty with simple linguistic tasks, despite their obvious intense and sustained effort. In general, the autistic child will not learn language spontaneously but must be laboriously taught every word and phrase.

Our own method of therapy (*5*) is based on operant conditioning in a setting of warmth and trust and includes the training of all people with whom the child comes in contact—parents, nursery school teachers, babysitters, and so forth. Food rewards can be gradually replaced by social rewards as therapy progresses.

Tinbergen's comparison of observations of autistic and of normal children is a potentially very productive approach, although one must use caution in assuming that particular activities exhibited in both normal and autistic children are similar in cause or ethological function. Lorenz (*6*) has discovered numerous examples of such changes in function of particular behavioral patterns in ducks and geese.

We hope that cooperation of ethologically oriented and other approaches will lead to advances in the understanding and treatment of autism which no approach can attain alone.

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5. D. Bridgeman, *"Paul": Behavior Sessions with an Autistic Child* (film) (Speech Clinic, Free University of Berlin, West Berlin, 1973).
6. K. Lorenz, *On Aggression* (Harcourt Brace & World, New York, 1966).

With Tinbergen, I have my doubts that the attribution of disorders of childhood, such as autism, specfic learning disabilities, and others, to genetic abnormalities can be more than mere inference at this point in our inquiry. However, I must object to his inference that behaviors, when found to be related to genetic effects, are "irreparable" or "incurable." The statement he makes in his Nobel address echo the assumption made by generations of well-meaning scientists and professionals that the effects of environmental causes are modifiable, but those due to genetic processes are not. Somehow environmental factors are viewed as having a more or less dynamic and fluid relation to behavior, while genetic factors are considered to produce more or less fixed and permanent deviations in behavior. Although Tinbergen does not advocate it, a still more extreme view one often finds expressed is that the environment exerts a continuous influence on behavioral development, but heredity has only an initiating function.

These statements are clearly inconsistent with current evidence on (i) gene action and (ii) treatment of "genetic" disorders. As to gene action, there is general agreement today that genes exert continuous control over the production of enzymes, which, in turn, influence the rate and efficiency of metabolic reactions (*1*). In this manner, genes play a significant role in the development of an organism and its behavior, and continue to affect the functioning of the organism throughout its lifetime. Furthermore, recent discussions of gene action emphasize regulatory mechanisms which afford flexible control and coordination of the activity and inactivity of particular gene loci (*2*). Indeed, if a pattern of behavior is found to be related to genetic effects [and there is a considerable body of data to indicate that many behaviors are a function of genetic anomalies (*3*)], then there is as much reason to assume that this relation is a dynamic and fluid one as there would be if the behavior were susceptible only to environmental control.

Similarly, there seems to be no logical or empirical basis for assuming that "environmental" behaviors are any more modifiable than "hereditary" behaviors. For one thing, changes in macroenvironments are not readily accomplished and do not always have stable effects (*4*). In this sense, the very real technological problems involved in altering behavior patterns are no greater for behaviors attributed to genetic defects than for those attributed to environmental stress. Second, the treatment of behavior disorders with known genetic causes (so-called "inborn errors of metabolism") and of those for which a genetic link is only suspected (for example, specific learning disabilities) has a short but impressive history. For example, Hsia (*5*) lists 28 hereditary metabolic diseases associated with subnormal intellectual functioning. Although the deficient enzyme has been identified for only nine of these diseases, methods of treatment are available for 12 of them. A case in point is phenylketonuria (PKU), which is a genetic disorder with well-documented behavioral effects. The retardation found in most untreated cases of PKU can be ameliorated or prevented by controlling the amount of phenylalanine in the diet.

For the most part, the relation between specific learning disabilities and genetic defects has not been established. However, genetic causes are suspect because of increased risk within families where one member has been identified as having a learning disability. In the case of some disorders, such as specific dyslexia, the evidence for a genetic relation is stronger (*6*). As a whole, these behavior dysfunctions often have been termed untreatable. Yet, remediation techniques, ranging from "patterning" to behavior modification, have been developed for dealing with several of the specific learning disabilities (*7*). An example of these approaches would be the use of phonetic drill and perceptual-motor training in the treatment of dyslexia (*8*).

We need not be pessimistic about the modifiability of behavior disorders that are found or suspected to be related to genetic effects. Indeed, the critical task of remediating behaviors seems to be less the analysis of causes than the analysis of the component responses which make up the normal pattern. This latter, of course, is the approach advocated by Tinbergen and the ethologists.

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5. D. Y. Hsia, in *Behavior-Genetic Analysis*, J. Hirsch, Ed. (McGraw-Hill, New York, 1967), p. 176.
6. B. Hallgren, *Acta Psychiatr. Neurol. Scand. Suppl.* **65**, 1 (1950).
7. B. R. Gearheart, *Learning Disabilities: Educational Strategies* (Mosby, St. Louis, 1973).
8. White it is true that these procedures are best described as palliative, in the sense that the cause of the disability is not eliminated, they might appropriately be called curative, in the sense that the individual is returned to normal functioning.

In a 1948 court action (*1*) in South Africa, two Nobel laureates in Physiology or Medicine, E. D. Adrian and Sir Henry Dale, testified against the claims of something called the Alexander technique, which Dale labeled "intensely dangerous quackery" that ought to be made "criminal" (*1*, vol. 10, p. 1941). The judge's decision, no victory for the technique irrespective of the legal aspects of the case, concurred with the testimony of the two Nobel witnesses that "in its claims to cure, the system constitutes dangerous quackery" (1, vol. 5, p. 976). Now Tinbergen devotes half of a curative system which he identifies with the Alexander technique. "It consists in essence," states Tinbergen, "of no more than a very gentle . . . corrective manipulation of the entire muscular system." Tinbergen describes the technique in terms such as "extraordinary," "seemingly fantastic," surprising," and "astonishing" and refers to "evidence given and documented by Alexander and Barlow" that the technique is presumed to have beneficial effects on a "very wide spectrum" of physical and mental disease: rheumatism, including various forms of arthritis; respiratory trouble, even potentially lethal asthma; circulation defects, which may lead to high blood pressure and also to some dangerous heart conditions; sexual failures, migraines and depressive states that often lead to suicide. Such evidence and documentation, however, in the writings of Alexander (*2*) (of which I am editor), or in Barlow's book (*3*), are nonexistent or sparse and anecdotal; moreover, some of the material that can be found in Tinbergen's sources tends to be highly ambiguous. Consider the example of "sexual failures," which rests upon a single anecdote (*3*, p. 127) about an impotent pianist who (having previously undergone psychotherapy) was brought to potency (upon seeing a photograph of a nude) during the course of his Alexander "treatment." Critically incomplete and susceptible of numerous interpretations, this turns out to be the sole "evidence" for Tinbergen's claim. In Alexander's writings, to which Tinbergen invites the attention of the medical profession, cancer, tuberculosis, and appendicitis are also included among the conditions which may be benefited by the technique. Tinbergen omits these: the treatment, he specifically notes, is not a cure-all "in every case." In Tinbergen's source material, there is no mention of controls. No proper allowance is made for the placebo effect. No satisfactory consideration is given to failures or dropouts. The material is selective: It deals with people who have entered this rather special treatment on a voluntary basis.

Particular attention should be paid to the diagrams and before-and-after pictures published with Tinbergen's lecture. Sheaves of convincing before-and-after posture photos are produced by almost every known school of physical manipulation, exercise, relaxation, and breathing today. Posture is too much a variable to provide a reliable index; it would be difficult to prove from a collection of such photographs whose methods are superior. Moreover, when one applies a microscope to the photographs accompanying Tinbergen's lecture, one discovers labels indicating that the "after" comes 3½ years later than the "before," which is not very supportive of what Tinbergen calls the "surprising, but indubitable fact that "even after 40 to 50 years of obvious misuse one's body can (one might say) snap back into proper, and in many respects more healthy, use" after a "short series" of Alexander sessions.

The two skeletal drawings reproduced on the cover of *Science* are supposed to be children (*3*, p. 143), but they are not so designated in the cover caption nor in Tinbergen's figure 6. In any case, they represent a contribution to anatomical science fiction, since the one on the right does not correspond to any x-ray, but projects a fantasy figure. In adult form he is shown more credibly in flesh in a detailed physical anthropology chart starting with Proconsul man 2 million years ago and culminating in "Alexander man," to be reached when "personal selection" has replaced natural selection (*3*, p. 34).

It should be noted that Alexander's work was not concerned with posture. He was concerned instead with what he called "use," by which he meant the complicated patterns of movement and rest which comprise all our usual activities, from the most strenuous to the least effortful (technical research at Tufts University over the past 25 years has been devoted to registering a precise index of change for these patterns). A side effect of improvement in "use" may or may not be an improvement in posture, but Tinbergen's photographic exhibit of slump, slouch, and postural vice—no different from what most of us have encountered in elementary physical education—has no special relevance to the Alexander technique. In quite the same way, it is confusing to intermingle thoughts on "misuse" and "use" with materials of postural research which have no relation to Alexander's concepts or terminology.

Tinbergen's emphasis on teachers and what they do with their hands is noteworthy ("therapists," he calls them, who give "treatment" to their "patients"). The Nobel speech outlines this corrective manipulation as starting with the head and neck, thence to shoulders and chest, and finally the pelvis, legs, and feet (with variations according to patient). "Compared with this," says Tinbergen, "many types of physiotherapy which are now in general use look surprisingly crude and restricted in their effect, and sometimes even harmful to the rest of the body." In a recent editorial (*4*) British osteopaths claim the technique as properly their own: "How much greater success should our highly trained osteopaths have now with the enormous understanding of detailed musculo-skeletal cause and effect that is at their finger-tips." They also challenge, as have more influential voices in physiotherapy and medicine, the qualifications of the Alexander teachers to observe and treat abnormal function in their students. What training in anatomy, physiology, study of the biodynamics of activity and its perversions, and knowledge of musculoskeletal pathology, etiology, and pathogenesis entitle these teachers to make such observations? "Admittedly, the training of a good Alexander teacher," says Tinbergen, "takes a few years." Certification by the Society of Teachers of the Alexander Technique does in fact, proceed mainly by a laying on of hands in apostolic succession from Alexander and those upon whom he bestowed certificates; and his own procedure in this respect was, to say the least, both highly personal and eccentric, since he tended to regard the technique as simply a matter of "doing his own thing." There exists no objective standard in the certification of Alexander teachers which could pass muster. Horace Kallen, John Dewey's colleague and a founder of the New School for Social Research, reported from personal knowledge a case where the technique "did a great deal of damage" (*5*). Only last summer it was rejected by all the hospitals of Sydney, Australia, in connection with a childbirth, which subsequently took place at home. John A. Mathews (*6*)

has ridiculed as a breach of logic worthy of being anthologized the confusion of medicine with education in discussion of the technique.

Perhaps what most needs to be emphasized is that Alexander learned the technique entirely by himself, without outside assistance of any kind; so too have others. If there is a subject matter here, it is something other than corrective manipulation. From this subject, Tinbergen's essay on massage provides a real distraction.

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When a scientist ventures into a neighboring field, he is likely to express unconventional views; and, if he utters unkind criticism as well, he has to expect countercriticism. But this is often the beginning of an interdisciplinary cross-fertilization. In my reply to the above comments on my Nobel address, I shall try to be constructive but brief.

*Early childhood autism.* Rimland argues that the fact that the 5-hydroxytryptamine from blood platelets was found to be significantly different in Kanner-type children than in other psychotic children points to the "far more plausible explanation that this type of biochemical error produces a predictable pattern of aberrant behavior"—more plausible, that is, than my opinion that this biochemical correlate is as yet no more than a characteristic with a place in the complex cause-effect web that cannot yet be determined. Revealing though it may one day become, this correlate still appears to me to have no different status from that, for instance, of retarded bone growth, for I fail to find evidence which supports Rimland's view. But I welcome his appeal for "plausibility" as the criterion for any hypothesis. The main point I have tried to make in my criticism of the various theories on the nature and origin of autism is that, in addition to being insufficiently supported, they do not try to link all the known components of Kanner's syndrome with either the idea of a "purely genetic" explanation or that of an organic explanation, in particular, structural brain damage. An ethological approach can help us focus more attention on (i) the observable behavior of both autists and normal children, and (ii) the external situations that either reduce or aggravate an entire set of symptoms in both. When this is done—and I submit that it has not been done in this branch of medical research with anything like the perceptiveness and discipline with which ethology has been applied to a number of animals—the interpretation of the autistic state (of *being* autistic in Kanner's sense) as one of motivational conflict is clearly well founded. In addition, this hypothesis is also plausible in another sense, namely, it has the merit of being consistent with, and almost requires the coappearance of, many of the described symptoms. Gaze (and other) aversions, keeping or increasing distance orientation of the whole body away from persons and novel situations, tantrums, and a number of more subtle signs are to be expected as indicators of withdrawal; stereotypies, overall arousal, and various kinds of muscular tension are to be expected as consequences of conflict motivation; and overall retardation, except for "islands of good performance," is to be expected as a secondary, long-term consequence of a self-imposed cutoff from most channels of learning. In our experience the "islands" appear in the very few situations with which the child has become familiar.

I also tried to treat the question of what makes the child autistic as distinct from that of the autistic condition. I argued, and have since, if anything, been strengthened in my belief, not only that early environmental influences cannot be ruled out, but that they even seem to be quite likely. Of course this is not to claim that there may not be "the possibility of a genetic determinant"—apart from monozygotic twins, no two children are genetically identical. But, as the Newsons (*1*) have so clearly shown, no two children are treated exactly alike either; in fact, even in the same family different children are treated very differently. But I feel that the statement, repeated by Bridgeman and Bridgeman, that "autism is associated with high intelligence" does not really hit the nail on the head as far as the parents are concerned. I believe that, in the search for possible environmental stresses originating in the behavior of parents (which I consider to be only a part of the total, very complex stress situation), the relevant factors have not yet been found. It may be more fruitful to look for such things as seriousness, preoccupation with outside interests, uncertainty and even anxiety in dealing with a child, and also inconsistency—all qualities that are often found in parents of autists, qualities that suppress alert responsiveness to the child and, above all, the cheerful and even joking atmosphere that children need so much. In part of a 1-hour lecture to a mixed audience I could, of course, not elaborate on the delicate and complex issue of the genesis of autism. I am convinced that Wing (*2*) is right when she points to the possibility that an unresponsive newborn can, through failing to provide the correct stimuli for the mother, set off a vicious circle of defective affiliation and socialization. If this continues it will, in turn, impair the child's later exploratory learning (for which it needs the "umbrella of security" that good affiliation alone can provide), as well as learning through social channels. It also goes without saying that the behavior of parents after they have lived for a time with an autistic child must not be taken as indicative of their behavior with the newborn. Studies of normal language development should throw more light on the specific difficulties that autists have and to which the Bridgemans refer. I have, for instance, found that many child psychiatrists do not know that pronoun reversal and echolalia are quite common, but passing, phases in the development of normal children. Some oddities in the reappearance of speech during speech therapy may well be due to the type of speech therapy that is applied —the catchword covers a number of quite different procedures. When not subjected to the more intrusive kind of speech therapy, some autists can suddenly reveal a surprising mastery of language, which they can only have learned "latently," by listening.

Schonebaum objects to my use of the words "irreparable" and "incurable." What I meant, and should perhaps have made clear, was "not completely curable." For instance, some mongols are educable to quite a surprising extent (*3*). But Schonebaum goes too far when he writes that "there seems to be no logical or empirical basis for assuming that 'environmental' behaviors are any more modifiable than 'heriditary'

behaviors." One of the important advances in psychology and ethology is that so much evidence is now taken seriously which shows that a great variety of learning abilities and learning failures are due to genetically determined "constraints" (and, I might add, "special potentials" as well) (*4*).

At the present juncture I claim neither more, nor less, with regard to autism in its various forms, its nature, its origins, and the most desirable treatments, than that we are still trying hard to see both the woods and the trees and are all still groping our way; and that an approach of the type that I have tried to outline deserves to be followed up. Our experience in the 1½ years since my Nobel address was written seems to us to justify the "glimmer of hope" of which I spoke in Stockholm. A more comprehensive treatment of our views will have to wait until more long-term results of various therapies become available.

*The Alexander technique.* In the criticisms of my views on autism I sense the promise of a more fruitful collaboration in the future, for they are very reminiscent of the development of such collaboration between American behaviorists and ethologists which started, some 30 years ago, with equally sharp reciprocal criticism. But I must admit that I see no such promise in Maisel's comments on the Alexander technique and my recommendation of it. He seems to agree with Adrian and Dale that the technique is "dangerous quackery." But it puzzles me why Maisel publishes at the same time a selection of "the essential writings of F. Matthias Alexander," with a lengthy introduction which (to join him in his game of quoting out of context) ends with the sentence: "For many today, living in cities, working indoors mostly at sedentary, small-muscle jobs, continually exposed to polluted air, and hampered in opportunities for any large recreation—for many of this latest brood of monsters, it could just turn the trick" (*5*, p. l). Trying to figure out exactly what Maisel is after, I find myself saying time and again, with Alice in Wonderland, "curiouser and curiouser." In the meantime, having continued my "watching and wondering" and having seen quite a number of people benefit, I can only repeat: Alexandering may be good for you—why not give it a try?"

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## University Reform

The article by F. Reif (3 May 1974, p. 537) presents a reasonable response to the problem universities face if they are to reform instructional practices. To that discussion I would like to add two points which are important elements in the planning for this kind of change.

First, there should be an explicit recognition of the kinds of educational reform going on outside the university. It would be a mistake to focus solely on the internal resources that are available and neglect the school systems, the state departments, the National Institute of Education, the research and development laboratories, and so forth. We are past the era of self-contained institutions that have little impact on or interaction with the larger community.

Second, the conceptions and plans for reform should be viewed through the eyes of the potential beneficiaries (the students), the reform designers, and the administrators of the university. With the acknowledged financial tightening at many universities, an intellectually honest and well-intentioned innovation could result in a sterile instructional process. It is conceivable that there is a way to provide for both quality education and financial realities, but unless all affected parties vigorously question and analyze propositions, in terms of both implementation and consequences, the exigencies of the moment may exert a stronger force than is ultimately desirable.

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## Methysergide Dosage

In the report by Zemlan, Ward, Crowley, and Margules (9 Mar. 1973, p. 1010), the systemic dosage level of methysergide maleate was erroneously reported as 3 milligrams per kilogram. The actual dosage used was 3 milligrams per animal.

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*Erratum*: The table accompanying Alan J. Grobecker's letter (28 Mar., p. 1145) was incorrectly printed. The correct table appears below.—ED.

Table 1. Estimated percentage of ozone reduction per 100 aircraft. [Adapted from table 1 in (*2*)*]

| Aircraft type | Fuel burned per year† (kg/year) | Altitude (km) | $NO_x$ emission index (EI) without controls (g/kg fuel) | Percentage of ozone reduction in Northern Hemisphere | | |
|---|---|---|---|---|---|---|
| | | | | Without controls | EI controls | |
| | | | | | 1/6 today | 1/60 today |
| Subsonic‡ | | | | | | |
| 707/DC-8 | $1 \times 10^9$ | 11 | 6 | 0.0034 | 0.00070 | 0.000070 |
| DC-10/L-1011 | $1.5 \times 10^9$ | 11 | 15 | 0.010 | 0.0020 | 0.00020 |
| 747 | $2.0 \times 10^9$ | 11 | 15 | 0.014 | 0.0025 | 0.00025 |
| 747-SP | $2.0 \times 10^9$ | 13.5 | 15 | 0.079 | 0.014 | 0.0014 |
| Supersonic | | | | | | |
| Concorde/TU-144 | $4 \times 10^8$ | 13.5 | 18 | 0.39 | 0.068 | 0.0068 |
| | $3 \times 10^9$ | 16.5 | | | | |
| Advanced SST | $3 \times 10^8$ | 16.5 | 18 | 1.74 | 0.32 | 0.032 |
| | $6 \times 10^9$ | 19.5 | | | | |

* Reference 2 in the original letter was A. J. Grobecker, S. C. Ceroniti, R. H. Cannon, Jr., *The Effects of Stratospheric Pollution by Aircraft, Report of Findings. Executive Summary* (National Technical Information Service, Springfield, Va., 1974); p. xvi † Subsonics assumed to operate at high altitude, 5.4 hours per day, 365 days per year. Supersonics assumed to operate at high altitude, 4.4 hours per day, 365 days per year. ‡ The present subsonic fleet consists of 1,217 707/DC-8's, 232 DC-10/L-1011's and 232 747's flying at a mean altitude of 11 km and is estimated to cause a 0.1 percent ozone reduction.