neuropathies within 3 weeks of "inactivated" influenza vaccination had a delayed onset of ADEM 3 months post-vaccination.

A case of ADEM is reported in a 34-month-old boy who presented with a seizure and left-sided weakness 5 days after vaccination against novel influenza A (H1N1). Following IV corticosteroids, symptoms improved and he recovered without neurologic sequelae (Lee ST et al. An adverse event following 2009 H1N1 influenza vaccination: a case of acute disseminated encephalomyelitis. **Korean J Pediatr** 2011 Oct;54(10):422-4). A PubMed search found 5 reports of ADEM following 2009 H1N1 vaccination, one occurring simultaneously with Guillain-Barre disease (Hoshino T et al. **Intern Med** 2012;51(12):1595-8). With increased demand for mandatory vaccination for health-care workers, recognition of the occasional association of ADEM with influenza vaccine should prompt early diagnosis and steroid therapy.

DEVELOPMENTAL DISORDERS

DIAGNOSTIC ALGORITHM FOR MICROCEPHALY

Investigators from Addenbrooke's Hospital, Cambridge, UK, provide a diagnostic structure to follow when presented with a child with microcephaly. An occipital-frontal-circumference (OFC) of >3SD below the age and sex expected is the definition used for microcephaly. "Primary" microcephaly is present at birth and "secondary" microcephaly develops after birth. Serial OFC measurements that follow the growth curve suggest a primary microcephaly, whereas an OFC that falls relative to the growth curve is usually a secondary microcephaly. In *primary* cases check for maternal and environmental factors including the TORCH screen, MRI, and fetal brain imaging. Cases with dwarfism and those with dysmorphic features and/or congenital anomalies may be recognized by phenotype (e.g. Cornelia de Lange syndrome- synophrys, dwarfism, limb anomalies) or may require cytogenetic testing. *Secondary* microcephaly cases may be static or progressive. The majority of chromosome disorders are associated with developmental delay and secondary microcephaly (e.g. Miller-Dieker syndrome caused by deletion of chromosome 17p13.3). Larger deletions are associated with a more severe phenotype of lissencephaly/pachygyria, and smaller deletions involve the LIS gene and a less severe form of lissencephaly. Rubinstein-Taybi syndrome is a Mendelian disorder causing secondary microcephaly and learning disorders. The diagnosis is clinical (distinctive facies, broad thumbs/big toes and postnatal growth retardation) and is confirmed by mutations in the CREBBP, EP300 or SRCAP gene.

If secondary microcephaly is associated with progressive neurologic findings, metabolic diseases should be considered. Genetic disorders such as Rett, PEHO, Cockayne, and Cohen syndromes are examples of secondary microcephaly where diagnosis by DNA testing is available. (Woods CG, Parker A. Investigating microcephaly. **Arch Dis Child** 2013 Sep;98(9):707-13). (Resp.: Dr. C. Geoffrey Woods. Clinical Genetics, Addenbrooke's Hospital, Cambridge, UK. E: cw347@cam.ac.uk).

COMMENT. A knowledge of neurological syndromes is helpful in the differential diagnosis of microcephaly. (Millichap JG. **Neurological Syndromes : A Clinical Guide to Symptoms and Diagnosis.** New York: Springer, 2013:279).