

BRAIN MECHANISMS OF HUMAN ACOUSTIC COMMUNICATION: A PHYLOGENETIC APPROACH AND ITS ONTOGENETIC IMPLICATIONS

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A recent comparative analysis of the neurobiological bases of vocal behavior suggests the basal ganglia to provide the crucial phylogenetic platform for the integration of primate-general mechanisms of acoustic communication with the human-unique faculty of articulate speech (Ackermann et al., 2014). More specifically, structural refinement of the basal ganglia and their connections with the cerebral cortex (corticostriatal loops) – driven, conceivably, by human-specific mutations of the *FOXP2* gene – might have represented a pivotal step towards the emergence of spoken language in our hominin ancestors, allowing the recruitment of the larynx as an articulatory organ. Though the notion that ontogeny recapitulates the adult stages of phylogeny has been dismissed decades ago, the constraints that canalized spoken language evolution must, nevertheless, be expected to have an impact upon speech acquisition. Against this background, a closer look at the contribution of the basal ganglia to the ontogenetic development of vocal behavior appears warranted.

Since congenitally deaf and blind children are capable to cry and laugh, even in the absence of tactile / haptic exploratory capabilities, these vocalizations have been classified – in a human-ethological perspective - as innate “fixed action patterns”, and a hierarchically organized network of, presumably, mammalian-general brainstem control mechanism appears to orchestrate the acoustic components of multimodal emotional displays (Jürgens, 2002). Besides innate affective utterances, the vocal behavior of neonates encompasses speech-related sounds in terms of quasi-resonant nuclei (quasi-vowels), produced in a laryngeal state of “normal phonation”, i.e., the supraglottal vocal tract being “at rest”. As a

rule, thus, the physiological prerequisites for the elaboration of a fully operational glottal sound source in our species – such as elaborate monosynaptic cortical projections to the brainstem nuclei that steer the laryngeal muscles (Kuypers/Jürgens hypothesis; Fitch et al., 2010) – must have been established already during (late) fetal life. By contrast, the production of mature consonant-vowel syllable sequences in terms of canonical or reduplicated babbling only arises months later (Oller, 2000). This temporal delay between an early development of speech-related laryngeal functions – starting already at birth – and a protracted emergence of adequately sequenced articulatory (supralaryngeal) constrictions parallels, within some limits, the available data on the maturation of the central nervous system. First, the corticostriatal circuits – especially, the efferent projections of the putamen to its pallidal targets – show a later onset and a prolonged time course of myelin formation as compared to the motor and sensory roots of the cranial nerves as well as the parastriatal aspects of the posterior limb of the internal capsule, encompassing the corticobulbar fibers (Gilles & Nelson Jr., 2012). In addition, second, the striatum displays a still “irregular cytoarchitectonic organization” at the time of delivery, by contrast to an already “very mature” texture in neonates of the infratentorial cranial nerve nuclei, engaged in the innervation of vocal tract muscles (Kostović, 1993).

In conclusion, preverbal vocal behavior appears to evolve across two levels: (i) neonates already master relatively well the operation of a glottal sound source, (ii) the subsequent myelogenetic and cytoarchitectural elaboration of corticostriatal networks then seems to allow for the implementation of syllabic vocal tract movement sequences, based upon the precise adjustment of laryngeal functions and supralaryngeal articulatory excursions.

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