lated. The evoked potential differences reported are therefore related more to the shape of the stimulus than to its size, and seem to constitute a physiological correlate of perceptual rather than sensory processes.

E. ROY JOHN
R. N. HERRINGTON
Brain Research Laboratories, Department of Psychiatry, New York Medical College, New York
SAMUEL SUTTON
Biometrics Research, New York State Department of Mental Hygiene, and *Columbia University, New York*

References and Notes

1. H. G. Vaughan, Jr., *Vision Research* **6**, Suppl. 1, 203 (1966); H. G. Vaughan, Jr., and R. C. Hull, *Nature* **206**, 720 (1965); T. Shipley, R. W. Jones, A. Fry, *Science* **150**, 1162 (1965); W. R. Uttal, *Psychol. Bull.* **64**, 377 (1965).
2. M. Haider, P. Spong, D. B. Lindsley, *Science* **145**, 180 (1964); E. Garcia-Austt, J. Bogacz, A. Vanzulli, *Electroencephalog. Clin. Neurophysiol.* **17**, 136 (1964).
3. H. Davis, *Science* **145**, 182 (1964); E. Callaway, R. T. Jones, R. S. Layne, *Arch. Gen. Psychiat.* **12**, 83 (1965); M. M. Gross, H. Begleiter, M. Tobin, B. Kissin, *Electroencephalog. Clin. Neurophysiol.* **18**, 451 (1965).
4. S. Sutton, M. Braren, P. Peterson J. Zubin, E. R. John, presented at Eastern Association of Encephalographers, Electroencephalographic Society (1965); S. Sutton, M. Braren, J. Zubin, E. R. John, *Science* **150**, 1187 (1965).
5. C. D. Geisler, L. S. Frishkopf, W. A. Rosenblith, *Science* **128**, 1210 (1958); I. Rapin, *Ann. N.Y. Acad. Sci.* **112**(1), 182 (1964); R. W. Lansing, *Electroencephalog. Clin. Neurophysiol.* **18**, 514 (1965); E. Donchin and D. B. Lindsley, *ibid.* **19**, 325 (1965); J. D. Wicke, E. Donchin, D. B. Lindsley, *Science* **146**, 83 (1964); H. G. Vaughan, Jr., *Vision Research* **6**, Suppl. 1, 203 (1966); H. Davis and S. Zerlins, *J. Amer. Soc. Agron.* **39**, 109 (1966).
6. P. H. Schiller and S. L. Chorover, *Science* **153**, 1398 (1966).
7. R. M. Chapman and H. R. Bragdon, *Nature* **203**, 1155 (1964); M. W. Van Hof, *Acta Physiol. Pharmacol. Neerlandica* **9**, 443 (1960); H. Begleiter, M. M. Gross, B. Kissin, *J. Psychophysiol.*, in press; R. Spehlman, *Electroencephalog. Clin. Neurophysiol.* **19**, 560 (1965); K. Lifshitz, *J. Psychophysiol.* **3**, 55 (1966).
8. Supported by NIMH grant MH-08579 (E.R.J.) and MH-0776 (S.S.) R.N.H. is a Sir Henry Wellcome traveling fellow from the Department of Psychological Medicine, University of Glasgow. We thank Dr. P. Schneidau and Dr. D. S. Ruchkin for specialized assistance.

12 December 1966

Mental Retardation

"In the Soviet Union," writes Zigler in *Science* (*1*), "no distinction is made between retardates having known organic impairment and that larger group whose retardation is of unknown etiology, nor are genetic or cultural factors considered to be determinants of mental retardation." This is not quite true, though both Russian and American commentators have contributed to the confusion. Pevzner, a leading Russian authority, in her book *Oligophrenia: Mental Deficiency in Children* (*2*), says explicitly, "In my definition of oligophrenia I include those forms of mental deficiency which arise as a result of intrauterine or early lesions of the central nervous system and which show no tendency to progress. . . . Foreign psychiatrists often include mentally deficient and backward children in one group. This unjustifiable widening of the concept of oligophrenia leads to erroneous conclusions regarding its etiology, pathogenesis and clinical pattern. Our investigation is directed to the study of a narrower group of conditions—namely, to oligophrenia." A distinction is thus clearly made between (i) forms of mental retardation due to demonstrated or presumed biological defect and (ii) the backwardness in mental development due to psychosocial or other causes. Children showing retardation of the former type are regarded as fit subjects for educational segregation and pathophysiological study; those showing backwardness of the latter type are regarded as primarily problems for corrective pedagogy in regular classes.

Psychometric testing and intelligence quotients were abandoned decades ago in the Soviet Union, and children are ordinarily not assigned to special educational facilities for defectives until they have been observed and taught for a year in a regular class and are then thought to be incapable of mastering a regular curriculum. They are then examined by a multidiscipline commission, for validation of the assumption of biological deficiency, before they can be remanded to special classes. As a result, only a fraction of 1 percent of the children are diagnosed as retardates, whereas the common use, in the United States, of the criterion of two standard deviations from the I.Q. mean inevitably results in classification of at least 3 percent of our children as retardates. Furthermore, since the Stanford-Binet test, the intelligence test most widely used in the United States, was standardized on the basis of a white and somewhat middle-class population, percentages of supposed retardation may run as high as 20 or 30 percent in some age groups of our poor Negro urban population (*3*).

In cases of retardation with an organic basis there is much to suggest that, regardless of etiology, the most common biological result is diffuse and minimal brain damage or defect. This is notoriously difficult to diagnose in infants by conventional neurological examination, and must usually be deduced from a compromising pregnancy, birth, or medical history; early developmental lag; motor awkwardness; articulatory speech defects, and strabismus or other "soft" neurological signs.

Psychosocial deprivation is related to poverty, and the good things of life are not distributed parametrically on a bell-shaped curve: there is, in fact, a considerable skewing to the left. Neither is pathology distributed on a Gaussian curve, since there is no hypernormality to balance the incidence of birth injury or the hazards of prematurity so commonly encountered among the poor. That is why no intelligence test has ever been found, in practice, to yield a normal curve.

In the context of these considerations, the "normal" variations of innate intellectual capacity, which undoubtedly exist, appear to play a relatively minor role; the psychological aggravation that comes from the chronic frustrations of backwardness also exists, but its relative importance can be questioned. The problem could be dealt with more effectively if we made a sharper distinction between biological and nonbiological types of retardation. The biological types would include a small proportion of individuals with medically diagnosable conditions and a large proportion of really defective individuals whose precise trouble we cannot diagnose. The nonbiological types would involve a large element of poverty, physical neglect, and psychosocial deprivation and a small element of frustration, poor motivation, and demoralization.

JOSEPH WORTIS
Morris J. Solomon Clinic for Retarded Children, Jewish Hospital of Brooklyn, Brooklyn, New York

References

1. E. Zigler, *Science* **155**, 292 (1967).
2. M. S. Pevzner, *Oligophrenia: Mental Deficiency in Children* (Consultants Bureau, New York, 1961) (English translation).
3. S. M. Gruenberg, in *Mental Retardation*, H. A. Stevens and R. F. Heber, Eds. (Univ. of Chicago Press, Chicago, 1964); E. Ginzberg and D. W. Bray, *The Uneducated* (Columbia Univ. Press, New York, 1953).

1 February 1967