sufficiently developed to keep pace with verbal output. Another theory invokes a faulty auditory processing, that may respond to delayed auditory feedback therapy. Neuroimaging in adults who stammer may show hemispheric asymmetries, and neurochemical studies report both increased and decreased levels of dopamine. Risperidone, a D2 antagonist, may reduce severity of stammering in some adults. Treatment recommendations in children vary from an indirect approach, with changes in the child's environment to reduce demands, to direct intervention, targeting speech output and capacity. Complete recovery, with or without therapy, is common before adolescence. Approximately 74% of children who stammer recover, 89% of young females. Boys are affected most frequently; those with a late onset of DS have the poorest prognosis, especially when complicated by speech and language delay. (Ward D. The aetiology and treatment of developmental stammering in childhood. **Arch Dis Child** January 2008;93:68-71). (Respond: Dr David Ward, School of Psychology and Clinical Language Sciences, University of Reading, Reading RG6 6AL, UK).

COMMENT. Stammering is developmental (idiopathic) or acquired. Particularly in adults, it may be secondary to organic brain disease or of psychogenic origin. In children it is most likely developmental and usually transient, requiring only an adjustment of parental handling or school pressure. If persistent and complicated by seizures or other neurologic manifestations, investigations should include an EEG and MRI. In rare cases, the differential diagnosis may include Landau-Kleffner syndrome; symptoms are associated with an acute onset of loss of speech comprehension and auditory agnosia, seizures and/or a paroxysmal EEG with bitemporal discharges. (Morrell F et al. **Brain** 1995;118:1529-1546).

Structural and functional abnormalities of the motor system in developmental stuttering are reported by researchers at the Departments of Experimental Psychology and Clinical Neurology, University of Oxford; and Department of Psychology, University College London, UK. (Watkins KE, Smith SM, Davis S, Howell P. **Brain** January 2008;131(1):50-59). Using functional and diffusion imaging, motor and language areas were examined in brains of 12 young subjects (aged 14-27 years;avg 18 years; 1 left-hander) who stutter. During speech production, people who stutter show overactivity relative to controls in the anterior insula, cerebellum and midbrain bilaterally and underactivity in the ventral premotor, Rolandic opercular and sensorimotor cortex bilaterally and Heschl's gyrus in the left hemisphere. The overactivity in the midbrain, at the level of the substantia nigra, red and subthalamic nuclei, is consistent with a previous report of excess dopamine in adults who stutter. Areas with underactivity are associated with articulation and speech production, and show loss of white matter. Stuttering is related to disruption in neural systems that support the execution of fluent speech.

BRAINSTEM MALFORMATIONS

Malformations of the brainstem in 138 patients identified aver a 10 year period are classified according to MRI findings and by embryological cause in a study at University of California at San Francisco, and University of Chicago, IL. The pons was involved in 114, midbrain in 45, and medulla in 14. More than 1 region was affected in 53 patients. Malformations were classified in four groups: 1) disorder of brainstem segmentation or induction; 2) segmental hypoplasia; 3) postsegmentation malformation (associated with migration abnormalities); and 4) abnormal cortical organization. *Segmentation anomalies*

included short pons, short midbrain/long pons with large cerebellum, and thick, short medulla. *Segmental hypoplasia* involving the pons in 59 patients was associated with microcephaly in 34. *Postsegmentation anomalies* included midbrain enlargement, enlarged quadrigeminal plates, midline clefts, 33 with congenital muscular dystrophies and O-glycosylation defects (10 with Walker-Warburg syndrome, 7 with muscle-eye-brain disease, and 2 with Fukuyama CMD), and 19 with Joubert's syndrome and the characteristic molar tooth malformation. *Associated cortical organization abnormalities* included polymicrogyria and cerebellar hypolasia with pontine hypoplasia in 11 patients. Disorders involving the cranial nerves usually had no brainstem abnormalities on imaging other than hypoplasia of the affected nerves. (Barkovich AJ, Millen KJ, Dobyns WB. A developmental classification of malformations of the brainstem. **Ann Neurol** Dec 2007;62:625-639). (Respond: Dr Barkovich, Neuroradiology Room L371, University of California at San Francisco, 505 Parnassus Avenue, San Francisco, CA 94143).

COMMENT. Brainstem malformations appear to be more common than generally recognized. This study and proposed classification should alert neurologists and radiologists to the diagnosis of congenital malformation of the brainstem in infants and children with nonprogressive cranial nerve and long tract signs. We can look forward to an anticipated separate account of cerebellar malformations from the same institutions. Intrauterine ischemic atrophy rather than a primary developmental malformation is suggested in some reports of brainstem lesions presenting with congenital apnea and failure of central respiratory drive (Cortez C, Kinney HC. **J Neuropathol Exp Neurol** 1996;55:841-849; Reviewed by Sarnat HB. Recent advances in congenital malformations. In: **Progress in Pediatric Neurology III**, Chicago, PNB Publ, 1997;365-369).

ACUTE BRAINSTEM SYMPTOMS WITH CHIARI TYPE 1 MALFORMATION

Two children who presented with rapidly worsening neurological symptoms attributable to a previously undiagnosed Chiari malformation Type 1 are reported from Children's Hospital, Birmingham, AL. One patient became hypopneic and dysphagic and developed a right hemiparesis in less than a 48-hour period. Another patient presented with a rapidly worsening right hemiparesis, ataxia, and anisocoria. MRI revealed the Chiari 1 in both patients, and a syrinx was also identified in the second patient. Following surgical posterior fossa decompression, symptoms immediately improved. (Wellons JC III, Tubbs S, Bui CJ, Grabb PA, Oakes WJ. Urgent surgical intervention in pediatric patients with Chiari malformation Type 1. Report of two cases. **J Neurosurg: Pediatrics** 2007;107(1). (Respond: Dr W Jerry Oakes, Division of Neurosurgery, Section of Pediatric Neurosurgery, Children's Hospital, Birmingham, AL).

COMMENT. Acute presentation of Chiari malformation Type 1 (CM-1) is rare, especially in children. Chiari 1 should be included in the differential diagnosis of acute onset of brainstem or long tract signs. In a study of CM-1 at the Children's Hospital, Birmingham, UK, abnormalities of the skull base were identified by MRI measurements, indicative of a mesodermal defect. (Sgouros S et al. **J Neurosurg (3 Suppl Pediatrics)** 2007;107:188-192).