coming afferent volleys were blocked irreversibly after administration of TD.

In two motoneurons after administration of TD had abolished almost all synaptic noise (Fig. 2, A and B), 1 ml of 0.2M KCl was dropped onto the same area from which the TD had diffused. On both occasions (Fig. 2, C and D), there was a great increase in the noise; the noise returned to the control level upon washing the area with Krebs-Ringer solution (Fig. 2E). Presumably this apparent increase in the frequency of synaptic potentials was due to depolarization of nerve terminals with spontaneous release of quanta of transmitter.

We conclude that most synaptic noise is due to transmitter release evoked by nerve impulses and can be removed by abolition of nerve impulses. Burke and Nelson indeed have demonstrated that some of the noise is due to spindle afferent activity (8). Study of cells in the feline cortex, particularly in isolated cortical slabs, has led to the conclusion that the synaptic noise in cortical cells is mostly evoked by nerve impulses (9).

There are undoubtedly some spontaneous potentials analogous to the miniature end-plate potentials of peripheral synapses. Presumably the potentials which persist at a low frequency after TD (Fig. 1C and Fig. 2, A and B) are representative of these. Application of TD to nervous tissue provides an unequivocal method of clearly distinguishing such spontaneous activity from the activity evoked by nerve impulses.

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Infant Vocalizations and Their Relationship to Mature Intelligence

Abstract. Correlations between infant development tests and later intelligence have been found previously to be very low. Through cluster analysis, six clusters of items were extracted from Bayley's California First Year Mental Scale. One item cluster composed principally of vocalizations did significantly correlate with girls' later intelligence, increasingly so with age, and more highly with verbal than performance scores.

It is generally believed that infant (0 to 2 years) developmental tests cannot predict the level of intelligence of the growing child or the mature adult. Virtually all developmental research reports (1) have reported insignificant correlations between the infant developmental test scores, which are based on extensive and somewhat similar samples of the infants' relatively limited behavioral repertoire, and later indices of intelligence. The presumption has grown that, until the effects of experience or the maturation of specific cognitive processes (hypothesis building, attention, extended memory) during the preschool years have occurred (2), testing infant intelligence for the purpose of estimating childhood or adult intelligence (as opposed to testing to detect infant developmental deviations) is a futile enterprise.

In arriving at this conclusion, total scores (that is, the total number of items passed) at a particular age level have customarily been used. Whether a partial score, based on an empirically derived factor or subset of items selected from these infant tests, might predict later intelligence or some factor of intelligence remained an open question. In pursuing this line of investigation, we extracted six relatively independent item clusters from the data on the 115-item California First Year Mental Scale for the total sample of children (males = 35, females = 39) in the Berkeley Growth Study (3). The frequent testing of these children (generally at 1-month intervals) permitted use of the age at which each child first passed each test item as the basic data for this analysis. This treatment of the longitudinal data permitted any test item to correlate highly with any other item, regardless of the difference in the "average passing age" of the two items, so long as the children who were precocious in passing the earlier item were similarly ranked in precocity in passing the second item. This statistical advantage, not available in factoring simple pass-fail item data from tests administered at a single age (where only a few test items are discriminating), permitted clusters to be composed of items drawn from the entire age range (0 to $1\frac{1}{2}$ years) covered by the Bayley Scale.

One of the six factors which were extracted from the combined sample did appear to be related to girls' intelligence (but not boys'), as measured at years 6 through 26. Six of the seven items defining this factor involved the girls' early vocalization behavior. The items ranged in age placement on the Bayley Scale from 5.6 to 13.5 months. clearly well before the age when schoolage intelligence is first predictable from total test scores. Predictability of school-age intelligence increases markedly between 2 and 4 years. A useful prediction of adult intelligence cannot be made until after 4 years of age, when verbal and performance skills have matured to a considerable degree (4).

The seven items and their age placements, in months, are: vocalizes eagerness (5.6), vocalizes displeasure (5.9), vocal interjections (8.1), says "da-da" or equivalent (8.5), pulls string adaptively (9.5), says two words (12.9). and uses expressive jargon (13.5). The seven items, with an average productmoment intercorrelation of .44, form a highly reliable scale (.84) as calculated by the Tryon cluster analysis computer program (5).

Figure 1 plots the correlations between this factor and girls' I.Q.'s from 6 through 26 years. As the girls mature, the correlations between their intelligence scores and this early vocalization factor increase, reaching a range generally between .40 and .60 during the 13- to 26-years period. For the 6- through 26-years period, the correlations for the boys' early vocalization factor with intelligence are predomiFig. 1 (right). Girls' vocalization-factor correlations with verbal, performance, and full-scale intelligence scores at ages 6 through 26 years. The bottom line represents a smoothed "base-line" curve of average 10- to 12-month total test scores' correlations with later full I.Q. The top smoothed curve indicates the increased prediction available from employing the vocalization-factor scores. Solid dots represent the actual vocalization factor-fullscale I.O. correlations. Fluctuation in the two curves could be due to either fluctuating numbers of subjects or different I.Q. tests administered at the different ages. Crosses and diamonds have been included to illustrate the better prediction by the vocalization factor of later verbal intelligence.

nantly negative and never reach statistical significance.

At eleven ages (6) during this 6- to 26-year age span, separate verbal and performance subscale scores were available, permitting us to detect whether this early vocalization factor correlated higher with verbal than with performance capabilities. Correlations with verbal intelligence were higher at 9 out of 11 ages.

Despite the consistent trend of positive and increasing correlations across time and despite the confirming evidence of higher correlations with verbal as opposed to performance measures of intelligence, further assurance that these findings were not due to chance effects seemed in order. We therefore correlated the factor coefficients (essentially the correlation of each item with the factor) on this early vocalization dimension for all 115 items from the Bayley Scale with each item's separate correlation with a composite intelligence score based on all available test scores from ages 13 through 18 years. In a sense, this procedure permitted evaluation of the predictive efficiency of the early vocalization dimension over all 115 items. In fact, the product-moment correlation coefficient describing this relation was .33. When the same set of 115 factor coefficients was correlated with separate verbal and performance composite intelligence scores (from data available at ages 14 through 18 years), as expected, the ver-

Fig. 2 (right). Scattergram of 115 Bayley First Year Mental Test items' factor coefficients on the vocalization factor plotted against each item's correlation with mean full-scale standardized intelligence scores from ages 13 to 18. Circled dots represent defining items of the vocalization factor.



bal intelligence correlation was higher (verbal r = .24, performance r = .00).

Figure 2 presents the scattergram corresponding to the first of these three correlations. Note that six of the seven definers fall in the outermost region of the top right quadrant, indicating that they contribute heavily to the correlation of .33. Of the four additional items which also occupy this area of the scattergram, one is an early vocalization item (vocalizes satisfaction, age placement = 6.5 months), while the other three are early assertive and adaptive acts (postural adjustment, 0.5 month; retains two cubes, 5 months; unwraps cube, 10.6 months).

These results strongly suggest that developmental psychologists need to rethink their previous conclusion that infant developmental test scores are unrelated to later measures of intelligence. Although these results should be replicated on data from other necessarily longitudinal studies, and although even if replicated their power to predict mature intelligence will be modest indeed, these findings, if found valid,



force us to reconsider our notions of the origins of intelligence (particularly with respect to sex differences), and to look more closely to the genetic structure of the child and the ecology provided by the parents for traces of these origins.

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References and Notes

- For example, the correlation between total development test scores at 8 months and I.Q. at 4 years is .02 [N. Bayley, J. Genet. Psychol. 75, 165 (1949)]. A correlation of .32 between 6 months and 5 years is reported by C. B. Hindley [J. Child Psychol. Psychiat. 6, 85 (1965)]. However, one study on preverbal vocalizations, not overall intelligence, between 6 and 18 months reports r's of about .45 with I.Q. at 3 years [F. L. Catalano and D. Mc-Carthy, J. Psychol. 38, 203 (1954)].
- Comparisons of the relations between children's I.Q.'s and abilities of true and of adoptive parents have been made by M. P. Honzik [Child Develop. 28, 215 (1957)] and by N. Bayley [Monographs Soc. Res. Child Develop. 29 (6, whole No. 97) (1964)]. See also G. M. Whipple, Ed., The Thirty-Ninth Yearbook of the National Society for the Study of Education. Intelligence: Its Nature and Nurture (Public School Publishing Co., Bloomington, Ind., 1940).
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 4. Correlations of .42 between 4 and 18 years, for 211 subjects, and .61 between 6 and 18 years, for 214 subjects, are reported by M. P. Honzik, J. W. Macfarlane, and L. Allen, J. Exp. Educ. 17, 309 (1948). N. Bayley [J. Genet. Psychol. 75, 165 (1949)], using an average of three consecutive tests to obtain more reliable scores, obtained r's of .62 between 4 and 18 years for 40 subjects.
- 5 Domain validity, as defined in the Tryon system, is equivalent to the correlation between subject's actual score in a particular domain of ability and a hypothetical score which measured without error his standing in that domain or area. See R. C. Tryon [Educational Psychol. Meas. 18, 3 (1958)] for compoutational formula.
- computational formula. 6 At 6 and 7 years of age the Berkeley Growth Study subjects were given the 1916 Stanford-Binet, for which no methods are available for determining separate verbal and performance scores. At 13 and again at 15 years they received the Terman-McNemar test, which also has no separate verbal and performance scores. At ages 8 through 12 and again at 14 and 17 the revised Stanford-Binet intelligence tests (L and M) were administered, for which verbal and nonverbal factor score formulas are available [Q. McNemar, *The Revision of the Stanford-Binet Scale: An Analysis of the Stanford-Binet Scale: An Analysis of the Stanfardization Data* (Houghton Mifflin, Boston, 1942)]. At the remaining ages (16, 18, 21, and 26 years) the Wechsler-Bellevue test, which is divided into verbal and performance sections, was used.
- tions, was used.
 Supported, in part, by grant MHO8135 from the National Institute of Mental Health. Marjorie P. Honzik contributed to earlier phases of work on this study.

Puromycin Effect on Memory May Be Due to Occult Seizures

Abstract. Intracerebral injections of puromycin, which have been shown to impair memory 3 hours after training, increase the susceptibility of mice to seizures after administration of normally subconvulsive doses of pentylenetetrazol. Cycloheximide, which antagonizes the puromycin-induced amnesia 3 hours after training, also antagonizes the puromycin effect on susceptibility to seizure. The anticonvulsant diphenylhydantoin antagonizes the puromycin effect on memory. The puromycin effect on memory may be due to occult seizures.

We have reported that mice that are injected intracerebrally with puromycin 5 hours before training can learn to escape shock in a one-choice maze but have markedly impaired retention when tested 3 hours later or thereafter (1). Since puromycin inhibited cerebral protein synthesis very extensively, it seemed possible that it impaired memory by interfering with the synthesis of protein required for memory storage. However, injections of cycloheximide, which inhibited cerebral protein synthesis more extensively than puromycin, did not interfere with memory 3 hours after training and antagonized the effects of puromycin (2). The studies with cycloheximide suggested that the puromycin effect on memory 3 hours after training might not be due to interference with the synthesis of a protein required for memory storage but rather to some other action. This impression was supported by our finding that recordings from the hippocampal region of the brain made 5 hours after intracerebral injection showed markedly diminished and irregular activity in puromycininjected mice but not in mice which had been injected with cycloheximide or saline (3). The present studies provide further evidence that the puromycin effect on memory 3 hours after training may be related to its production of abnormalities in cerebral electrical activity.

To extend our observations of the effects of intracerebral injections of drugs on electrical activity of the brain, recordings were made at a number of times after injection. We found that, within the first few hours after intracerebral injection of puromycin, recordings from the hippocampal region of the brain showed frequent seizure activity which was not apparent from observation of the animal's behavior. Recordings from cycloheximide- or saline-injected mice showed far less frequent seizure activity. It seemed, therefore, that the marked decrease in electrical activity observed in the puromycin-injected mice 5 hours after administration of the drug probably represented a post-ictal phenomenon after repetitive occult convulsive discharges. Since cycloheximide was found to antagonize the amnesic effect of puromycin 3 hours after training, the effect of a combination of cycloheximide and puromycin on cerebral electrical activity was determined. Cycloheximide was found to antagonize the electrical abnormalities produced by puromycin, but the finding was difficult to qualify. Quantification of data on large numbers of mice was considerably simplified by studying the effects of normally subconvulsive doses of pentylenetetrazol on overt seizure activity in mice which had been injected intracerebrally with puromycin or other drugs. Since the recordings that we made suggested that the primary abnormality that puromycin produces is an increase in seizure activity, we reasoned that subconvulsive doses of pentylenetetrazol might produce overt seizure activity in puromycin-injected mice, and that the cycloheximide antagonism to puromycin might be expressed in a reduction in susceptibility to pentylenetetrazol-induced seizures.

The details of the procedure that we used and the results we found are shown in Table 1. Puromycin-injected mice were far more prone to develop seizures than were cycloheximide-, acetoxycycloheximide-, or saline-injected mice when pentylenetetrazol was administered subcutaneously either 1 or 5 hours after intracerebral injections. Addition of cycloheximide to the puromycin solution markedly decreased the effect of puromycin on susceptibility to seizure. Hydrolysis of the puromycin also diminished its effect.

Studies were also made of susceptibility to pentylenetetrazol administered 5 minutes after intracerebral puromycin. Such treatment produced no increase in seizure activity. This suggests that it may be necessary for puromycin to diffuse more extensively than it has at 5 minutes, or to be chemically altered, before it can act to increase brain irritability. The product of such a chemical alteration might be peptidyl-puromycin, which is formed by incorporation of puromy-