Oxygen insufficiency at birth

The inferior colliculi were prominently damaged in monkeys subjected to asphyxia at birth [1, 2]. This damage was part of what Myers (1972) referred to as "a monotonous rank order of brainstem nuclei" [2]. Impairment of brainstem structures was not viewed with an appropriate sense of alarm. A lapse in respiration at birth is generally considered harmless. In experiments with monkeys resuscitation was only required after about eight minutes of asphyxia at birth. Four to six minutes of oxygen deprivation at birth may go completely unnoticed, especially if the Apgar score by five minutes after birth appears adequate.

One of the problems, still today, is that outcomes of difficult birth and early developmental delay are not investigated into adulthood. No one wants to admit to low Apgar scores, and parents are admonished if they worry about developmental delay. Pediatricians can stipulate "normal limits" for stages of motor and language development, but who can be complacent about their own child's delay in learning to roll over, sit, crawl, stand, walk, or talk?

Even in monkeys who recovered from asphyxia without the need for resuscitation, Faro and Windle (1969) found “transneuronal degeneration” (or decreased numbers of neurons in wide areas of the brain) following survival for months or years [3]. No visible damage to the brain had at first been detected in monkeys subjected to briefer periods (4 to 6 minutes) of asphyxia. The over-optimistic notion of infant "brain plasticity" needs to be questioned. The evidence indicates just the opposite. Every effort must be made to avoid injury or compromise of the infant brain. The paper by Faro and Windle was published in the journal Experimental Neurology, and is now available online as a PDF file. I have included it here because of the great importance of the data reported.

Monkeys subjected to asphyxia at birth were developmentally delayed, but appeared to "catch up." Windle (1969) did warn that although the asphyxiated animals became more normal with time, their brains were damaged, this damage was permanent, and disrupted maturation of the cerebral cortex [1]. Lack of good manual dexterity was a persistent problem in monkeys that otherwise appeared to fully recover. Manual dexterity is often a problem for children with autism. Large, laboriously produced hand-writing is often observed even in high-functioning adults with autism [4].

Manual dexterity develops and comes under control from an area in the precentral gyrus of the cortex that is close to the locus from which movements of the tongue and other oral components are initiated (see figure 16). Fingers, thumb, lips, jaw and tongue are controlled from an area of the precentral gyrus adjacent to the speech production region of the frontal cortex (Broca’s area) [5]. Maturation of these areas of close proximity in the cerebral cortex may depend upon integrity of interconnected circuits of the brainstem [6].

The major difference between monkeys and humans is that monkeys do not learn to speak. Therefore, damage of the auditory system by asphyxia at birth may not interfere that much with development of a monkey. But language development is of primary importance for the
human child. The importance of auditory acuity for normal language development should go without saying. Children with autism do exhibit problems with hearing; the speech they acquire lacks normal intonation. Echoing of prefabricated phrases may sound like perfect imitations, but failure to change intonation to fit a new context is as bizarre as failing to reword the phrase.

Damage to the auditory system is serious for a human infant, and should be recognized as a possible consequence of any complication at birth. That the damage of brainstem auditory nuclei is not static, but affects maturational growth throughout the brain, makes it even more imperative that every effort be made to prevent any lapse in respiration at birth. Low Apgar scores at birth should be regarded as ominous. Early recognition and intervention for developmental delays cannot be expected to make up for the effects of oxygen insufficiency at birth.

References
Figure 16: Homunculus – illustrating the locations of motor control in the cerebral cortex. From http://www.brainconnection.com/ web resource of http://www.positscience.com/