

Comments for the IACC Meeting December 12, 2008

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Thank you, first and foremost, for allowing me to speak at the meeting held November 21, on the need to investigate: (1) impairment of auditory function as an impediment to learning to speak, and (2) clamping of the umbilical cord at birth, before the first breath, as a factor in cases of neonatal “respiratory depression” that could lead to auditory system impairment.

Following are my thoughts on the issues of vaccine injury, regression, Kanner autism, co-morbid conditions, male vulnerability, and genetics:

Vaccine injury should be included in the research plan, but only with a focus of how thimerosal and other additives might impair brain systems important for language development. The paper by Oyanagi et al. (1988) might be a start [1]. Can the roles of dopamine, serotonin, GABA, methylation and other factors in neurotransmission be shown to be important for synaptogenesis and maturation of the temporal and frontal language areas of the cerebral cortex? The papers by vonHungen et al. (1975) and Kungel & Friauf (1995) could be relevant [2, 3]. Focus on how the insult at time of vaccination affects the brain, and how subsequent maturation might be derailed. Vaccination is important for the health of all children, but why is vaccination needed in the newborn nursery? It might be good to return to the old schedule.

Regression may be too subjective to be taken as solid evidence that vaccine injury caused a sudden loss of preexisting language and motor skills. We watched with baited breath the development of our son Conrad. His brother, Anders, was seriously delayed in rolling over, sitting up, crawling, standing, and walking; and Dr. Charles Barlow at Children’s hospital told me when he was 21 months old, “He has a mild form of cerebral palsy.” Conrad was on-time with all milestones, and he began speaking with clear pronunciation right on time, and in complete sentences! These sentences were exact repetitions of things he had heard others say, and we thought it was because Anders (who learned to read at 2 years + 2 months of age) loved to read to Conrad and coach him in speaking – I had done a lot of coaching of Anders because of his slow development of speech and his poor pronunciation. Conrad was also very musical and just before he turned 2 years old could sing all the Christmas carols, including the Twelve Days of Christmas! What a shock when his nursery school teacher suggested at age 3 that he be evaluated for autism. If I had known about regression, I might have grabbed onto this idea as an explanation of Conrad’s failure to develop after the age of 2 or 3. Autism is still not easy to diagnose before 2 to 4 years of age. Regression is a very fuzzy term.

Kanner (1943) began his famous paper stating:

“Since 1938 there have come to our attention a number of children whose condition differs so markedly and uniquely from anything reported so far, that each

case merits – and, I hope, will eventually receive – a detailed consideration of its fascinating peculiarities.” [4, p217]

The autistic disturbances of affective contact that he described were not seen as commonly as today, and the fathers of four of these patients were also psychiatrists [4, pgs 229, 230, 232, 237]. Barr (1898) reviewed the literature of his day on the rare finding of echolalic speech, which Kanner later referred to as metaphorical and irrelevant [5, 6]. Barr claimed to have examined 1525 cases of mental deficiency but only 2 patients manifested echolalia. Barr’s patient, Kirtie, is similar to the cases reported by Kanner – see pp25-29 of Barr’s paper, which I have posted at <http://www.conradsimon.org/BarrEcholalia1898ocr.pdf>. DeSanctis (1908) and Heller (1908) reported cases of “dementia infantilis” using the nomenclature of Kraepelin [7, 8, 9]. Bender (1947) and Yakovlev (1948) described their work with schizophrenic children using the nomenclature of Bleuler [10, 11, 12]. Bender (1955) and Putnam (1955) wrote about psychosis in childhood as a rare condition [13, 14]. Landau and Kleffner (1957, 1960) described aphasia with seizure disorder and damage found post mortem in the auditory pathway, especially the medial geniculate bodies [15, 16]. Childhood psychosis with loss or abnormal development of language was rare in the past. The current increased prevalence appears to be real.

Co-morbid conditions like GI disturbances are common in autism (I work with mentally ill adults, and see GI disorders as a frequent co-morbid condition). Conrad had horrible colic and projectile vomiting during his first year. He also had a “collapsing trachea” especially during sleep, which was often frightening. I think he could have been a case of sudden infant death. Conrad was pale and life-less at birth, slow to begin crying, and he developed jaundice a day or two after birth. GI problems can be the result of brainstem impairment. The article by Windle (1969) on asphyxia at birth made me aware that brainstem damage (especially in the auditory pathway) is the result of the kind of asphyxia Conrad suffered at birth [17]. Multi-organ injury is a common finding in infants who suffer asphyxia at birth [18]

Male/female differences were a surprise finding in my dissertation research on neonatal asphyxia in the rat [19]. At a meeting of the Fetal and Neonatal Society in 2006, the greater vulnerability of males to any perinatal insult became one of the important themes of the meeting. My poster presentations on the historical context of the Apgar score are posted online at <http://www.inferiorcolliculus.org/fnps.html>.

Genetic disorders are among the many medical conditions associated with autism. I have listed some at <http://www.conradsimon.org/WorkingPaper2003.html#GeneticPredispositions>. Note that phenylpyruvic and phenylacetic acid metabolites were detected by Folling (1934) before DNA had been isolated [20]. The abnormal metabolites produced by faulty phenylalanine hydroxylase, as well as excess phenylalanine could cause breakdown of the blood-brain barrier, and as is the case with bilirubin, the brainstem nuclei of high metabolic rate are more likely to be affected, especially the auditory system [20, 21]. The strategic plan should focus on loci in the brain, not loci of genes on chromosomes.

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Anders reading stories to Conrad, which Conrad learned to recite verbatim. We came to believe that Conrad's traumatic anoxic birth had not affected his development, but then at age 3 his nursery school teacher suggested we have him evaluated for autism. He fit the description of classic Kanner autism. – more at www.conradsimon.org