hyperfractionated radiotherapy, there was a statistically significant improvement in progression-free survival rate. Further studies seemed warranted. (Packer RJ et al. Hyperfractionated radiotherapy for children with brainstem gliomas: A pilot study using 7,200 cGy. Ann Neurol Feb 1990; 27:167-173).

COMMENT. Brainstem gliomas account for approximately 10% of all childhood central nervous system tumors and are the most resistant to therapy. High risk patients with tumors involving the brain stem diffusely rarely survive after conventional doses of radiotherapy. Hyperfractionated radiation therapy offers greater potential benefit.

DEVELOPMENTAL DISORDERS

CHIARI II MALFORMATION

The theories of the basic embryological defect that lead to the Chiari II malformation are reviewed and a unified theory proposed from the Division of Pediatric Neurosurgery, The Laboratory for Oculo-Cerebrospinal Investigation, The Children's Memorial Medical Center and Northwestern University Medical School, Chicago, IL. Chiari II malformation is almost invariably associated with myelomeningocele and a progressive hydrocephalus. Associated anomalies include small posterior fossa, Luckenschadel of the skull, caudal displacement of pons, medulla, and basilar artery, and upward herniation of the superior cerebellum. Syringomyelia occurs in 50%, and aqueduct stenosis, polymicrogyria, cortical heterotopia, and agenesis of the corpus callosum occur occasionally. Previous theories include the following: 1) Herniation of the posterior fossa contents resulting from supratentorial hydrocephalus with leakage of cerebrospinal fluid into the amnion and herniation of the hindbrain; 2) Traction theory suggesting that the caudal spinal cord may pull the cerebellum and medulla into the lower cervical canal because of tethering; 3) Dysgenesis of the hindbrain and developmental arrest; 4) Small posterior fossa due to mesodermal insufficiency and overgrowth of neuroepithelium causing a neural tube defect. The unified theory proposed by the authors incorporates the previous observation of Padget that leakage of cerebrospinal fluid is one factor in the cause of a small posterior fossa and emphasizes the role of distention of the embryonic and fetal ventricular system in normal cerebral development. The neural tube defect and defective occlusion are the developmental factors that cause the Chiari II malformation and the interrelated cerebral and skull anomalies. Altered inductive pressure on the surrounding mesenchyme is the cause of the Chiari II malformation and Luckenschadel whereas the lack of distention of the developing telencephalic ventricles results in cerebral anomalies, e.g., dysgenesis of the corpus callosum, cortical heterotopia, and polymicrogyria. Chiari II malformation is the result of a series of interrelated time dependent defects in the development of the ventricular system leading to multiple anomalies in brain development,

according to this theory. (McLone DG, Knepper PA. The cause of Chiari II malformation: A unified theory. Pediatr Neurosci 1989; 15:1-12).

COMMENT. Chiari described three types of cerebellar malformation which he had found in cases of congenital hydrocephalus. In type I the medulla was displaced downwards into the spinal canal and was covered by peg-like processes arising from the cerebellar hemispheres, their lower ends opposite the origin of the third cervical nerve roots. This anomaly was found in a girl of 17 in whom the malformation had caused no symptoms during life. Type II was a similar malformation of the lower parts of the cerebellum associated with an elongation of the fourth ventricle which extended into the spinal canal in a six month old baby with hydrocephalus. There were heterotopic nodules of gray matter in the walls of the lateral ventricles, the cerebellum was small, the pons was elongated, the medulla was entirely in the spinal canal, and the lower cranial nerves were elongated. Hydromyelia and meningomyelocele were associated. Chiari type III was a single case of cervical spina bifida in which there was herniation of the cerebellum through the bony defect, a form of occipital meningoencephalocele. Arnold's description of a case of meningomyelocele without hydrocephalus was less detailed than the earlier description by Chiari. Schwalbe and Gredig gave the name Arnold-Chiari malformation to type II which was always associated with meningomyelocele. For a full description of the historical and pathological aspects of Chiari malformations see Greenfield's Neuropathology, Baltimore, Williams and Wilkins Co. The section on malformations of the nervous system was written by Dr. R. M. Norman.

CORTICOSPINAL TRACT IN NEWBORNS

The maturation and function of the corticospinal and corticobulbar tracts in the human newborn are reviewed from the Departments of Paediatrics, Pathology and Clinical Neurosciences, University of Calgary Faculty of Medicine, Calgary, Alberta, Canada. The myelination of these tracts begins in late gestation but is not complete until two years of age. Functions attributed to these descending pathways in the full term human newborn include the following: 1) Development of passive muscle tone and resting postures; 2) Enhancement of suck and swallow reflexes; 3) Relay of cortical epileptic discharges; and 4) Inhibition of complex stereotyped motor reflexes ("subtle seizures"). The antagonistic balance between flexors and extensors are controlled by the subcortical spinal and corticospinal pathways. If the corticospinal tract is impaired as with perinatal asphyxia the infant assumes distal flexion and proximal extension postures which reflect the influence of subcorticospinal pathways when corticospinal tract antagonism is lacking. The "tonic seizures" of preterm infants with intraventricular hemorrhages are probably not true epileptic phenomena but rather episodes of decerebration. A weak suck, poor feeding, and impaired coordination