Lenneberg's Contributions to the Biology of Language and Child Aphasiology: Resonation and Brain Rhythmicity as Key Mechanisms

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This paper aims to re-evaluate the legacy of Eric Lenneberg's monumental *Biological Foundations of Language*, with special reference to his biolinguistic framework and view on (child) aphasiology. The argument draws from the following concepts from Lenneberg's work: (i) language (latent structure vs. realized structure) as independent of externalization; (ii) resonance theory; (iii) brain rhythmicity; and (iv) aphasia as temporal dysfunction. Specifically, it will be demonstrated that Lenneberg's original version of the critical period hypothesis and his child aphasiology lend themselves to elucidating a child aphasia of epileptic origin called Landau-Kleffner syndrome (LKS), thereby opening a possible hope for recovery from the disease. Moreover, it will be claimed that, to the extent that the language disorder in LKS can be couched in these terms, it can serve as strong "living" evidence in support of Lenneberg's critical period hypothesis and his view on child aphasiology.

Keywords: (child) aphasiology; brain rhythmicity; critical period hypothesis; latent and realized structures; resonance theory

1. Introduction

Boeckx & Longa aptly and succinctly describe the value of Eric Lenneberg's pioneering and seminal work *Biological Foundations of Language* published in 1967 as being "regarded as a classic" and add:

> Like all classics, it deserves to be re-read at regular intervals, not only to appreciate the success (and limitations) of previous attempts at a synthesis among fields, but also to learn things that we all too often forget.
>
> (Boeckx & Longa 2011: 255)

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It is not an exaggeration to claim that Lenneberg's book set the highest standard of interdisciplinary biolinguistic investigation and has served as an unmatched exemplar in the field of biolinguistic research broadly construed. Even a cursory look at the table of contents of the book does not fail to strongly impress us with the fact that Lenneberg already covered virtually all the relevant topics that scholars in the current field of biolinguistics are still actively pursuing (see, e.g., Jenkins 2000, Boeckx & Grohmann 2013). The issues explored are the anatomy of the human body and brain, language and cognition, language evolution and genetics, the critical period for first language development, and language disorders—among many others.

One of Lenneberg's most significant contributions to the field of biolinguistics in particular and to the field of neurosciences in general is his insight into the significance of studying cognitively handicapped children including child aphasics as a means of uncovering the nature of human language and its brain mechanisms (see Lenneberg 1967: 304–326). Most concisely summarised in the following passage:

> It is often said that it is difficult enough to understand the development of behavior in the healthy individual and that we should, therefore, not complicate our task by trying to understand at the same time behavioral development in the presence of disease. Such a statement is based on the false assumption that disease results in more complicated behavior. However, we may consider it axiomatic that disease processes *do not usually add to the complexity of structure* of behavior.
>
> (Lenneberg 1967: 304–305; emphasis in original)

Accordingly, the main purpose of this paper is to re-visit Lenneberg's original version of the critical period hypothesis and his view on (child) aphasiology, which have been often forgotten, sometimes ignored or even misunderstood, in order to highlight their importance, relevance, and validity in exploring the nature of a certain child aphasia of epileptic origin called Landau-Kleffner syndrome (LKS; Landau & Kleffner 1957). It will be argued that LKS provides strong "living" evidence demonstrating the validity of Lenneberg's original version of the critical period hypothesis and his view on (child) aphasiology, to the extent that they are conducive to illuminating the very nature of the language disorder in LKS.

The structure of this paper is as follows: Section 2 deals with Lenneberg's view on human language, addressing the two fundamental underlying assumptions: brain-internal language as language capacity and its independence from externalization. I will also review some crucial concepts closely related to these assumptions: Lenneberg's first language development model, critical period hypothesis, and brain rhythmicity for speech production. Section 3 then is more specifically concerned with Lenneberg's view on child aphasia and its application to LKS. I will argue that Lenneberg's critical period hypothesis and child aphasiology can benefit our understanding the nature of the language disorder in LKS, opening up a novel possibility for an effective non-invasive medical intervention. Section 4 concludes.

2. Language Capacity and Externalization

2.1. Lenneberg's Critical Period Hypothesis and Related Concepts

In this subsection, I will lay out the two fundamental assumptions that underlie Lenneberg's (1967) version of the critical period hypothesis: (i) brain-internal language as language capacity; (ii) language capacity as independent from its externalization.

2.1.1. Brain-Internal Language as Language Capacity

Lenneberg conceives of human language as a brain-internal biological system and distinguishes between two biological levels of human language: latent structure and realized structure, explaining that

> the unfolding of language is a process of actualization in which latent structure is transformed into realized structure. The actualization of latent structure to realized structure is to give the underlying cognitively determined type a concrete form. (Lenneberg 1967: 376)

This process can be illustrated as in figure 1.[1]

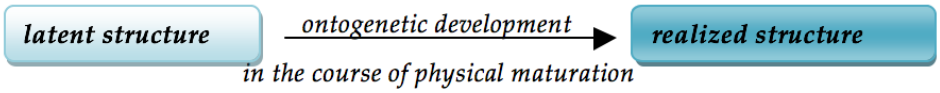

Figure 1: The actualization process from latent structure to realized structure in accordance with Lenneberg's (1967) formulation.

Crucially, he notes that

> [t]he actualization process is not the same as 'beginning to say things.' In fact, it may be independent from certain restraints that are attending upon the capacity for making given responses. Actualization may take place even if responses are peripherally blocked; in this case actualization is demonstrable only through signs of understanding language. (Lenneberg 1967: 376)

Let me emphasize at this juncture that this point is extremely important in correctly understanding a certain childhood aphasia of epileptic origin which I will be addressing in section 3.

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[1] Lenneberg states the following in a footnote on the same page:

> This formulation might be regarded as the biological counterpart to what grammarians have for centuries called universal and particular grammar. Latent structure is responsible for the general type of all features of universal grammar; realized structure is responsible both for the peculiarities of any given statement as well as those aspects that are unique to the grammar of a given natural language. (Lenneberg 1967: 376)

It is remarkable that about five decades ago Lenneberg already conceived of human language as a biologically determined cognitive system in the brain. The underlying cognitively specified latent structure and the realized structure basically correspond to the initial state of I-language and the steady state of I-language in generative grammar, respectively. Indeed, Lenneberg touches upon the notion of "universal grammar" (i.e., latent structure; see also fn. 1) and claims that

> universal grammar is of a unique type, common to all men, and it is entirely the by-product of peculiar modes of cognition based upon the biological constitution of the individual. (Lenneberg 1967: 377)

More specifically, Lenneberg points out that the latent structure is determined by the following biological properties of the human form of cognition:

> The forms and modes of categorization, the capacity for extracting similarities from physical stimulus configuration or from classes of deeper structural schemata, and the operating characteristics of the data-processing machinery of the brain (for example, time-limitations on the rate of input, resolution-power for the analysis of intertwined patterns such as nested dependencies, limits of storage capacities for data that must be processed simultaneously, etc. (Lenneberg 1967: 375)

Lenneberg (1967: 375–376) maintains that maturation of a child will bring cognitive processes to what he calls "language-readiness" as a state of latent structure. He expounds on the claim by saying that

> it might be more fruitful to think of maturation, including growth and the development of behavior such as language as the traversing of highly unstable states; the disequilibrium of one leads to rearrangements that bring about new disequilibria, producing further rearrangements, and so on until relative stability, known as maturity, is reached.
>
> (Lenneberg 1967: 376)

Accordingly, on Lenneberg's conception of maturation, including the growth/development of human language, the child will go through various states of cognitive disequilibria until reaching a state of cognitive relative equilibrium/stability, as depicted in figure 2.

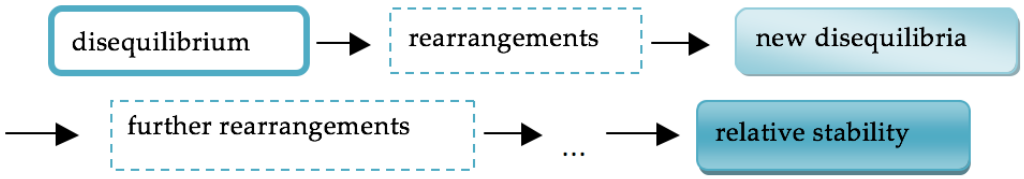

Figure 2: Schematic of the traversing of various cognitive states in maturation according to Lenneberg (1967).

As he remarks:

> Language-readiness is an example of such a state of disequilibrium during which the mind creates a place into which the building blocks of language may fit, (Lenneberg 1967: 376)

where the created place corresponds to what he refers to as a "biological matrix" (p. 394) of the latent structure, which virtually corresponds to Chomsky's (1965) language acquisition device (LAD) for first language acquisition in generative grammar. Lenneberg also adds:

> The realized structure or outer form of the language that surrounds the growing child serves as a mold upon which the form of the child's own realized structure is modeled. (Lenneberg 1967: 377)

Hence, Lenneberg's first language development model can be depicted as shown in figure 3.

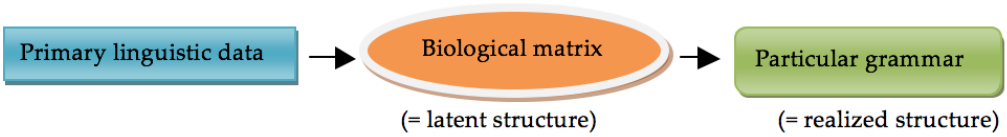

Figure 3: Schematic of Lenneberg's (1967) biological matrix for first language acquisition.

Furthermore, Lenneberg (1967: 383) clearly states that the biological capacity for utilizing the primary linguistic data as the language input is to be regarded as "breaking down of elements and resynthesizing them" to construct a particular grammar on the basis of the biological matrix of the latent structure. In fact, regarding the process of first language acquisition undertaken by the child by employing a biological matrix, Lenneberg furnishes the following explanation:

> Maturation brings cognitive processes to a state that we may call *language-readiness*. The organism now requires certain raw materials from which it can shape building blocks for his[/her] own language development. The situation is somewhat analogous to the relationship between nourishment and growth. The food that the growing individual takes in as architectural raw material must be chemically broken down and reconstituted before it may enter the synthesis that produces tissues and organs. The information on how the organs are to be structured does not come in the food but is latent in the individual's own cellular components. The raw material for the individual's language synthesis is the language spoken by the adults surrounding the child. The presence of the raw material seems to function like a releaser for the developmental language synthesizing process.
>
> (Lenneberg 1967: 375; emphasis in original)

Thus, Lenneberg's conception of the relation between primary linguistic data and language growth can be illustrated as in figure 4 which shows this relation in direct comparison with that between nourishment and physical growth.

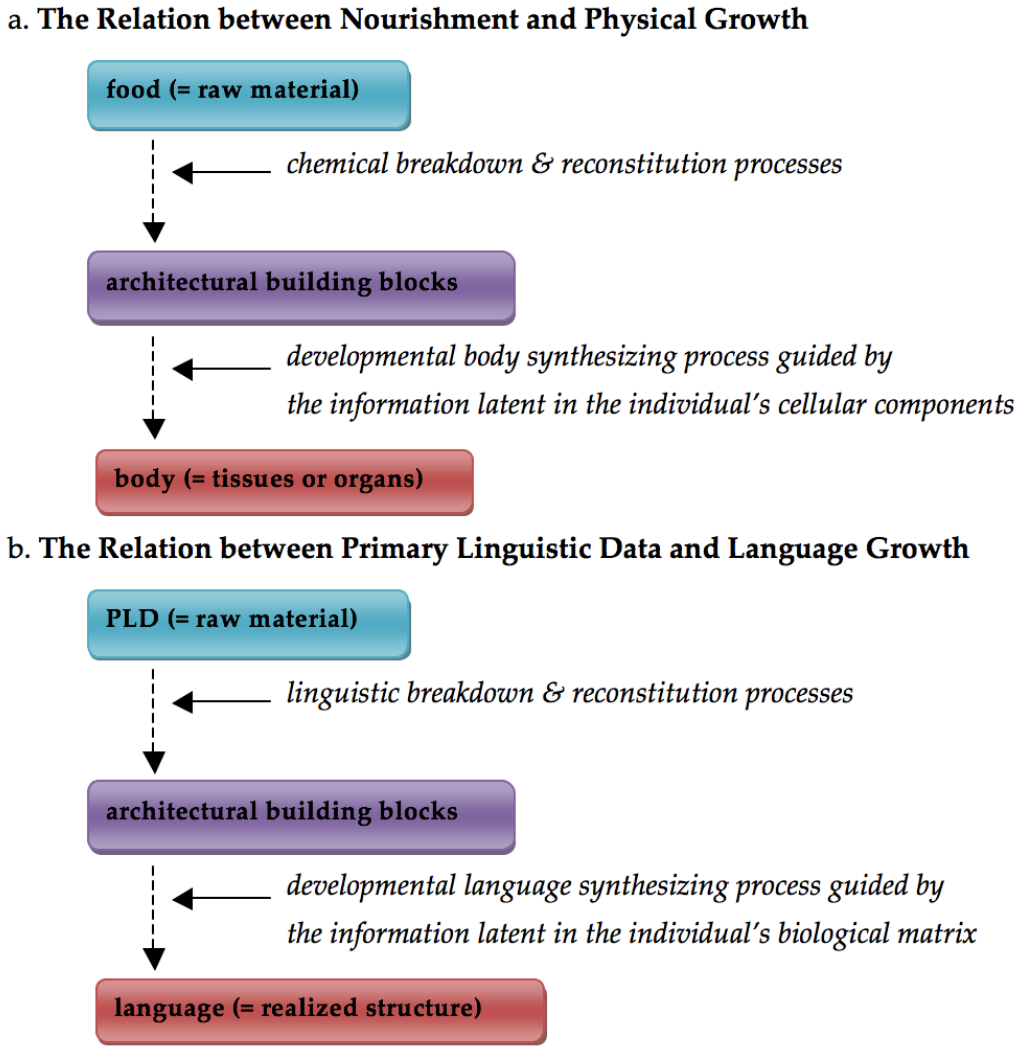

Figure 4: Comparison between food and language. The relation between nourishment and physical growth in (a), as well as primary linguistic data and language growth in (b).

While the architectural building blocks in the case of physical growth can be assumed to be proteins, the counterparts in the case of language growth can be regarded as lexical items LIs (rather than words) entering into structure-building in the sense standardly used in generative grammar. As investigation into this issue itself deserves a separate paper, I will not pursue it further here.

2.1.2. Language Capacity as Independent From Its Externalization

As summarized in Chomsky (2013, 2016, 2017a, 2017b), the generative enterprise of investigation into the nature of human language has reached the clear conclusion that we should regard "language as meaning with sound (or some other externalization, or none)" on the basis of empirical evidence concerning structure-dependence over linear order in syntax and semantics, reversing Aristotle's (1938)

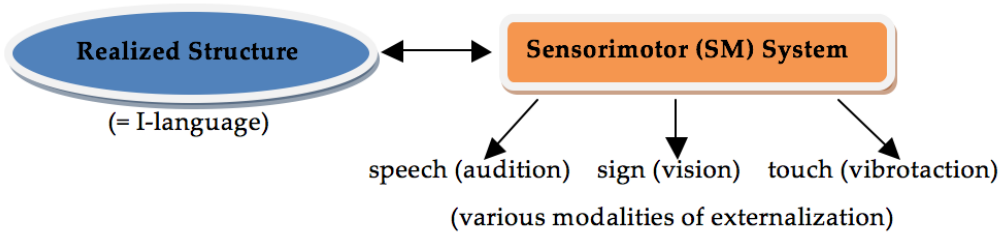

Figure 5: Architecture of human language according to Lenneberg (1967). Given that the realized structure can be actualized from the latent structure through the Tadoma method, vibrotaction is included as another modality for externalization.

classic dictum that "language is sound with meaning" (see also Berwick et al. 2013, Everaert et al. 2015).

In this connection, it is worth noticing that Lenneberg (1967) already held an idea similar to Chomsky's (1965, 1967) distinction between competence and performance (see Lenneberg 1967: 284 for reference to Chomsky 1967). Lenneberg considers the brain-internal language system as the capacity for language, which is independent from its externalization. He asserts that externalization, for instance, by speech, is "accessory" to the development of the language capacity in light of considerations of various cases of child language disorders (see also Lenneberg 1962, 1964 for more detailed discussion on child language disorders).[2] For instance, the following excerpt on a case of a child with congenital anarthria clearly illustrates this point:

> Congenital anarthria, as reported here, is a rare condition, but the case is by no means unique, and the discrepancy between speech skills and the capacity for understanding may, indeed, be observed in every child. The theoretical importance of the extreme dissociation between perceptive and productive ability lies in the demonstration that the particular ability which we may properly call *'having knowledge of a language'* is not identical with *speaking*. Since knowledge of a language may be established in the absence of speaking skills, the former must be prior, and, in a sense, simpler than the latter. Speaking appears to require additional capacities, but these are accessory rather than criterial for language development. (Lenneberg 1967: 308; emphasis in original)

With respect to the independence of the capacity for language from input modalities, Lenneberg is perceptive enough to point this out in chapter 8:

> Language acquisition is not dependent in man upon processing of acoustic patterns. There are many instances today of deaf-and-blind people who have built up language capacities on tactually perceived stimulus configurations. (Lenneberg 1967: 330–331)

(Also see C. Chomsky 1986 and Gleitman & Landau 2013 for intriguing discussion on language acquisition by deaf-blind children on the basis of a vibrotactile method

[2] Note that, as mentioned above, this view is virtually in line with the recent thesis in the minimalist program that externalization is altogether ancillary to I-language (see Chomsky 2013, 2016, 2017, Berwick & Chomsky 2016 and references therein).

of speech perception called the Tadoma method of speechreading.) Furthermore, the case of sign as a relevant modality is also now familiar to us due to remarkable progress in investigation of sign languages (see Petitto et al. 2016 and references therein *inter alia*).

In sum, Lenneberg's picture of human language architecture can be depicted as shown in figure 5 above.

2.2. Lenneberg's First Language Development Model

2.2.1. Critical Period Hypothesis

One of the integral components of Lenneberg's biolinguistic framework lies in the very notion of a "critical period" for first language development. Although the concept of the critical period for first language development was originally entertained by Penfield & Roberts (1959), it was clearly proposed in the context of biological foundations of language by Lenneberg (1967) for the first time.

The critical period for first language development corresponds to a time span during which an individual can automatically acquire his/her mother tongue by mere exposure to some samples of it without any conscious and labored effort, and, as Lenneberg depicts,

> the individual appears to be most sensitive to stimuli at this time and to preserve some innate flexibility for the organization of brain functions to carry out the complex integration of subprocesses necessary for the smooth elaboration of speech and language. (Lenneberg 1967: 158)

Here, "some innate flexibility for the organization of brain functions" in question refers to plasticity of the brain with respect to potentiality for physiological readjustment of either hemisphere to assume the language function in the case of brain lesions, which is operative before the end of the critical period (see Lenneberg 1967: 150–152).

With figure 2 above in mind, Lenneberg (1967) put forth the critical period hypothesis for first language acquisition, in which the onset of the critical period is the point of language-readiness as a state of disequilibrium and its end marks the point of relative stability (also see Lenneberg 1969). Taking stock of a variety of cases of child language acquisition (both normal and handicapped), he hypothesizes that the critical period at stake corresponds to the time span from around 2 years to around 12 or 13 years, as illustrated in figure 6 below.[3]

The rationale behind the presumed onset and end of the critical period at around 2 and 12–13 years of age, respectively, comes from the following facts,

[3] The critical period of the auditory system is known to be much earlier than two years (see also fn. 20 in the text). Shultz et al. (2014) examined neural responses to speech sounds compared with non-speech sounds in 1 to 4 months old infants using fMRI and observed neural specialization of left temporal cortex for speech in the first months of life. While the brain region in question continued to be responsive to speech sounds, it became less responsive to non-speech sounds. Although the exact formulation of the notion of critical period for first language development in other domains has been controversial in the literature ever since the proposal in Lenneberg (1967), I will not get into the debate here (see, e.g., Piekarski et al. 2017. Also see Hoshi & Miyazato 2016 and references cited therein for discussion and reflection on this debate).

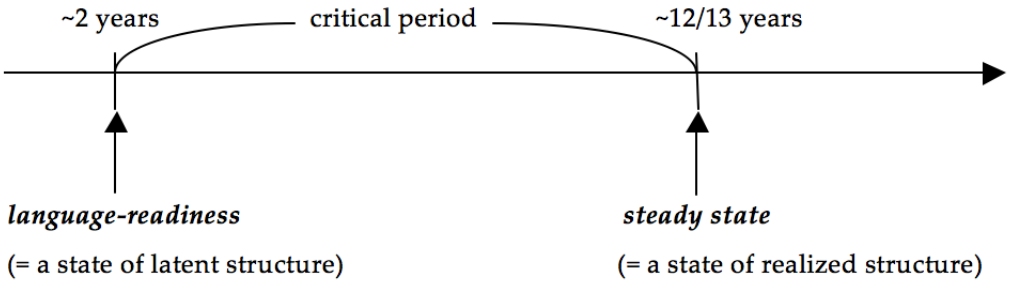

Figure 6: Schematic depiction of Lenneberg's original conception of the critical period for first language development.

among others:[4] First of all, even if brain lesions occur in either side of the brain in a child during the first two years of life, he/she could begin to speak at the usual time; otherwise, speech disturbances would result, though they are overcome in less than two years' time before the critical period ends around 12–13 years of age (Lenneberg 1967: 151). Furthermore, while the acquisition of language proceeds through the same fixed developmental stages in the retarded as well, such as children with Down's syndrome, their language development tends to come to a standstill after age 12–13 (Lenneberg 1967: 154–157).

During this period children could acquire their mother tongue on a biologically determined course of language development, given appropriate linguistic input from their environment. Lenneberg also remarks that

> it is interesting that the critical period coincides with the time at which the human brain attains its final state of maturity in terms of structure, function, and biochemistry (electroencephalographic patterns slightly lag behind, but become stabilized by about 16 years). Apparently the maturation of the brain marks the end of regulation and locks certain functions into place. (Lenneberg 1969: 639)

As amply documented and clearly demonstrated in Lenneberg (1967: Chs. 4 & 5), in ordinary child aphasia, brain lesions in the left hemisphere in a child do not prevent other parts (of the same hemisphere or) of the right hemisphere in the child from ontogenetically developing the language function by taking over the role and establishing a properly functioning language-related neural network, to the extent that he/she is still within the critical period thanks to plasticity of the brain (see also Hirsch et al. 2006). See also the discussion in section 3.

Although the fact that Lenneberg (1967) formulated the critical period hypothesis for first language acquisition is well-known among linguists, unfortunately, his original version of the critical period hypothesis does not seem to be understood correctly and appreciated fairly in the literature. First of all, Lenneberg's critical period is only concerned with first language acquisition and he does not say anything clear about second/foreign language acquisition. Furthermore, although

[4] Genie's case in Curtiss (1977) has also been known as a strong piece of evidence in support of Lenneberg's (1967) critical period hypothesis in the literature. See the short discussion of this in section 4.

the term has been commonly used in the broad notion of "first language acquisition" in the literature, it should be noted that Lenneberg's original version of the critical period hypothesis only applies to the ontogenetic development of language capacity and crucially it claims that linguistic output/externalization, for example, by articulation, is *not* subject to such a critical period (see Lenneberg 1967: 158). Hence, the development of the system for articulatory motor skills is free from the critical period at stake under Lenneberg's (1967) critical period hypothesis for first language development.

In sum, the essence of Lenneberg's (1967) original version of the critical period hypothesis for first language development can be stated as follows: Only the capacity for language is subject to the time constraint of the critical period (i.e. from approximately 2 years of age to 12–13 years of age) and thus externalization of the capacity for language does not respect the time constraint of the critical period.

2.2.2. Resonance Theory

Then, how do human children take in the primary linguistic data for developing their first language in the schematic in figure 3? Lenneberg rightly notes the need for "social settings as a trigger that sets off a reaction" (Lenneberg 1967: 378) for first language development. He appeals to the concept of "resonance" as a metaphor in his conceptualization of first language development model as follows:

> Perhaps a better metaphor still is the concept of resonance. In a given state of maturation, exposure to adult language behavior has an excitatory effect upon the actualization process much the way a certain object begins to vibrate in the presence of the sound. In the case of language onset, the energy required for the resonance is, in a sense, supplied by the individual him[/her]self. (Lenneberg 1967: 378)

Regarding the resonance analogy, he also adds that it vividly illustrates "how slight variations in the frequencies that impinge on the resonator may affect the quality or nature of the resonance" (Lenneberg 1967: 378). For that matter, he also discusses social aspects of behavior including language as follows:

> Certain social phenomena among animals come about by spontaneous adaptation of the behavior of the growing individual to the behavior of other individuals around him[/her]. … In all types of developing social behavior, the growing individual begins to engage in behavior as if by resonance; he[/she] is maturationally ready but will not begin to perform unless properly stimulated. If exposed to the stimuli, he[/she] becomes socially "excited" as a resonator may become excited when exposed to a given range of sound frequencies […].
>
> (Lenneberg 1967: 373–374)

Thus, under this view, the growing child is regarded as a biologically determined specific "linguistic resonator" that spontaneously resonates to the speech spoken by (an) adult(s) around him/her in social settings, as depicted in figure 7 below.

If you consider the spontaneous resonation in a child for first language acquisition in terms of the relation among the sensorimotor (SM) system, the latent

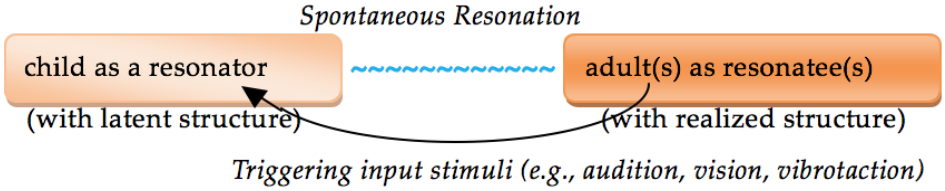

Figure 7: Schematic illustration of the child's spontaneous resonation for first language acquisition according to Lenneberg (1967).

structure, and the realized structure, you can regard audition, vision, or vibrotaction via the SM system as triggering spontaneous resonation in the child armed with the latent structure, and the latent structure of the resonating child will be led to his/her realized structure by gradually and spontaneously "synchronizing with" the comparable realized structure in the adult(s) interacting with him/her.

It seems that Lenneberg's metaphorical notion of "spontaneous resonance" in the child acquiring his/her first language goes well with his strong thesis in the beginning of his chapter 1 that says:

> A major objective of this monograph is [...] to show that reason, discovery, and intelligence are concepts that are as irrelevant for an explanation of the existence of language as for the existence of bird songs or the dance of bees.[5] (Lenneberg 1967: 1)

If the spontaneous linguistic resonation is one of our biologically determined instincts, the notion nicely captures our factual observation that a child is able to acquire his/her first language without any conscious effort in achieving that goal.

With regard to the relation between the critical period and the resonation for first language acquisition, Lenneberg states that

> [o]nce the critical period during which resonance may occur is outgrown, one language is firmly established, and exposure to new and different natural languages is no longer resonated to.
>
> (Lenneberg 1967: 378)

Thus, once the critical period has passed, the biologically determined autochthonous sensitivity of spontaneous resonance to the primary linguistic data will fade out.

Recall that the raw material in the primary linguistic data is processed and broken down into the linguistically-relevant building blocks for the child's own language, which must be resynthesized out of them while he/she is engaging in spontaneous resonation for first language acquisition, as illustrated in figure 4 above. Importantly, Lenneberg (1967: 376) clearly submits that this kind of developmental language synthesizing process on the basis of spontaneous linguistic resonation by the child is only operative during the critical period of biologically-determined, limited duration. In this connection, Lenneberg also notes:

[5] See Crain et al. (2016), Sugisaki (2016), and Yang et al. (2017) *inter alia* for an in-depth theoretical and empirical discussion of first language acquisition from a biolinguistic perspective.

> Resonance is linked to a postnatal state of relative immaturity and a concomitant lengthening of infancy and childhood, so that environmental influences (the molding after patterns available in the environment) can actually enter into the formative processes. (Lenneberg 1967: 392)

2.3. Speech Production and Brain Rhythmicity

Based on the view that the neural messages that are relevant to speech and language are "temporally coded signals" (Lenneberg 1967: 222), Lenneberg already entertained the following conjecture on brain oscillations:

> [s]pontaneous rhythmic activities of ganglia and even individual cells and fibers have been studied for many years now. It is entirely conceivable that these oscillations may serve a function that is analogous to the FM carrier frequency. (Lenneberg 1967: 216)

Furthermore, concerning brain rhythmicity, Lenneberg also notes that:

> It has long been known that the universally observed rhythmicity of the vertebrate brain [...] or central nervous tissue, in general [...] is the underlying motor for a vast variety of rhythmic movements found among vertebrates. If our hypothesis is correct, the motor mechanics of speech (and probably even syntax) is no exception to this generalization, and in this respect, then, speech is no different from many other types of animal behavior. In man, however, the rhythmic motor subserves a highly specialized activity, namely speech. (Lenneberg 1967: 119)

On the recognition that "rhythmic activity is a fundamental property of the vertebrate brain" (Lenneberg 1967: 116), Lenneberg also makes the following remark about a spontaneous dominant steady brain rhythm of approximately 7 Hz or faster over the temporo-parietal regions that is closely related to speech:[6]

> It is also interesting to note that children do not begin to develop speech until their brains have attained a certain degree of electro-physiological maturity, defined in terms of an increase with age in the frequency of the dominant rhythm. Only when this rhythm is about 7 cps [= Hz] or faster (at about age two years) are they ready for speech development.
>
> (Lenneberg 1967: 117)

In addition, Lenneberg proposes that there exists a physiological rhythm in speech which functions as an organizing principle/timing device for articulation with a duration of one-sixth of a second as the basic time unit in the programming of motor-speech patterns, virtually corresponding to the time unit for a syllable,

[6] The need for the emergence of this particular brain rhythmicity over the temporo-parietal regions for speech development in children seems to make sense, given that the dorsal-pathway for speech production crucially involves these brain regions, according to Hickok & Poeppel's (2007) dual-stream model of speech processing (see also Hickok 2009, 2012, Hickok et al. 2011). Also see Deonna & Roulet-Perez (2016) and Hoshi & Miyazato (2016) for some discussion on the dual-stream model of speech processing in connection with LKS, which will be taken up in section 3 in the text.

and that the timing mechanism renders the temporal ordering of speech events physically possible, using the metaphor that "[t]he rhythm is the grid, so to speak, into whose slots events may be intercalated" (Lenneberg 1967: 119).

The rationale behind Lenneberg's (1967) "basic timing mechanism" hypothesis is that neuromuscular automatisms for speech require an underlying physiological rhythm of periodic changes of "states" at a rate of approximately six cycles per second, i.e., 6+/–1 Hz. Thus, it is assumed that about one-sixth of a second is a fundamental temporal unit for articulatory programming in speech.

Interestingly, Lenneberg (1967) links this basic temporal frequency of 6+/–1 Hz for speech to the dominant steady brain rhythm of about 7 Hz over the temporoparietal regions mentioned above as an instance of neurological correlates in electroencephalogram (EEG).[7] Although the frequency band in the frontal lobe, especially (pre-)motor areas and Broca's area, necessary for speech development in a child is not reported in Lenneberg (1967), I will assume that a comparable or higher frequency range is required in these regions as well for speech development in a child.[8] This can be validated by Giraud et al. (2007), who incorporate MacNeilage & Davis's (2001) Frame/Content (F/C) theory for speech production and Poeppel's (2003) Asymmetric Sampling in Time (AST) theory for speech perception. Following MacNeilage & Davis (2001) and Poeppel (2003) in seeking to link speech processing to neural oscillations in the brain, Giraud et al. make the following observation on the basis of simultaneous EEG and fMRI recordings:

> spontaneous EEG power variations within the gamma range (phonemic rate) correlate best with left auditory cortical synaptic activity, while fluctuations within the theta range correlate best with that in the right. Power fluctuations in both ranges correlate with activity in the mouth premotor region, indicating coupling between temporal properties of speech perception and production. (Giraud et al. 2007: 1127)

Moreover, particularly interesting observations in Giraud et al. (2007) are the correlations between EEG and hemodynamic (fMRI) fluctuations in (pre)motor cortices. They found that, while 3–6 Hz EEG band is related to the motor region that controls movement of the mouth, 28–40 Hz EEG band is linked with the motor region that is responsible for controlling movement of the tongue. In light of this result, Lenneberg's (1967) observation on the relation between brain rhythmicity and speech development in a child seems to make sense. Given that speech production involves fine articulatory movements of the mouth and the tongue and that the 3–6 Hz EEG band and the 28–40 Hz EEG band are required to control movement of the mouth and the tongue in adults, respectively, it does not come as a surprise that brain oscillations with approximately 7 Hz or faster frequencies are needed to initiate speech development in a child, involving fine articulatory movements of both

[7] It is well-known that the most common brain rhythms have been classified by frequency, e.g., delta (δ) (0.5 – 4 Hz), theta (ϑ) (4 – 10 Hz), alpha (α) (8 – 12 Hz), beta (β) (10 – 30 Hz), and gamma (γ) (30 – 100 Hz), and those brain oscillations are reflections of synchronized neuronal activities in various cortical and subcortical structures (see Buzsáki 2006, Buzsáki & Watson 2012, Murphy 2015, 2016, Fernández 2015 *inter alia*). Delta /theta waves and beta/gamma waves are classified as slow waves and fast waves, respectively (see Bear et al. 2007).

[8] See Benasich et al. (2008) and Gou et al. (2011) for recent findings that the emergence of resting high-frequency gamma power neural synchrony in the frontal lobe across the first three years is crucial for early linguistic and cognitive development in a child.

the mouth and the tongue. I submit that these specific frequency bands should be taken into account in applying the non-invasive neuromodulation technology of transcortical direct current stimulation (tDCS; see Nitsche & Paulus 2000 *inter alia*) as a possible medical intervention for speech recovery in LKS, which will be addressed in the next section (see Hoshi & Miyazato 2016 for the proposal of using tDCS for speech and comprehension recovery in LKS).

In other words, Lenneberg (1967) makes the case that proper and stable development of particular brain rhythmicity as reflected in EEG patterns is required for emergence of speech in language development. In the final analysis, it is to be highly evaluated that, at the time when EEG-based investigations into language and cognition were not as sufficiently sophisticated as in our time, Lenneberg had already paid attention to the importance of scrutinizing patterns of brain oscillations in searching the biological nature of human language. In this sense, Lenneberg (1967) precedes the above-mentioned works in identifying the fundamental direction for theorizing on the relation between neuronal oscillatory patterns and linguistic performance of speech perception and speech production in natural language.[9]

3. Lenneberg's Child Aphasiology and its Application to LKS

3.1. Lenneberg's View on Aphasia in General

Lenneberg (1967) argues that aphasia in general has the following properties: (i) aphasic patients' capacity for language is not lost, but it is merely interfered with; (ii) aphasic symptoms are caused by disorders of timing/temporal integration mechanisms in charge of yielding proper orders of linguistic units.

Concerning (i), Lenneberg makes the following point on the basis of a survey of the clinical pictures of a variety of language and speech disorders, including receptive disorders, expressive disorders (subfluency, superfluency, semantic disturbances, difficulty in word finding, paraphasic disturbances), disorders of manner of production (errors of order, dysarthria, discoordinations):

> The most striking common denominator in aphasia is the ubiquitous evidence that the patient has not literally "lost" language; that is, he[/she] is not returned to a state of no language such as an animal or even a person who forgot everything he[/she] once knew in a foreign language. [...] In the literal sense of the word, the patient's language skills are merely interfered with; there are disturbances of cerebral function. Neither discrete words nor discrete grammatical rules are neatly eliminated from the store of skills. [...] [C]areful observation of the recovery process during the critical post-morbid period, makes it very plain that the patient does not start with specific lexical or grammatical lacunae, but that some basic physiological processes relating to activating, monitoring, or processing of speech are deranged. If there is clinical improve-

[9] It might be worth investigating whether or not the postulated ability/function for spontaneous resonation in a child acquiring his/her mother tongue in figure 7 would be significantly correlated with the emergence and development of a particular brain rhythmicity in him/her in the maturational course.

> ment, it is not due to the acquisition of new vocabulary or grammatical rules, but to release from inhibitory factors, to faster acting memory, to better controlled organization of elements, etc. Thus the distinction between loss of language and interference with skills leads to different approaches in rehabilitation and management of patients with aphasia.
>
> (Lenneberg 1967: 207)

Thus, Lenneberg regards aphasics as just having lost the ability of utilizing the language inside the brain for comprehension and production due to interference by brain lesions rather than having lost the language capacity itself. Accordingly, the realized structure (= capacity for language) established should remain there within the brain in aphasics.

With respect to (ii), in light of the tenet that time is the most significant dimension in language physiology, Lenneberg argues that aphasic symptoms in general can be characterized as disorders of timing/temporal integration mechanisms,[10] as can be appreciated by the following passage in the conclusion of chapter 5 of his book:

> Language is never totally and specifically lost except in combination with complete disruption of cognition. All disorders are aspects of interference with physiological processes prerequisite for the normal function of speech and language. Aphasic symptoms give no evidence of a fragmentation of behavior, that is, of dissolution of associatively linked 'simpler percepts.' Most of the symptomatology may be seen as disorder of temporal integration, of 'lack of availability at the right time.'
>
> (Lenneberg 1967: 222)

In addition, from Lenneberg's perspective of the architecture of human language as depicted in figure 5 above, (ii) can be interpreted as claiming that aphasia is generally caused by dysfunction of the sensorimotor (SM) system that is responsible for programing and implementing temporal sequencing of linguistic units for externalization, while the hierarchically structured expressions (associated with such linguistic units) generated by core syntax in the realized structure remain virtually intact.

3.2. Lenneberg's View on Child Aphasia

On a par with acquired adult aphasia, acquired ordinary child aphasia involves some sort of organical brain lesions due to traumas, tumors, or cerebrovascular damages. Interestingly, however, unlike aphasia incurred in adulthood, ordinary child aphasia will generally be overcome if it strikes the child early enough in life

[10] Lenneberg is also perceptive in suggesting that

> [c]ortical lesions primarily interfere with temporal integration of a higher order (words or grammatical category), whereas deeper lesions disrupt the necessary convergence of various afferent signals and the intimate coordination and integration for efferent impulses, thus producing disorders of production.
>
> (Lenneberg 1967: 222)

(Lenneberg 1967, 1969).[11] In fact, Lenneberg summarizes the relation between plasticity and lateralization of the brain in aphasia involving some brain lesions as follows:

> Aphasia is the result of direct, structural, and local interference with neurophysiological processes of language. In childhood such interference cannot be permanent because the two sides are not yet sufficiently specialized for function, even though the left hemisphere may already show signs of speech dominance. Damage to it will interfere with language; but the right hemisphere is still involved to some extent with language, and so there is a potential for language function that may be strengthened again. In the absence of pathology, a polarization of function between right and left takes place during childhood, displacing language entirely to the left and certain other functions predominantly to the right [...]. If, however, a lesion is placed in either hemisphere, this polarization cannot take place, and language function together with other functions persist in the unharmed hemisphere.
>
> (Lenneberg 1967: 153)

Thus, according to Lenneberg, the following generalization emerges for acquired ordinary child aphasia:

(1) ***Lenneberg's Generalization on Acquired Ordinary Child Aphasia***
The earlier the onset of the disorder is, the better the prognosis for recovery will be (see Lenneberg 1967: 153, 178. See also Lenneberg 1969, 1975 for further discussion).[12]

The pattern for ordinary child aphasia in (1) seems to be quite expected in the light of plasticity of the child brain in connection with Lenneberg's (1967) critical period hypothesis. If the onset of the language disorder is earlier, the relevant language function would be relocated or compensated for by the use of other parts of the (ipsilateral or contralateral) language-related brain regions to the extent that the child is still within the critical period.[13] This means that, in the case of ordinary

[11] See also Alajouanine & Lhermitte (1965) for a similar conclusion that the prognosis for acquired aphasia with lesions in childhood is definitely better than that in the adult.

[12] In fact, Lenneberg infers that "*language learning* can take place, at least in the right hemisphere, only between the age of two to about thirteen," (Lenneberg 1967: 153) namely, during the critical period for first language acquisition that Lenneberg postulated.

[13] There is a statement in Lenneberg (1967) that complicates the situation:

> If aphasia strikes the very young during or immediately after the age at which language is acquired (between 20 to 36 months of age), the recovery is yet different. Cerebral trauma to the two or three year old will render the patient totally unresponsive, sometimes for weeks at a time; when he[/she] becomes cognizant of his[/her] environment again, it becomes clear that whatever beginning he[/she] had made in language before the disease is totally lost, but soon he[/she] will start again on the road toward language acquisition, traversing all stages of infant vocalization, perhaps at a slightly faster pace, beginning with babbling, single words, primitive two-word phrases, etc., until perfect speech is achieved. In the very young, then, the primary process in recovery is acquisition, whereas the process of symptom-reduction is not in evidence.
>
> (Lenneberg 1967: 146, 150)

child aphasia, the child could overcome the aphasic state by appealing to plasticity of the neural network under development in the brain before the full maturity of the neural network is attained in accordance with the biologically determined critical period for first language acquisition.

Provided that Lenneberg's original version of the critical period hypothesis and his general view on aphasia were on the right track, it would yield a significant implication to the study of child aphasia. Even if a child is suffering from childhood aphasia, it is predicted that, in principle, there should be a case where *externalization* of "inner language," i.e., language capacity as either a partially actualized form or a fully actualized form of the latent structure, could happen even *after* the critical period ends, once the deficit in the neural system for articulatory motor skills for externalization is removed or disappears, on the condition that the core biologically determined system of the latent structure remains virtually intact in the child with aphasia and that acquisition of the mental lexicon along with language-particular morpho-phonology, syntax, and semantics should become possible by the end of the critical period. As Hoshi & Miyazato (2016) point out, this prediction is borne out by a certain childhood aphasia of epileptic origin, which I will be addressing in the next section.[14]

3.3. Landau-Kleffner Syndrome and Lenneberg's Critical Period Hypothesis

3.3.1. LKS: Epileptogenic Child Language Disorder

Landau-Kleffner syndrome (LKS) is a clinically rare language disorder of acquired childhood aphasia involving epilepsy (with or without clinical seizures), which was first reported by Landau & Kleffner (1957).[15] LKS emerges with epileptiform electroencephalographic (EEG) abnormalities typically involving continuous spike waves during slow sleep (CSWS) over the temporal regions, and unlike ordinary childhood aphasia, it does not implicate any particular brain lesions (Gordon 1990, Deonna 1991, 2000). While computed tomography (CT) and magnetic resonance

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If this is the case in general, in acquired ordinary child aphasia with early onset, there is a possibility that partially or fully acquired realized structure will be lost by brain lesions. Nevertheless, it is not clear whether the partially or fully attained realized structure in such very young aphasics is truly lost altogether, or whether it is in fact still there in the brain but its externalization by speech is either merely interfered with and blocked or does not start yet (recall that a child's dominant brain rhythm over the temporo-parietal regions will reach 7 Hz or faster around 2 years of age), given Lenneberg's view on aphasia in general (see Lenneberg 1967: Ch. 5).

[14] Considering and comparing significant differences between LKS and other childhood aphasias/language disorders would go well beyond the scope of the current paper. See Bishop & Leonard (2000) *inter alia* for invaluable discussion and relevant references.

[15] See, for example, Deonna & Roulet-Perez (2016) for the most recent review and the most comprehensive discussion on LKS in the framework of epilepsy-aphasia spectrum. See also Steinlein (2009) for an in-depth description of LKS in the framework of epilepsy-aphasia syndromes. A *Biolinguistics* reviewer rightly remarked that LKS is not as clearly defined in the literature as one would probably expect, pointing out that Rapin et al. (1977) among others do not regard LKS as childhood aphasia but as verbal auditory agnosia. However, given that LKS patients, who suffer from verbal auditory agnosia, typically exhibit expressive language disorder as well at least during the acute period, I will include LKS under the general categorial term "childhood aphasia" in the text by broadly construing the notion of "aphasia" as referring to the state of either apparently sensory or expressive language disorders or both.

imaging (MRI) findings on patients with LKS are normal, single photon emission computed tomography (SPECT) and positron emission tomography (PET) studies on the patients show temporal lobe abnormalities in brain perfusion and glucose metabolism, i.e., decreased perfusion and hypometabolism, respectively (DaSilva et al. 1997, Pearl et al. 2001, and references therein; see Deonna & Roulet-Perez 2016 for other patterns.)

The child with LKS first undergoes a period of normal development of language, but usually after the onset of the disorder, the "language attained thus far" starts regressing. In LKS, it is quite common that both language comprehension and language production acutely or insidiously become extremely difficult or impossible, often leading to apparent deafness and mutism in the child suffering from it (see Gordon 1990, 1997, Tharpe & Olson 1994, Kaga 1999, 2011, Pearl et al. 2001 *inter alia* for more details). Moreover, in addition to the language disorder, the EEG abnormalities in LKS also cause behavioral and psychiatric disturbances such as hyperactivity, aggressive behavior, impulsivity, and attentional problems, which resemble autism spectrum disorders (ASD) (Stefanatos 2011, Mikati et al. 2010).

To be more specific about the language disorder in LKS, the epileptic discharges over the temporal regions, as reflected in the EEG abnormalities, will result in the child with LKS having extreme difficulty or impossibility of hearing linguistic sounds due to the dysfunction of the system of processing linguistic sounds in the non-primary auditory cortices during the active phase of LKS (Hirsch et al. 2006). Given that LKS patients could acquire a sign language even if they cannot restore their original spoken expressive language (see Deonna et al. 2009, Deonna & Roulet-Perez 2016 and references therein), it is clear that the childhood aphasia in LKS, which is caused by abnormal epileptic discharges over the temporal regions, only affects the SM system responsible for speech perception and speech production while the system of the language capacity remaining virtually intact.

This crucially means that virtually no linguistic input would become available during the active period and that no further primary linguistic data (PLD) would become accessible if the child with LKS were to be still in the process of first language development. I believe that the very notion of spontaneous resonance is also of service in considering LKS, because it is plausible to assume that spontaneous resonance illustrated in figure 7 is fatally blocked or disturbed in such a case of childhood aphasia, due to the epileptic discharges over the temporal regions affecting both hemispheres, which hampers brain plasticity, and the related difficulty of taking in verbal auditory input by the deficit of the system of spectrotemporal analysis in the superior temporal gyri (see Hoshi & Miyazato 2016 for details). This, in turn, suggests that the ability of processing linguistic sounds would, in principle, come back once the EEG abnormalities are either removed or disappear in LKS.

Moreover, the clinical seizures are generally infrequent and LKS-related epileptic clinical seizures themselves can be easily controlled by a single anti-epileptic medication: benzodiazepines such as clobazam (Pearl et al. 2001), valproate, and ethosuximide (Mikati et al. 2010). Although the epileptiform EEG abnormalities themselves cannot be easily suppressed by such anti-epileptic medication, the paroxysmal EEG abnormalities will improve gradually and usually disappear spontaneously by around 14 years of age in LKS (Massa et al. 2000, Robinson et al. 2001, Ramanathan et al. 2012, Deonna & Roulet-Perez 2010, 2016). Thus, it is quite nat-

ural to assume that verbal auditory input would become possible gradually well before 14 years of age and the quality of linguistic input would concurrently improve during the process of gradual amelioration of the EEG status in LKS.

Given that a child will acquire the core linguistic competence by around three years of age (Lenneberg 1967, Pinker 1994, O'Grady 2005) in a normal course of first language acquisition and that approximately 80 % of LKS has the onset ranging from three years old to eight years old (Kaga 2000), but that the earliest onset of LKS occurs around 18 months of age (Uldall et al. 2000), Hoshi & Miyazato (2016) divide LKS into two broad sub-types of what they call early LKS and ordinary LKS, defining them as follows:

(2) a. *Early LKS* has the onset before 3 years of age, when the affected child has not yet established the core linguistic competence sufficiently.

b. *Ordinary LKS* refers to all other cases of LKS.

Interestingly enough, there are some children with early LKS who would be able to experience something like a linguistic big bang. Uldall et al. (2000) observe that their patient with early LKS (with onset at 18 months) speeded up language acquisition in his "catch-up periods" in such a way that he acquired vocabulary that would have normally taken one whole year to acquire was developed in just three months after the age of five years. They remark that

> the normal spurt of vocabulary usually seen at the age of 17–19 months seemed to have been blocked until it was 'released' by the prednisone course at the age of 5 years. (Uldall et al. 2000: 85)

As this case indicates, as long as the "inner language," or the realized structure in the sense of Lenneberg (1967) or I-language in the sense of Chomsky (1986), is established before the critical period ends, externalization of the inner-language would be possible even later in life, presumably producing the linguistic big bang.[16] Notice that, as long as externalization of the inner language is not subject to the critical period, as claimed in Lenneberg (1967), such a linguistic big bang, in principle, could occur even after the critical period ends in early LKS.

Thus, the prognosis patterns of LKS patients is that approximately 50 % of the patients recover fully and about 50 % of the remaining patients recover partially after a certain period of time (Mikati et al. 2010), which is remarkable in contrast with the case of autistic regression in autism spectrum disorders (ASD) (see Hoshi & Miyazato 2016 and references therein).[17] Furthermore, curiously enough, unlike ordinary child aphasia, for which Lenneberg's generalization in (1) holds, LKS tends to display just the opposite pattern. Namely, the earlier the onset of the disorder is, the worse the prognosis will be (see, e.g., Bishop 1985).[18] That is, a younger

[16] Note that Lenneberg's remark cited in the excerpt on page 96 also applies to the case of the linguistic big bang in early LKS in that the patient will not lose the acquired linguistic knowledge and will not re-start language acquisition from scratch.

[17] While the patient with ordinary LKS would be highly likely to recover from the state of aphasia in a relatively short period of time, the patient with early LKS would either recover from such a state after a relatively long period of time or not recover from it.

[18] This description does not express absolute correlations but just tendencies. In reality, much more complicated and varied patterns are clinically observed in specific cases in the literature

age of the onset of the language disorder is generally related to a dimmer prognosis for recovery from the state of aphasia in LKS.

Accordingly, the general tendency of childhood aphasia in LKS appears to be just the opposite of Lenneberg's generalization on the surface. However, notice that, due to the very nature of LKS as a form of non-lesional, epileptogenic aphasia in a child who is still under maturational development of linguistic and cognitive functions, unlike ordinary child aphasia, plasticity of the brain would not readily come into play in LKS, because epileptic discharges typically in the form of CSWS that originally emerge unilaterally would be bilateralized by propagating to the contra-lateral hemisphere, hampering the use of brain plasticity and impeding normal functioning of the language-related neural network (Hirsch et al. 2006, O'Hare 2008). Hence, Lenneberg's generalization in (1) cannot be applied to LKS on a fundamental ground in the first place.

This conjecture seems to fall into place, once Lenneberg's (1967) perspective and biolinguistic framework are taken into account. Recall from section 2.3. that Lenneberg fundamentally assumes that language is realized in the brain as temporally coded patterned activities with various oscillations (see Lenneberg 1967: Chs. 4 & 5 for details). Thus, if the language-related oscillations are disturbed, as reflected in EEG abnormalities, it is naturally expected that "language behavior" is also disturbed accordingly.

Nevertheless, it is important to recall that, as discussed above, such paroxysmal EEG abnormalities will gradually ameliorate and usually disappear spontaneously by around 14 years of age (Massa et al. 2000, Robinson et al. 2001, Ramanathan et al. 2012, Deonna & Roulet-Perez 2010, 2016). Thus, in accordance with the improvement of the EEG abnormalities, the quality of linguistic input would become better little by little as the child with LKS passes through the critical period. This should in turn facilitate at least the development of the realized structure in the sense of Lenneberg (1967) even in the case of early LKS.

3.3.2. Some Implications to LKS

First of all, if LKS falls under the category of child aphasia, Lenneberg's child aphasiology suggests that the capacity of language itself is not lost but should exist within the brain of the child with LKS, although it is interfered with somehow (but see fn. 13). As already discussed in sections 3.2. and 3.3.1., in the case of ordinary child aphasia, the interference is due to brain lesions; whereas, in the case of LKS, it is caused by epileptic discharges over the temporal regions that involve bilateralization of an originally unilateral focus. Given that the critical period spans from around 2 years of age to around 12/13 years of age, it is imperative that linguistic input become available again to the child with LKS before the critical period ends. This would guarantee that the latent structure be actualized into the full-fledged realized structure within the critical period in the sense of Lenneberg (1967), particularly in the case of early LKS. Recall that the epileptogenic EEG abnormalities

(see Deonna et al. 1977 for the varied prognosis of LKS depending on factors other than the onset of the disorder. See also Deonna & Roulet-Perez 2016 and references therein for detailed discussion). Moreover, the degree of recovery from LKS depends on not only the time of onset but the response to anti-epileptic medication and the severity of communication problems (see Pullens et al. 2015). Thus, this prognostic pattern is merely a general tendency in LKS.

in LKS will ameliorate gradually and disappear spontaneously by around 14 years of age. In other words, there exists a time lag between the end of the critical period and that of the EEG abnormalities in LKS. Thus, from a medical point of view, it is imperative to control the EEG abnormalities by the end of the critical period in order to obtain a better prognosis in LKS (see Hoshi & Miyazato 2016 for details).[19]

However, recall also that externalization of the language capacity, for example, by speech, is exempt from the time constraint of Lenneberg's critical period. As such, to the extent that the language capacity can develop within the critical period in the child with LKS, he/she will have a chance to (re-)start speaking the mother tongue even after the critical period ends.

To sum up, the relevant conjectures on LKS can be stated as follows:

(3) ***LKS Conjectures***

a. The language capacity is not lost but exits as either a partially realized structure (i.e. *early LKS*) or a fully realized structure (i.e. *ordinary LKS*).

b. Once linguistic input becomes available again, first language development could re-start in *early LKS* within the critical period and the use of language could be restored in *ordinary LKS*.

c. Externalization of the language capacity by speech, for instance, could take place without any influence of the time constraint of the critical period.

It seems to be quite plausible to guess that the three LKS conjectures in (3), which are based on Lenneberg's (1967) original version of the critical period hypothesis and his view on child aphasiology, are responsible for the fact that approximately 50 % of the LKS population recover fully and about 50 % of the remaining patients recover partially in the use of their mother tongue (Mikati et al. 2010).

LKS can be interpreted as a situation where the spontaneous resonating process for first language development has been hampered due to the disturbance of normal brain rhythmicity by epileptiform EEG abnormalities such as CSWS. As first language development will proceed via spontaneous resonance during the critical period, according to Lenneberg (1967), it is of vital importance to secure as much spontaneous resonance as possible during the critical period especially for the child with early LKS. If the spontaneous resonance can be secured, re-development of the language capacity on the basis of what the LKS patient achieved on the latent structure should be theoretically possible even in the child with early LKS. Recall that the dominant brain rhythm over the temporo-parietal regions has to reach about 7 Hz or faster in order for a child to be ready for speech development.[20] Thus, it is significant to get rid of the epileptiform EEG abnormalities typically with CSWS in order to secure steady spontaneous brain rhythms of

[19] See, for example, Faria et al. (2012) for an attempt to modulate epileptic activity focally in patients, including a patient with LKS by employing the non-invasive neuromodulation technology of tDCS successfully. See also Arle & Shils (2017) for an introduction to the latest development of innovative clinical neuromodulation, including tDCS.

[20] Lenneberg (1967: 117) states that the dominant brain rhythm of about 7 Hz or faster frequencies over the temporo-parietal regions is attained at around age 2 years in a child as a prerequisite for speech development. Given that the onset of the critical period is around 2 years of age in Lenneberg's critical period hypothesis and early LKS could start around 18 months, it is clear that the child with early LKS cannot start engaging in full development of speech.

that frequency range or a much higher frequency range over those regions and possibly over (pre)motor regions including Broca's area in the frontal lobe in the child with LKS.

In the history of aphasiology, aphasic symptoms have often been analyzed as "disconnection" syndromes along the lines of studies by prominent scholars such as Carl Wernicke, Ludwig Lichtheim, Hugo Liepmann, Jules Dejerine and Norman Geschwind (see Catani & Mesulam 2008a, b *inter alia* for a review). Lenneberg takes issue with this position, claiming that

> the ablation experiments reviewed earlier should be a warning to 'aphasiologists' not to interpret specific clinical symptoms of aphasic patients as disruption of associations or structural, cortical disconnections. There is no experimental evidence that any associative bonds may be disrupted by discrete cortical lesions. (Lenneberg 1967: 217)

(Also see Lenneberg 1975 for arguments in favor of such an anti-disconnection view on aphasia.[21])

If Lenneberg's (1967) biolinguistic framework and his view on child aphasia are fundamentally on the right track and are extendable to LKS as well, as argued above, it will open up a new possibility for rehabilitation/medical intervention on LKS patients who are still suffering from partial or no restoration of speech comprehension and/or production. Hoshi & Miyazato (2016) propose to use tDCS in facilitating speech production and comprehension in LKS patients on the basis of Hickok & Poeppel's (2007) dual-stream model of speech processing.

Since early LKS and autistic regression in autism spectrum disorders (ASD) present with similar "autistic" behaviors such as hyperactivity, aggressive behavior, impulsivity, and attentional problems as well as cognitive regression including language disorders (Stefanatos 2011, Mikati et al. 2010), the two cases are particularly confusing and easily susceptible to misdiagnosis (Deonna & Roulet-Perez 2010, Stefanatos 2011. See also Hoshi & Miyazato 2016 for details). Therefore, differentiating early LKS from autistic regression in ASD should be carried out as early as possible within the critical period in terms of the following risk markers: (i) whether or not the child in question has the LKS-characteristic EEG abnormalities typically with CSWS; (ii) whether or not the epileptic seizures in the child, if any, can be readily controlled by anti-epileptic medication such as benzodiazepines

On the other hand, as Kuhl (1993) and Kuhl et al. (2005) *inter alia*. show, the "critical period" for phonetic perception/discrimination in infancy comes well before the end of the 1st year of life. Note that this is not in variance with Lenneberg's critical period hypothesis, because such a task of phonetic perception/discrimination is handled by the sensorimotor (SM) system, which lies outside the domain of application of Lenneberg's original version of the critical period hypothesis as discussed in the text. If this and Lenneberg's view on aphasia are on the right track, it suggests that even the child with early LKS has already acquired the ability for phonetic perception/discrimination before the onset of the aphasia and still keeps the potential for use (but see fn. 13).

[21] Based on Hickok & Poeppel's (2007) dual-stream model of speech processing as a theoretical foundation, Hoshi & Miyazato (2016) propose to analyze the apparently disconnection outlook of LKS (see Tsuru & Hoeppner 2007) as an epiphenomenon, in line with Lenneberg's (1967) anti-disconnection view on aphasia. More specifically, they attribute the aphasic state in LKS to the result of a "domino effect" over the dorsal pathway and the ventral pathway, arising from the dysfunction of the system of spectrotemporal analysis in the dorsal superior temporal gyrus (STG).

including clobazam. Once it is found that the child is affected by LKS, proper anti-epileptic medication should continue to be administered in order to consistently control epileptic seizures, if any, and hopefully to precipitate improvement of the EEG abnormalities (see Deonna & Roulet-Perez 2016) in order to make the quality of linguistic input better so that the language capacity would grow ontogenetically within the critical period in the LKS-affected child.[22]

4. Concluding Remarks

In this paper, I have revisited Lenneberg's (1967) biolinguistic framework and his view on (child) aphasiology in an attempt to re-evaluate his insights on biological aspects of human language and his pioneering contributions to the field of child aphasiology.[23]

As mentioned in fn. 4, one of the strong supports for Lenneberg's (1967) critical period hypothesis has come from a study on a language-isolated child called Genie, who was discovered isolated from language experience at age 13 and who was only able to develop a rudimentary syntax (Curtiss 1977). In this paper, I have aimed to show that children with LKS should be added as another type of language-isolated children that can provide further strong empirical evidence backing up Lenneberg's biolinguistic concepts. In the case of Genie, extreme deprivation of linguistic input during the critical period by her parents' intentional isolation and thus virtually no chance of spontaneous resonation to any adults surrounding her led to under-actualization of the realized structure out of the underlying latent structure in Lenneberg's (1967) framework.

On the other hand, LKS affords a rare opportunity to verify the validity of Lenneberg's critical period hypothesis from a different angle. In the case of LKS, while sudden deprivation of linguistic input occurs either before or after the onset of the critical period (i.e. around 2 years of age) and such a devastating condition will continue either for a relatively short period or for a relatively long period until presumably around 14 years old. Either way, it is highly likely that linguistic input will gradually come back, as discussed previously, before the end of the critical period (recall the dramatic recovery case of early LKS in Uldall et al. 2000). The fact that approximately 50 % of the LKS population recover fully and roughly 50 % of the remaining patients recover partially in the use of their first language (Mikati et al. 2010) clearly indicates that LKS can indeed serve as strong "living" evidence

[22] Even if the EEG abnormalities with epileptic discharges have gone by puberty/adolescence (Massa et al. 2000, Robinson et al. 2001, Deonna & Roulet-Perez 2010, 2016), there are still cases where restoration of speech comprehension and/or production would not likely occur fully or at all in LKS patients (see Hoshi & Miyazato 2016 for a detailed review and discussion on the various patterns of recovery in LKS patients), presumably because of an aftermath of the acquired dysfunction of those language-related regions that involved a long-lasting local impairment in the neural network in the regions, as reflected in residual glucose hypometabolism in the relevant areas that used to be hypermetabolic during the active phase of LKS (see Hirsch et al. 2006 for more discussion on the reason why some cases of LKS present with difficulty for language restoration).

[23] See, e.g., Benítez-Burraco (2016) for a concise but informative review of the current state of the field of clinical linguistics. See also Tsimpli et al. (to appear) for discussion on language disorders from the perspective of UG.

demonstrating the validity of both Lenneberg's (1967) original version of the critical period hypothesis and his fundamental view on (child) aphasia.

As already discussed as a consequence of medical import, it should become possible to differentiate early LKS and genuine ASD with autistic regression as a first step, on the basis of the fact that patients with LKS present with characteristic EEG abnormalities typically with CSWS and that epileptic seizures, if any, can be easily controlled by anti-epileptic medication (see Hoshi & Miyazato 2016 for more details). Therefore, it is expected that a much larger LKS-potential population within the ASD-diagnosed patients might turn out to fall under the category of early LKS and to become medically curable. In addition, although the EEG abnormalities in LKS will normally improve gradually and eventually disappear by adolescence, as mentioned before, amelioration of the EEG abnormalities could possibly be medically facilitated by the use of tDCS (see Faria et al. 2012) before the end of the critical period so that the child with LKS would have a better prognosis in both linguistic comprehension and linguistic production (see also Hoshi & Miyazato 2016 for a concrete suggestion on the use of tDCS for such a medical purpose). Taken altogether, I firmly believe that more attention should be paid to LKS in the context of (neuro)biolinguistics as well as medicine.

Two other major findings in Lenneberg's work are resonation and brain rhythmicity. As for resonation, it is expected that not only the language disorder but also the behavioral disturbances observed in children with LKS and a sub-population of children with AR in ASD might be related to malfunctioning of their proper resonation with relevant stimuli in their environment, including individuals interacting with them, due to brain rhythmicity disturbances (see Hoshi & Miyazato 2016 and references therein for some discussion on LKS and AR). Another speculation concerning brain rhythmicity is that application of tDCS may be instrumental to enhance the brain oscillations of the affected children up to the normal range of frequencies, viz., approximately 7 Hz or faster frequencies, which is reported by Lenneberg for speech development. Thus, I believe that these two groundbreaking concepts are likely to contribute to elucidating the mechanisms underlying child language and developmental disorders.

Before closing this section, I would like to refer to Lenneberg's (1967) research strategy in pursuing the biological aspects of human language. Recently, there is a burgeoning interest in looking into a variety of cognitive disorders and syndromes that affect language development such as autism spectrum disorders (ASD), specific language impairment (SLI), schizophrenia, Down's syndrome, and Williams syndrome among many others (see Bishop 1982, 2000, Bishop & Leonard 2000, Billard et al. 2009, Benítez-Burraco 2013, 2016, Benítez-Burraco & Boeckx 2014, 2015, Benítez-Burraco & Murphy 2016, Hinzen et al. 2015, Barceló-Coblijn et al. 2015, Hinzen & Rosselló 2015, Murphy & Benítez-Burraco 2016a, b, Tsimpli et al. to appear *inter alia*). Lenneberg (1967) also investigated not only (child/adult) aphasia but also Down's syndrome in an attempt to construct an integrative theory of the biological foundations of language. It should be recognized that it was Lenneberg (1967) that clearly demonstrated that such an enterprise requires the need for in-depth comparison between the nature of language behavior of those people with language-related cognitive disorders and syndromes and that of normally developed people.

In this connection, last but not least, let me draw the reader's attention to another legacy of *Biological Foundations of Language*, which is unlikely to be mentioned in academic settings: Lenneberg's dedicated sense of mission as a professional toward improving the fate of cognitively handicapped children and adults, as revealed by the following passage in the book:

> A particularly promising approach seems to be the systematic evaluation of patients with various deficits, especially [...] the mentally retarded. Modern advances in technology and methodology in behavior research are likely to lead to new knowledge about language function, and thus the patients whose misfortune serves as source material for new studies may, hopefully, eventually profit from the new advances in our understanding of language. (Lenneberg 1967: viii)

It should be kept in mind that *Biological Foundations of Language* is an immortal classic in the field of biolinguistics which not only set forth the fundamental tenet of the discipline but also encouraged a noble, humanistic attitude to engage ourselves to the biolinguistic enterprise, keeping in mind those people who are often forced to exist "on the periphery" in society, due to some chance misfortune. To the extent that what I suggested above holds some promise, it may well be possible to rescue a potentially large population of LKS-affected language-isolated children in the world with Lenneberg's "two legacies" in mind.

References

Alajouanine, Théophile & François Lhermitte. 1965. Acquired aphasia in children. *Brain 88, 653–662*.

Aristotle. 1938. *On Interpretation*, with Greek text translated by Harold P. Cook. Loeb Classical Library. Cambridge, MA: Harvard University Press.

Arle, Jeffrey & Jay Shils (eds.) 2017. *Innovative Neuromodulation*. London: Academic Press.

Bear, Mark F., Barry W. Connors & Michael A. Paradiso. 2007. *Neuroscience: Exploring the brain*. Lippincott Williams & Wilkins/Wolters Kluwer Health.

Benasich, April A., Zhenkun Gou, Naseem Choudhury & Kenneth D. Harris. 2008. Early cognitive and language skills are linked to resting frontal gamma power across the first 3 years. *Behavioural Brain Research* 195, 215–222.

Benítez-Burraco, Antonio. 2013. Genetics of language: Roots of specific language deficits. In Cedric Boeckx & Kleanthes K. Grohmann (eds.), *The Cambridge Handbook of Biolinguistics*, 375–412. Cambridge: Cambridge University Press.

Benítez-Burraco, Antonio. 2016. A biolinguistic approach to language disorders: towards a paradigm shift in clinical linguistics. In Koji Fujita & Cedric Boeckx (eds.), *Advances in Biolinguistics: The Human Language Faculty and Its Biological Basis*, 256–271. London: Routledge.

Benítez-Burraco, Antonio & Cedric Boeckx. 2014. Language disorders and language evolution: Constraints on hypotheses. *Biological Theory* 9, 269–274.

Benítez-Burraco, Antonio & Cedric Boeckx. 2015. Approaching motor and language deficits in autism from below: A biolinguistic perspective. *Frontiers in Integrative Neuroscience* 9, 1–4.

Benítez-Burraco, Antonio & Elliot Murphy. 2016. The oscillopathic nature of language deficits in autism: from genes to language evolution. *Frontiers in Human Neuroscience* 10: 120. doi:10.3389/fnhum.2016.00120.

Berwick, Robert C., Angela D. Friederici, Noam Chomsky & Johan J. Bolhuis. 2013. Evolution, brain, and the nature of language. *Trends in Cognitive Sciences* 17, 89–98.

Berwick, Robert C. & Noam Chomsky. 2016. *Why Only Us: Language and Evolution*. Cambridge, MA: MIT Press.

Billard, Catherine, Joel Fluss & Florence Pinton. 2009. Specific language impairment versus Landau-Kleffner syndrome. *Epilepsia* 50 (Suppl. 7), 21–24.

Bishop, Dorothy V.M. 1982. Comprehension of spoken, written and signed sentences in childhood language disorders. Journal of Child Psychology and Psychiatry 23, 1–20.

Bishop, Dorothy V. M. 1985. Age of onset and outcome in 'acquired aphasia with convulsive disorder' (Landau-Kleffner syndrome). *Developmental Medicine and Child Neurology* 27, 705–12.

Bishop, Dorothy V. M. 2000. Pragmatic language impairment: a correlate of SLI, a distinct subgroup, or part of the autistic continuum? In Dorothy V.M. Bishop & Laurence B. Leonard (eds.), *Speech and Language Impairments in Children: Causes, Characteristics, Intervention and Outcome*, 99–113. London/New York: Routledge.

Bishop, Dorothy V. M. & Laurence B. Leonard (eds.) 2000. *Speech and Language Impairments in Children: Causes, Characteristics, Intervention and Outcome*. London/New York: Routledge.

Boeckx, Cedric & Víctor M. Longa. 2011. Lenneberg's views on language development and evolution and their relevance for modern biolinguistics. *Biolinguistics* 5, 254–273.

Boeckx, Cedric & Kleanthes K. Grohmann (eds.). 2013. *The Cambridge Handbook of Biolinguistics*. Cambridge: Cambridge University Press.

Buzsáki, György. 2006. *Rhythms of the Brain*. Oxford: Oxford University Press.

Buzsáki, György & Brendon O. Watson. 2012. Brain rhythms and neural syntax: Implications for efficient coding of cognitive content and neuropsychiatric disease. *Dialogues in Clinical Neuroscience* 14, 345–367.

Catani, Marco & Marsel Mesulam. 2008a. What is a disconnection syndrome? *Cortex* 44, 911–913.

Catani, Marco & Marsel Mesulam. 2008b. The arcuate fasciculus and the disconnection theme in language and aphasia: History and current state. *Cortex* 44, 953–961.

Chomsky, Carol. 1986. Analytic study of the Tadoma method: Language abilities of three deaf-blind subjects. *Journal of Speech and Hearing Research* 29, 332–347.

Chomsky, Noam. 1965. *Aspects of the Theory of Syntax*. Cambridge, MA: MIT Press.

Chomsky, Noam. 1967. The formal nature of language. Appendix A in Lenneberg (1967), pp. 397–442. New York: Wiley.

Chomsky, Noam. 1986. *Knowledge of Language: Its Nature, Origin and Use*. New York: Praeger.

Chomsky, Noam. 2013. Problems of projection. *Lingua* 130, 33–49.

Chomsky, Noam. 2016. *What Kind of Creatures Are We?* New York: Columbia University Press.

Chomsky, Noam. 2017a. The language capacity: Architecture and evolution. *Psychonomic Bulletin & Review* 24, 200–203.

Chomsky, Noam. 2017b. Language architecture and its import for evolution. *Neuroscience and Biobehavioral Reviews*. doi:10.1016/j.neubiorev.2017.01.053.

Crain, Stephen, Loes Koring & Rosalind Thornton. 2016. Language acquisition from a biolinguistic perspective. *Neuroscience and Biobehavioral Reviews*. doi:10.1016/j.neubiorev.2016.09.004.

Curtiss, Susan. 1977. *Genie: A Psycholinguistic Study of a Modern Day "Wild Child."* New York: Academic Press.

DaSilva, Ednéa, A., Diane C. Chungani, Otto Muzik & Harry T. Chungani. 1997. Landau-Kleffner syndrome: metabolic abnormalities in temporal lobe are a common feature. *Journal of Child Neurology* 12, 489–495.

Deonna, Thierry. 1991. Acquired epileptiform aphasia in children (Landau-Kleffner syndrome). *Journal of Clinical Neurophysiology* 8, 288–298.

Deonna, Thierry. 2000. Acquired epileptic aphasia (AEA) or Landau-Kleffner syndrome: from childhood to adulthood. In Dorothy V.M. Bishop & Laurence B. Leonard (eds), *Speech and Language Impairments in Children: Causes, Characteristics, Intervention and Outcome*, 261–272. London/New York: Routledge.

Deonna, Thierry, Anne Beaumanoir, François Gaillard & Gil Assal. 1977. Acquired aphasia in childhood with seizure disorder: a heterogeneous syndrome. *Neuropädiatrie* 8, 263–273.

Deonna, Thierry, Anne-Claude Prelaz-Girod, Claire Mayor-Dubois & Eliane Roulet-Perez. 2009. Sign language in Landau-Kleffner syndrome. *Epilepsia* 50, 77–82.

Deonna, Thierry & Eliane Roulet-Perez. 2010. Early-onset acquired epileptic aphasia (Landau-Kleffner syndrome, LKS) and regressive autistic disorders with epileptic EEG abnormalities: the continuing debate. *Brain & Development* 32, 746–752.

Deonna, Thierry & Eliane Roulet-Perez. 2016. *The Epilepsy-Aphasia Spectrum: From Landau-Kleffner Syndrome to Rolandic Epilepsy.* London: Mac Keith Press.

Everaert, Martin B.H., Marinus A.C. Huybregts, Noam Chomsky, Robert C. Berwick & Johan J. Bolhuis. 2015. Structures, not strings: Linguistics as part of the cognitive sciences. *Trends in Cognitive Sciences* 19, 729–743.

Faria, Paula, Felipe Fregni, Fernando Sebastião, Ana I. Dias & Alberto Leal. 2012. Feasibility of focal transcranial DC polarization with simultaneous EEG recording: preliminary assessment in healthy subjects and human epilepsy. *Epilepsy & Behavior* 25, 417–425.

Fernández, Javier Ramírez. 2015. Locality in language and locality in brain oscillatory structures. *Biolinguistics* 9, 74–95.

Giraud, Anne-Lise, Andreas Kleinschmidt, David Poeppel, Torben E. Lund, Richard S.J. Frackowiak & Helmut Laufs. 2007. Endogenous cortical rhythms determine cerebral specialization for speech perception and production. *Neuron* 56, 1127–1134.

Gleitman, Lila & Barbara Landau. 2013. Every child an isolate: Nature's experiments in language learning. In Massimo Piattelli-Palmarini & Robert C. Berwick (eds.), *Rich Languages from Poor Inputs*, 91–104. Oxford: Oxford University Press.

Gordon, Neil. 1990. Acquired aphasia in childhood: the Landau-Kleffner syndrome. *Developmental Medicine and Child Neurology* 32, 270–274.

Gordon, Neil. 1997. The Landau-Kleffner syndrome: increased understanding. *Brain & Development* 19, 311–316.

Gou, Zhenkun, Naseem Choudhury & April A. Benasich. 2011. Resting frontal gamma power at 16, 24 and 36 months predicts individual differences in language and cognition at 4 and 5 years. *Behavioural Brain Research* 220, 263–270.

Hickok, Gregory. 2009. The functional neuroanatomy of language. *Physics of Life Reviews* 6, 121–143.

Hickok, Gregory. 2012. Computational neuroanatomy of speech production. *Nature Reviews Neuroscience* 13, 135–145.

Hickok, Gregory, John Houde & Feng Rong. 2011. Sensorimotor integration in speech processing: Computational basis and neural organization. *Neuron* 69, 407–422.

Hickok, Gregory & David Poeppel. 2007. The cortical organization of speech processing. *Nature Reviews Neuroscience* 8, 393–402.

Hinzen, Wolfram & Joana Rosselló. 2015. The linguistics of schizophrenia: thought disturbance as language pathology across positive symptoms. *Frontiers in Psychology* 6. doi:10.3389/fpsyg.2015.00971.

Hinzen, Wolfram, Joana Rosselló, Otávio Mattos, Kristen Schroeder & Elisabet Vila. 2015. The image of mind in the language of children with autism. *Frontiers in Psychology* 6. doi:10.3389/fpsyg.2015.00841.

Hirsch, Edouard, Maria Paola Valenti, Gabrielle Rudolf, Caroline Seegmuller, Anne de Saint Martin, Pierre Maquet, Norma Wioland, Marie-Noëlle Metz-Lutz, Christian Marescaux & Alexis Arzimanoglou. 2006. Landau-Kleffner syndrome is not an eponymic badge of ignorance. *Epilepsy Research* 70, 239–247.

Hoshi, Koji & Kyoko Miyazato. 2016. Architecture of human language from the perspective of a case of childhood aphasia—Landau-Kleffner syndrome. *Biolinguistics* 10, 136–196.

Jenkins, Lyle. 2000. *Biolinguistics: Exploring the Biology of Language*. Cambridge: Cambridge University Press.

Kaga, Makiko. 1999. Language disorders in Landau-Kleffner syndrome. *Journal of Child Neurology*, 118–122.

Kaga, Makiko. 2000. Yoosyoozi no tyookakusitunin-Landau-Kleffner syookoogun to herupesu nooen kooisyoo [Auditory agnosia in children—Landau-Kleffner syndrome and herpes encephalitis sequela]. In Kimitaka Kaga (ed.), *Tyuusuusei tyookaku syoogai no kiso to rinsyoo* [*Basics and Clinical Medicine in Central Nervous Auditory Disorders*], 90–94. Tokyo: Kanehara Shuppan.

Kaga, Makiko. 2011. Landau-Kleffner syndrome. *Rinsyoo Seisin Igaku* 40, 325–327.

Kuhl, Patricia K. 1993. Early linguistic experience and phonetic perception: implications for theories of developmental speech perception. *Journal of Phonetics* 21, 125–139.

Kuhl, Patricia K., Barbara T. Conboy, Denise Padden, Tobey Nelson & Jessica Pruitt.

2005. Early speech perception and later language development: Implications for the "critical period." *Language Learning and Development* 1, 237–264.

Landau, William & Frank Kleffner. 1957. Syndrome of acquired aphasia with convulsive disorder in children. *Neurology* 7, 523–530.

Lenneberg, Eric. 1962. Understanding language without ability to speak: A case report. *Journal of Abnormal and Social Psychology* 65, 419–425.

Lenneberg, Eric. 1964. Language disorders in childhood. *Harvard Educational Review* 34(2), 152–177.

Lenneberg, Eric. 1967. *Biological Foundations of Language*. New York: Wiley.

Lenneberg, Eric. 1969. On explaining language. *Science* 164, 635–643.

Lenneberg, Eric. 1975. In search of a dynamic theory of aphasia. In Eric H. Lenneberg & Elizabeth Lenneberg (eds.), *Foundations of Language Development: A Multidisciplinary Approach, vol.2*, 3–20. New York: Academic Press / Paris: The UNESCO Press.

MacNeilage, Peter F. & Barbara L. Davis. 2001. Motor mechanisms in speech ontogeny: phylogenetic, neurobiological and linguistic implications. *Current Opinion in Neurobiology* 11, 696–700.

Massa, Rita, Anne de Saint-Martin, Edouard Hirsch, Christian Marescaux, Jacques Motte, Caroline Seegmuller, Catherine Kleitz, Marie-Noëlle Metx-Lutz. 2000. Landau-Kleffner syndrome: sleep EEG characteristics at onset. *Clinical Neurophysiology* 111 (Suppl. 2), 87–93.

Mikati, Mohamad Abdul, Rana M. Kurdi & Alhan N. Shamseddine. 2010. Landau Kleffner syndrome. In Harry A. Whitaker (ed.), *Concise Encyclopedia of Brain and Language*, 259–263. Oxford: Elsevier.

Murphy, Elliot. 2015. The brain dynamics of linguistic computation. *Frontiers in Psychology* 6. doi:10.3389/fpsyg.2015.01515.

Murphy, Elliot. 2016. The human oscillome and its explanatory potential. *Biolinguistics* 10, 6–20.

Murphy, Elliot & Antonio Benítez-Burraco. 2016a. Language deficits in schizophrenia and autism as related oscillatory connectomopathies: An evolutionary account. *Neuroscience and Biobehavioral Reviews.* doi:10.1016/j.neubiorev.2016.07.029.

Murphy, Elliot & Antonio Benítez-Burraco. 2016b. Bridging the gap between genes and language deficits in schizophrenia: An oscillopathic approach. *Frontiers in Human Neuroscience* 10, 422. doi:10.3389/inhum.2016.00422.

Nitsche, Michael A. & Walter Paulus. 2000. Excitability changes induced in the human motor cortex by weak transcranial direct current stimulation. *Journal of Physiology* 527, 633–639.

O'Grady, William D. 2005. *How Children Learn Language*. Cambridge: Cambridge University Press.

O'Hare, Anne. 2008. Commentary: Age of onset and outcome in Landau-Kleffner syndrome (1985). *Developmental Medicine and Child Neurology* 50, 724.

Pearl, Philip L., Enrique J. Carrazana & Gregory L. Holmes. 2001. The Landau-Kleffner syndrome. *Clinical Science* 1, 39–45.

Penfield, Wilder & Lamar Roberts. 1959. *Speech and Brain-Mechanisms*. Princeton: Princeton University Press.

Petitto, Laura-Ann, Clifton Langdon, Adam Stone, Diana Andriola, Geo Kartheiser

& Casey Cochran. 2016. Visual sign phonology: Insights into human reading and language from a natural soundless phonology. *WIREs Cognitive Science*. doi:10.1002/wcs.1404.

Piekarski, David J., Carolyn M. Johnson, Josiah R. Boivin, A. Wren Thomas, Wan Chen Lin, Kristen Delevich, Ezequiel M. Galarce & Linda Wilbrecht. 2017. Does puberty mark a transition in sensitive periods for plasticity in the associative neocortex? *Brain Research* 1654, 123–144.

Pinker, Steven. 1994. *The Language Instinct: How the Mind Creates Language*. New York: Harper Collins.

Poeppel, David. 2003. The analysis of speech in different temporal integration windows: cerebral lateralization as 'asymmetric sampling in time'. *Speech Communication* 41, 245–255.

Pullens, Pim, Will Pullens, Vera Blau, Bettina Sorger, Bernadette M. Jansma & Rainer Goebel. 2015. Evidence for normal letter-sound integration, but altered language pathways in a case of recovered Landau-Kleffner Syndrome. *Brain and Cognition* 99, 32–45.

Ramanathan, Ramnath Santosh, Tina Ahluwalia & Ankush Sharma. 2012. Landau-Kleffner syndrome- a rare experience. *Eastern Journal of Medicine* 17, 36–39.

Rapin, Isabelle, Steven Mattis, A. James Rowan & Gerald G. Golden. 1977. Verbal auditory agnosia in children. *Developmental Medicine and Child Neurology* 19, 192–207.

Robinson, Richard O., Gillian Baird, Gary Robinson & Emily Simonoff. 2001. Landau-Kleffner syndrome: Course and correlates with outcome. *Developmental Medicine & Child Neurology* 43, 243–247.

Shultz, Sarah, Athena Vouloumanos, Randi H. Bennet & Kevin Pelphrey. 2014. Neural specialization for speech in the first months of life. *Developmental Science* 17, 766–774.

Stefanatos, Gerry. 2011. Changing perspectives on Landau-Kleffner syndrome. *The Clinical Neuropsychologist* 25, 963–988.

Steinlein, Ortrud K. 2009. Epilepsy-aphasia syndromes. *Expert Review of Neurotherapeutics* 9, 825–833.

Sugisaki, Koji. 2016. Structure dependence in child English: New evidence. In Koji Fujita & Cedric Boeckx (eds.), *Advances in Biolinguistics: The Human Language Faculty and Its Biological Basis*, 69–82. London: Routledge.

Tharpe, Anne Marie & Barbara J. Olson. 1994. Landau-Kleffner syndrome: Acquired epileptic aphasia in children. *Journal of the American Academy of Audiology* 5, 146–150.

Tsimpli, Ianthi Maria, Maria Kambanaros & Kleanthes K. Grohmann. Language pathology. To appear in Ian G. Roberts (ed.), *The Oxford Handbook on Universal Grammar*. Oxford: Oxford University Press.

Tsuru, Noriko & Thomas J. Hoeppner. 2007. Brain mechanism of language and its disorder: From the viewpoint of Landau-Kleffner syndrome. *Rinshounouha* 49, 305–311.

Uldall, Peter, Lene Sahlholdt & Jørgen Alving. 2000. Landau-Kleffner syndrome with onset at 18 months and an initial diagnosis of pervasive developmental disorder. *European Journal of Paediatric Neurology* 4, 81–86.

Yang, Charles, Stephen Crain, Robert C. Berwick, Noam Chomsky & Johan J. Bolhuis. 2017. The growth of language: Universal Grammar, experience, and principles of computation. *Neuroscience and Biobehavioral Reviews*. doi:10.1016/j.neubiorev.2016.12.023.

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