

Table 3. Attempted recovery under hypnosis compared with recovery while the subject is awake. Means and standard deviations are given. Four subjects were under high sedation and five under low.

Condition	High sedation (14-30 μg/ml)	Low sedation (8-13 μg/ml)
<i>Recognition items correctly identified (out of 10)</i>		
Awake	1.50 (1.00)	6.20 (1.79)
Hypnosis	3.50 (1.00)	6.00 (3.16)
<i>Associated pairs correctly recalled (out of 6)</i>		
Awake	2.50 (1.73)	1.60 (0.55)
Hypnosis	2.75 (1.89)	2.00 (0.71)

from earlier testing to be highly susceptible to hypnosis, the 24-hour retention test was followed by an effort to recover more information through hypnosis. To avoid the effect of exposure during the earlier test while awake, these subjects were tested for only half the items while they were awake, and the other half was saved for the test for recall under hypnosis. The subjects are grouped in Table 3 by the concentration of thiopental in their venous blood during the final learning period. In the four subjects with higher concentrations of the drug in their blood (mean, 16 μg/ml) there was some slight recovery of the recognition items. All four subjects recovered something more under hypnosis, while only one of the five in the lower sedation group did. There was no evidence of recovery of the associated pairs. It appears that for those who suffer large decrements under the drug more has been registered than is recovered when they are normally conscious.

That short-term memory is interfered with is shown by the greater number of trials required to learn the easy associated pairs when the subject is under sedation. Some of the items entered into long-term storage, for they were recovered after 24 hours; compared, however, with the perfect retention by control subjects, the marked losses after 30 minutes (under sedation) and after 24 hours (tested while awake) show impairment of longer-term memory.

The alternative possibility to interference with memory storage is that retrieval is interfered with. The evidence for some recovery under hypnosis (particularly of recognition materi-

al) indicates that more may in fact be stored than is recovered. There was, in fact, some spontaneous recovery of recognition material over the 24 hours for the sedated group, not under hypnosis, a fact which supports the same conclusion. However, the observation that material learned before administration of thiopental was recovered without impairment during deep sedation indicates that the memory defect is not simply one of retrieval. That material learned while awake was readily retrieved under sedation adds to our other observations that the drug-induced memory deficits were not state-dependent.

Results do not give unequivocal support to any of the current theories of consolidation processes in memory. In general, they are coherent with the interpretation of the sedated state as one of lowered intellectual functioning, in which attentive, discriminative, and associative processes are interfered with, with the consequent impairment of both learning and retention characteristics of the poorer learner.

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6. Samples of arterial blood were drawn from the last five subjects for comparison between the venous and arterial levels. While the amount of thiopental in the arterial samples averaged about 10 percent higher than in the venous samples, the relative amounts per subject remained sufficiently alike that analysis has been confined to venous samples.
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Mental Retardation

Although I agree with many aspects of Zigler's "developmental" theory of retardation (1), several points appear to merit further discussion and clarification. A key portion of his developmental theory is given in the following: "... the familial retardate's cognitive development differs from that of the normal individual only in respect to its rate and the upper limit achieved. Such a view generates the expectation that, when rate of development is controlled, as is grossly the case when groups of retardates and normals are matched with respect to mental age, there should be no difference in formal cognitive processes related to I.Q." (1, p. 294).

In this statement, Zigler defines mental age (MA) as the rate of intellectual development. In the same paragraph, however, he refers to MA as the "level" of intellectual functioning. Zigler's apparent failure to distinguish rate of development from level of development leads to a questionable prediction from his theory—namely that retardates and normals of the same MA will be similar with respect to their cognitive functioning.

Mental age is a transformation of the score made in an intelligence test and is a measure of the current level of intellectual functioning, not of the rate of accumulation of knowledge. If an individual's chronological age (CA) is also known, then the intelligence quotient (I.Q.) may be calculated: $I.Q. = (MA/CA) \times 100$. The I.Q. score is a rough index of the amount of information accumulated in a given number of years of life; thus it is a measure of rate.

According to Zigler, if groups of retardates and normals are matched for MA there should be no difference in formal cognitive processes related to I.Q. Figure 1 represents the growth in mental age of a hypothetical normal child, born in 1955, and progressing at the rate of one MA unit per year ($I.Q. = 100$), and of a retarded child, born in 1950, who is progressing at the rate of one-half MA unit per year ($I.Q. = 50$).

Assume that the two children were chosen for a learning experiment in 1960 because they both had MA's of 5 years. According to Zigler, if nonintellectual factors are held constant, the performance of the retarded child should equal that of the normal child. But note that the two children have dif-

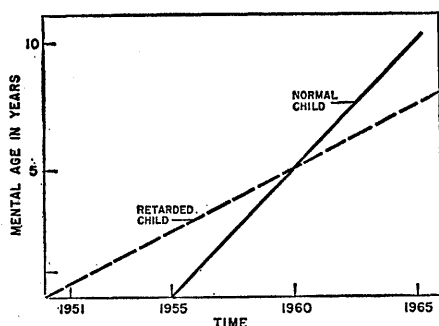


Fig. 1. Growth of mental age of two hypothetical children, one of I.Q. 100 and the other of I.Q. 50.

ferent rates of intellectual growth. These differential rates should not only appear as long-term phenomena but should also be evident in short-term laboratory tasks. It therefore appears imperative that Zigler's developmental theory should predict that the two children will perform differently, providing the task they are given is sufficiently complex to be sensitive to the abilities responsible for the differential growth shown in Fig. 1. The fact that these two children made identical MA scores on the intelligence test may be accounted for if one assumes that the intelligence test is more a test of recall of past learning, particularly vocabulary, than it is a test of the child's ability to deal with new and unfamiliar materials. Thus the MA score is basically a measure of achievement and may not be greatly affected by factors which determine the rate of accumulation of knowledge.

As evidence for his hypothesis of "equal-MA, equal cognitive functioning," Zigler cites research that demonstrates that the performances of normals and retardates, matched for MA, do not differ when motivational factors are controlled. However, it appears doubtful that the tasks used in the research cited are sensitive to the abilities which determine the rate of intellectual growth. The tasks appear to involve a minimum of learning and information processing, and even one which is said to be relevant to "problem solving" is, according to the authors (2, pp. 501-502), "an extremely simple task with successful performance depending primarily upon compliance with E's instructions."

To summarize, I maintain that Zigler's developmental theory should predict differential performance of retardates and normals of equal MA on complex cognitive tasks, because such individuals differ drastically in the rate

at which they are developing intellectually. The fact that Zigler and his associates have not found such differences is probably a function of the type of task which they have employed—one which is typically very simple and which would not be expected to be sensitive to those factors that produce differential intellectual growth rates.

One final aspect of Zigler's article also deserves comment. The basic difference between his "developmental" theory of retardation and the so-called "defect" theories may be more apparent than real. There may have been theorists who separated individuals into two populations, one "retarded" and the other "normal," and claimed that the normals "had" something that the retardates did not. However, I do not think such a belief is prevalent in modern American psychology. I suggest that the term "deficit" is used in a relative sense by most modern retardation theorists; it is not that these theorists believe that normals "have" something that retardates do not, but instead that retardates may have less of something than normals do.

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In Zigler's paper on the dilemma of mental retardation, an attempt is made to differentiate between two types of retardation—borderline familial and severe organic (1). At this stage it is difficult to establish with certainty the difference between intellectual superiority and inferiority, let alone discriminate between types and degrees of retardation. Zigler cites a number of theories and experiments on mental retardation, implying that the proponents of the theories and the experimenters really dealt with problems of mental retardation. As a matter of fact, the difficulty common to all such reports is the assumption that the individuals being investigated are mentally retarded individuals, when in reality they may not be. When multiple criteria are used in the determination of mental retardation, the I.Q. misclassifies four out of every five alleged retardates (2).

While Ellis's idea that the I.Q. represents neural integration is to some extent acceptable, it is also true that

the greater part of the variance of the I.Q. does not represent neural integration. Until the multivariate nature of a test score (I.Q. or any other) is fully explained and its intellectual variance is determined, no one will know for sure who is retarded and who is not. That is why many a potential genius languishes in special classes and schools for the retarded and many a retarded individual supports himself in the community without ever having had his "cognitive rigidity" tested.

Luria's idea that defects in the medium of language are related to mental retardation is either circular or based on studies of highly constricted population samplings. When the Russians extend their sphere of interest, some surprises will await them, since severe speech handicaps are present in people of all degrees of intelligence. Neither language defects nor "cognitive rigidity" are typical of the retarded; they occur in all people at random.

Zigler's somewhat dogmatic references to cognitive processes as if they should be considered intellectual are but unconfirmed assumptions. Many behavioral scientists believe that the thinking functions, such as concept formation, judgment, and reasoning, have a larger emotional than cognitive variance. Zigler is apparently coming around to a point of view which he rejected even a few years ago. Vague speculations about motivational, cultural, genetic, and emotional influences only increase the ambiguities about which he is complaining. Speculations will not be necessary when we devote our full attention to the fundamental issue of the multivariate nature of behavioral measures and to the development of an acceptable theory of intelligence. Then the above factors may be measurable, and the two types of retardates discussed by Zigler will probably be found to have no existence in reality.

In the meantime we will make a real contribution to the study of mental retardation when we frankly admit that neither physicians nor psychologists know much about it, even in such so-called clear-cut cases as those accompanying phenylketonuria, mongolism, cretinism, microcephaly, cerebral palsy, and so forth. Above all, no one should take it for granted that an article or a reported experiment on mental retardation does really deal with mental retardation. A close look at the experimental population of any study would expose the astonishing fact that

it comprises a mishmash of individuals with a wide variety of adjustment problems that have little to do with mental retardation. The true mental competency of these individuals is rarely established except by the most superficial of methods. We simply do not have the tools to do the job right. And the best tools we have are often misapplied and misinterpreted as measuring what they don't, for there are as many capricious theories and elegant nonmeasures of mental retardation as there are experimenters. Narrowly conceived experiments carried out on narrowly selected but heterogeneous groups only compound the unbelievable confusion in this important area of study.

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Jastak's argument centers about the fact that I have used one classification system to categorize the mentally retarded, whereas he prefers another. As I have pointed out, provided two classificatory systems have satisfactory reliabilities, one cannot be considered "truer" than the other. The question is not one of truth or falsity but rather one of the usefulness of the particular system, usually defined by the number and magnitude of the behavioral correlates associated with class membership within the system. If one is employing the conventional classificatory principle used in my article, then it is Jastak's system that results in the misclassification of the vast majority of mental retardates. How useful any classification system will be in the development of an adequate theory of intelligence is an empirical question. Such a theory is, I agree, badly needed. One does not always have to decide between competing classificatory systems. Within an area several classificatory systems may exist side by side, provided those using the different systems have different goals in mind (for example, prediction of different behavioral correlates of class membership).

Jastak is wrong in suggesting that we cannot differentiate organic from familial retardates. Although we are not completely errorless, we can and do make this discrimination. A major

point in my article was not that this two-category system of classification would illuminate all intellectual variables of interest, but that this simple differentiation must be made before any legitimate test can be conducted of issues separating developmental from defect theorists. I believe that these two broad types of retardation reflect two different types of etiology. Such a separation of the two, therefore, represents a reasonable first step preceding the construction in which much finer distinctions are made, systems which I think both Jastak and I would prefer. Nevertheless I take exception to Jastak's view that such a system would demonstrate that the two types of retardates I spoke of had "no existence in reality." To the extent that a classificatory system represents a conceptual construction of reality, then any system is just as "real" as any other system. To the extent that reality refers to the palpability or the physical evidence of the existence of an entity, Jastak is certainly wrong in regard to the organic types of mental retardation, where such evidence is readily available. It would appear premature to assert that, with advances in genetics, such evidence will not be forthcoming for the second broad class of retarded individuals now referred to as familial.

I wholly agree with Jastak's point that performance on so-called intellectual tasks invariably has an important emotional and motivational component. In fact, much of my article was directed toward supporting such a position. With respect to my changing my point of view, I see no great merit in an investigator never changing his mind. However, I do not believe I have changed my point of view over the past decade concerning the behavior of the retarded.

Weir makes a number of points meriting reply. His first criticism is based more on how I used the particular word "control" than on any substantive disagreement between us concerning the meaning of MA and I.Q. As should be clear from the total context of the paragraph cited by Weir, I asserted that in the MA-matched paradigm one takes into consideration the different rates of cognitive development (I.Q.'s) of normals and retardates. This procedure controls for known past differences in rate, and thus guarantees that, at the point in time at which the comparison is made, the two types of subjects are at the same cognitive level. The semantic confusion possible

when one attempts to distinguish between rate and level of cognitive development is demonstrated in Weir's view that the I.Q. score, which is a measure of rate, is "a rough index of the amount of information accumulated in a given number of years of life." This is erroneous since the amount of information at any point is a level phenomenon. How long it took to acquire that amount of information is a rate phenomenon.

In whatever way I used the word "control" and Weir the phrase "amount of information," we are in total agreement that the I.Q. is a rate measure and MA is a level measure. However, I cannot agree that it is my failure to distinguish rate of development from level of development that leads to questionable predictions from my theory. As should become apparent in the remainder of my reply, developmental theorists such as myself may be wrong, but we are certainly not confused. Weir's major point hinges on one's conception of the cognitive characteristics of two individuals who at the same point in time are at the same cognitive level, but who have manifested different rates in achieving that level. The crucial question is: What does this different rate imply? Weir assumes that the rate phenomenon with its I.Q. measurement reflects speed of learning or information processing. Given this assumption, Weir predicts that at every cognitive level the child with a low I.Q. will do worse than that with a high I.Q. on tasks demanding such learning or information processing. But is the I.Q. indisputably a reflection of these cognitive abilities? Of course not. The I.Q. is only a rate measure in the sense that it relates a nonpsychological measure (passage of time) to a psychological one (level of cognition achieved). Approached in this way it is the MA (level) and not the I.Q. (the relationship of MA to chronological age) that determines the exact nature, including the rate, of learning any task. If one really thinks that the rate of learning or information processing is related to I.Q. rather than to MA, I suggest that he compare the learning processes of a 3-year-old with an I.Q. of 150 and an 8-year-old with an I.Q. of 100.

Weir makes much of the different slopes of the MA curves presented in his figure, and argues that they tell us much about the cognitive functioning of normal and retarded children at particular points in time. Alas, under-

standing cognitive functioning is not so simple. If one took the trouble to extend Weir's curves for the two individuals through their adulthood, he would discover that eventually the slopes would be the same. The individual with an I.Q. of 50 would level off at MA 8 at the age of approximately 16, and therefore his MA would best be represented by a straight line. This is also true of the other individual, except that his MA curve would level off at MA 16. If it is the slope that allows us to make predictions concerning the quality of cognitive functioning, can we then argue that in adulthood the cognitive performances of normal and retarded individuals will be the same?

The major point is that one makes a number of theoretical assumptions when he asserts that, since the I.Q. is a measure of one kind of rate, then it must also be a measure of another kind of rate, namely a measure of the rate of learning or information processing on individual tasks. One can, of course, assert that both MA (level) and I.Q. (an hypothesized determinant of rate of cognitive functioning) influence cognitive tasks. But this is exactly the argument examined in my article. The person who holds that the I.Q., independent of level or MA, determines rate of cognitive functioning on short-term learning tasks is a difference or defect theorist. Which general position is correct is open to investigation, but there is no doubt that the two major approaches examined in my article generate quite different predictions.

I am in sympathy with Weir's argument that the MA obtained on standard intelligence tests is a far from perfect indicator of the nature of cognitive functioning (2). Indeed, if there were a consensus that the MA was a perfectly adequate measure of the formal features of cognition (for example, rate of information processing), there would be no argument between developmental and defect theorists, since by definition individuals of the same MA level would have identical cognitive structures. However, in his efforts to champion the predictive efficacy on cognitive tasks of the I.Q. over the MA, Weir appears to go too far. To argue that the MA is not an important determinant in the quality (including rate) of the child's learning of new and unfamiliar cognitive tasks is an error. Evidence on this point is clear, and I doubt whether anyone working in the area of cognition would take exception to it. In spite of its shortcomings, the single MA

measure and its factorial components have more cognitive correlates, including performance on purer Piaget-like cognitive tasks, than any other measure in psychology.

With respect to Weir's task argument, he and I probably could agree that an investigator should use a task sensitive to the particular factor that the investigator would like to demonstrate as being operative. Thus one interested in demonstrating the effect of motivational factors employs experimental tasks sensitive to these factors. There is no argument, therefore, that if one wishes to test the hypothesis that I.Q. is a measure of rate of information processing he should use a task that makes this type of cognitive demand on his subjects. My criticism of the various difference or defect positions was not based solely on findings obtained with motivational tasks, but rested also on the fact that the findings obtained by the supporters of these positions on tasks of their own choosing frequently have been equivocal. Furthermore, to imply that the holders of the developmental position have been reluctant to adequately test their views by using cognitively demanding tasks is to do them an injustice. They have frequently employed the same tasks used by the expounders of the various defect positions. These tasks include not only the concept-switching tasks referred to by Weir but a variety of discrimination learning, reversal learning, transposition, and learning of set tasks. Indeed, workers sympathetic to the developmental position have employed the probability-learning task used by Weir in his laboratory. Although Weir does not state the criteria by which we might know if a task were truly cognitive in nature, I find it difficult to believe that none of these tasks involves information processing and that they are therefore inadequate tests of the hypothesis of "equal MA=equal cognitive" functioning.

Weir attempts to close the gap between the developmental theory of familial mental retardation and the various "defect" positions by noting that certain "defect" theorists argue that retardates have less of something that normals of the same MA have, rather than having something that the normals do not have. This is true; however, other defect theorists have argued that retardates are qualitatively different from normals. It is for this reason that throughout my article I referred

to the general approach as a defect or difference orientation. It is the difference between familial retardates and normals of the same MA that is the point of contention between the developmental theorist and the difference theorist, whatever the hypothesized deficit underlying this difference may be. The gap between the developmental theorist and all the defect or difference theorists remains a wide one since the developmental position generates the hypothesis that there are no differences in formal cognitive functioning between familial retardates and normals matched on general level of cognition (typically measured by MA). What should be emphasized is that the developmental position at this point in time represents a tenable hypothesis. As long as the hypothesis clearly generates behavioral predictions, I would certainly entertain the possibility that it is wrong. Clearly, as my article pointed out, most theoretical workers in the area are entertaining this possibility. The argument presented in Weir's letter indicates that he shares their views. Fortunately, resolution of this can be achieved through thoughtful experimentation.

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Feedback of Speech Muscle Activity during Silent Reading: Two Comments

Hardyck, Petrinovich, and Ellsworth report that the presentation of auditory feedback from the speech muscles produces a "long-lasting cessation of the subvocalization" that occurs during silent reading (1). The auditory cue is effective, they conclude, because it allows subjects to make fine motor adjustments in their speech musculature. While this conclusion is consonant with other findings (2), the lack of systematic control data weakens the strength of the inference that auditory feedback is the critical variable. It is possible that the complex of giving the subjects a set to the effect that their subvocaliza-