cant contribution. In fact, one of the most successful features of the symposium was the degree of interaction which was achieved and the feeling that new horizons had opened up.

The meeting was organized in cooperation with the John C. Eccles Laboratory of Neurobiology; the Departments of Biophysical Sciences, Mathematics, Computer Sciences, and the Center for Theoretical Biology of the State University of New York at Buffalo; and the Center for Visual Science of the University of Rochester.

In these times of financial stringency it was gratifying to receive support from University funds allocated through the Graduate School and supervised by the Research Foundation of the State University of New York at Buffalo. Some further support came from the Clynes Biocybernetic Foundation and the Departments of Biophysical Sciences, Mathematics, and Computer Science of SUNY.

The proceedings of the symposium will be published by Springer-Verlag in 1969 under the title *Information Processing in the Nervous System.*

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Mental Retardation Caused by Physical Trauma

The relationship of physical trauma to the production of mental retardation was considered 13–16 October 1968 at the University of Nebraska, Lincoln, by 45 participants including neurosurgeons, neuropathologists, obstetricians, pediatricians, and psychologists from the United States and overseas. It was the fourth of seven research conferences on the etiology of mental retardation recommended in 1962 by the epidemiology of postnatal and perinatal trauma, clinicopathologic correlations, and pathogenesis of traumatic damage to the developing brain.

The opening address underscored some of the problems confronting observers and research workers in the definition and estimation of the qualitative differences in function which are called mental retardation. The scope, incidence, and size of the patient population at risk or affected is not accurately known or defined.

All parameters of behavioral change are not examined in the standard testing of children, thus making it difficult to estimate the incidence of minor neurological impairment following physical trauma. The question was raised whether, with such tests, we uncover a diffuseness of representation or a diffuseness of lesion. The possibility that an elementary function might be spared at the expense of a general loss of higher functions was suggested.

The use of animal models is a direct approach to the problems, experimental reproduction of events with time and specific cortical areas are controlled. Work was reviewed which demonstrated the capacity of the young animal to compensate for large losses of brain mass, but with not always predictable results.

Results of experimental cortical ablation suggested that with the maturation of subcortical motor systems, the presence of the inhibitory functions of the localized areas of the cortex become increasingly important, although not necessarily a function, of the mass of cortical tissue removed. The inability of the operated adult or older infant subjects to do better on testing would appear to be intimately associated with the appearence of hyperactivity and distractability. These studies point to the need for both long-term clinical

evaluation and the assessment of dysfunction not quantitated by standard psychological testing.

Analyses of the cerebral pathology in subjects continuously institutionalized with histories of perinatal trauma or hypoxia were presented. The distinctive neuropathology of the premature was employed to differentiate the effects of anoxia from those of direct trauma. The cerebral cortex of the premature, unlike the full-term infant, is relatively resistent to hypoxia. A description was given of a possible sequence of hypoxia, venous stasis, and thrombosis leading to hemorrhagic infarction consistently located in the periventricular area in the premature or in the full-term infant with prenatal anoxia.

The role of trauma to the neck leading to cerebral damage was emphasized by showing, in 17 percent of perinatal deaths, one or both vertebral arteries were damaged. Despite the serious difficulties in the analysis of late effects of birth trauma, it was thought valid to correlate the distribution of lesions produced in adults by vertebral artery damage with functional deficits typical of cerebral palsy—cortical blindness in association with lesions of the calcarine cortex, ataxic cerebellar dysfunction, and temporal lobe epilepsy with scaring in the area supplied by the vertebral arteries.

The contrasts were reviewed between reaction to injury of the adult brain, typically the formation of dense glial scar, with that of the immature brain of the rat. The latter is characterized by rapid resorption of the necrotic tissue by lipid-filled macrophages, dissolution, and formation of a smooth walled cyst by the end of the second week. It was postulated that the more rapid removal of necrotic tissue might be due to the higher water content of the immature brain or a difference in metabolic activity of the macrophages. Of clinicopathologic significance is the fact that application of adult reaction patterns may lead to false impressions as to the etiology of diseases such as porencephaly, hydranencephaly, and aqueductal stenosis.

Most attractive were the elegant biophysical analyses of prenatal obstetrical events. The mechanical energy associated with uterine contractions may cause fetal distress by application of pressure directly to the fetal body (chiefly the vertex); by occlusion of the umbilical cord; and by impeding venous outflow or arterial inflow in the intervillous space. The application of sophis-

ticated techniques to the recording of alterations in amniotic fluid pressures, fetal heart rate, EEG, and acid-base balance, all rapidly related in time sequence to fetal-maternal exchange, provides a sensitive measure of physiologic events during labor. It would appear that anoxia due to physiologic effects on the infant's cardiovascular system is not due to trauma itself and is potentially responsible to pharmacologic manipulation.

Also presented was an analysis of simultaneous recordings from intrauterine pressure receptors and fetal heart rate. Decelerative patterns coinciding with the maximum peak of uterine contraction, called type I dips, are abolished by atropine and thought to be due to cephalic compression and a transient stimulation of the vagus nerve, but do not alter the Apgar score. Decelerative dips, type II, occurring after the uterine contraction are associated with hypoxia, acidosis, and a low Apgar score in the neonate. Monitoring to detect these changes has considerable clinical application.

Current epidemiologic studies of perinatal trauma all focus on the obstetrical history and the classical criteria for retardation. Drawing upon material from the Collaborative Study on the Etiology of Cerebral Palsy, Mental Retardation, and Other Neurological Disorders, offspring identified as having cerebral palsy or mental retardation seemed to provide evidence that obstetrical history has only a minor effect on WISC performance while racial-socio-cultural factors are associated with major deviations.

Other reports of data from the Collaborative Perinatal Research Project drew statistical correlations between the size of the maternal pelvis, position of the fetal head during delivery, and forceps delivery with neuropsychological outcome. It is suggested that as early as the time of birth, sufficiently lateralized neuropsychological differentiation appears to have taken place for injury to have an effect on the individual's intellectual capacity. The mean Binet scores of all reported groups was in the 104 to 110 range which would not fit accepted definitions of mental retardation.

The epidemiologic problems of postnatal head injury were introduced and a statement was made that a conservative estimate of the annual incidence of head injury in infants and children was 3 percent of the population under the age of 6 years. However, the incidence of significant postnatal trauma is low;

but there are no reliable estimates of postnatal injury as a cause of less than optimal performance in the noninstitutionalized.

Preliminary results from longitudinal interdisciplinary clinical study seeking to investigate the early and long-term effects of head injury in children from birth to 14 years showed a rapid regression of posttraumatic neurological deficits with a plateau reached by 3 months.

Impairments in adaptive behavior were considered in conjunction with intellectual deficits in children and adolescents following protracted coma due to accelerated concussion. The sequelae which are constant after moderate or severe concussion are mainly subjective: anxiety or irritability, difficulty with sustained mental concentration, impaired memory, distractability, perserveration, and excessive liability to fatigue. The severity of these symptoms is related directly to the period of posttraumatic amnesia. A newer group of more severely retarded patients are those resuscitated after the hypoxic insult of cardiorespiratory arrest.

As summarized it would appear that the perinatal problem is much larger than that of postnatal trauma. Although perinatal trauma and anoxia together may be causative in 10 percent of institutionalized retardates (Malamud) and birth trauma alone related to 16 percent of the diagnosed cases of retardation or cerebral palsy in 7-year-olds (Drorbaugh and Clifford), the much greater effect of racial-socio-cultural factors performance on standard I.Q. tests by the school age population deserves appropriate consideration. Postnatal trauma is a much less significant cause of severe retardation but the population at risk is enormous and the problem of head injury deserves both full demographic analysis and pragmatic efforts at prevention or amelioration of severity. The estimation of incidence of both perinatal and postnatal injury is obscured by the iceberg quality of cerebral dysfunction, and there is a need for long-term follow-up with attention to selected indices of complex performance. Animal models were considered to offer a challenging and hopeful field of investigation to outline precise behavioral changes with respect to neurological lesions defined as to location, extent, and time.

The delineation of the role of hypoxia and of direct trauma in the production of perinatal brain damage was a major achievement of the conference. The recurring theme of sequential hy-

poxia, acidosis, stasis, thrombosis, and infarction scored the crucial nature of obstetrical factors. The pathophysiology of placental function would seem deserving of attention, at least equal to that lavished on liver and kidney. Hon and Dodge were among those emphasizing the need for centers of perinatal biology capable of the application of sophisticated methodology to the biophysical and biochemical events of labor. Such centers would bring together obstetricians, pediatricians, physiologists, biochemists, biomedical engineers, and other specialists to focus on the most vulnerable period in the life of the child.

The meeting was sponsored by the U.S. Department of Health, Education, and Welfare, National Institutes of Health, National Institute of Neurological Diseases and Stroke (NINDS). The detailed proceedings of the conference will be published by NINDS.

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Calcified Tissues

Useful, new contributions to the field of calcified tissues were reported at the Sixth European Symposium on Calcified Tissues held in Lund, Sweden, 21–24 August 1968.

In the first session, specialized instruments offered hope for the future. The presentation by Hobdell (London) on scanning electron microscopy was of interest since it brought into perspective how bone is constructed at the ultrastructural level. Bones from different animals were fixed and extracted with fat solvents, mounted, and then scanned at magnifications up to 20,000. Such specimens showed a great deal of detail of the lining of the lacunae, the nature of the interlamellar material, and the pattern of mineral collagen fibers and fiber bundles. Removal of collagen by solvents resulted in a pattern of the mineral front alone, suggesting that it separates one collagen fiber bundle from the next one overlaying it.

Höhling (Münster) provided further evidence of the power of probe methods in establishing the nature of mineralization at the subcellular level. Hitherto, electron-probe analysis has been a somewhat crude technique, incapable of locating the site of the mineral deposi-