Review Article

Electrophysiological Correlates of Auditory Temporal Processing: A Hierarchical Framework from Brainstem to Cortex

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Short running title: Electrophysiological Correlates of Auditory…

Highlights:

- ABR and FFR objectively assess hierarchical temporal encoding
- Temporal coding gradually shifts from brainstem to cortical processing
- Electrophysiology objectively tracks auditory training-induced plasticity.

ABSTRACT

Background and Aim: Auditory temporal processing (ATP), the ability of the auditory system to encode and interpret rapid acoustic changes over time, is essential for speech perception, language development, and effective communication. Impairments in ATP are a hallmark of several auditory and neurodevelopmental disorders. This narrative review synthesizes current evidence on the electrophysiological correlates of ATP and proposes a hierarchical neurophysiological framework linking brainstem and cortical processing through objective electrophysiological measures.

Recent Findings: Contemporary evidence indicates a progressive shift in the relative contribution of temporal and rate-based neural coding across the auditory pathway rather than an abrupt transition between subcortical and cortical processing. Brainstem responses, including the Auditory Brainstem Response (ABR) and Frequency-Following Response (FFR), provide objective measures of neural synchrony and temporal fidelity, whereas cortical auditory evoked potentials, such as the N1–P2 complex and Mismatch Negativity (MMN), reflect increasingly complex sensory and cognitive processing. Integrating these complementary measures enables more precise characterization of neural dysfunction in clinical populations, including auditory neuropathy spectrum disorder, developmental dyslexia, and age-related hearing loss. Furthermore, growing evidence demonstrates that

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auditory training can induce experience-dependent plasticity, enhancing both cortical processing and the fidelity of subcortical neural encoding.
Conclusion: Electrophysiological techniques offer a non-invasive means of assessing ATP across multiple levels of the auditory system and provide promising objective biomarkers for diagnosis, monitoring intervention outcomes, and guiding individualized rehabilitation. Continued progress toward standardized recording protocols, multimodal assessment strategies, and longitudinal validation will facilitate the translation of hierarchical electrophysiological models into routine clinical practice.
Keywords: Auditory temporal processing; auditory brainstem response; frequency-following response; cortical auditory evoked potentials; auditory plasticity; central auditory processing.

Introduction

Auditory temporal processing (ATP), defined as the ability of the auditory system to detect, encode, and interpret acoustic changes over time, is a fundamental component of central auditory processing and a prerequisite for effective communication [1, 2]. Accurate temporal processing enables listeners to extract rapidly changing acoustic cues that are essential for speech perception, language acquisition, and auditory scene analysis [3, 4]. Deficits in ATP have been associated with impaired perception of rapid speech, reduced phoneme discrimination, and difficulties in processing prosody, rhythm, and musical information, ultimately affecting communication and learning [5–8].

ATP comprises several interrelated processes, among which temporal resolution and temporal ordering are considered the core functional components. Temporal resolution refers to the ability to detect brief acoustic events or gaps occurring within milliseconds, whereas temporal ordering reflects the capacity to perceive the sequential arrangement of auditory stimuli [9–13]. Although these processes are commonly evaluated using behavioral paradigms, behavioral performance may be influenced by attention, motivation, language proficiency, and cognitive abilities. Consequently, objective electrophysiological techniques have become increasingly important because they provide direct measures of neural activity across multiple levels of the auditory pathway, independent of overt behavioral responses.

Recent advances in auditory neuroscience have demonstrated that ATP is supported by a hierarchical neural network extending from the auditory nerve and brainstem to the auditory cortex. Rather than reflecting an abrupt transition, there is a gradual shift in the relative contribution of temporal and rate-based neural coding across the auditory hierarchy. Brainstem responses, including the Auditory Brainstem Response (ABR) and Frequency-Following Response (FFR), primarily reflect neural synchrony and temporal fidelity, whereas cortical auditory evoked potentials, such as the N1–P2 complex, Mismatch Negativity (MMN), and P300, represent progressively higher levels of sensory and cognitive processing. Accordingly, this review synthesizes current evidence on the electrophysiological correlates of ATP within this hierarchical framework (Figure 1), evaluates their application in characterizing auditory temporal processing deficits in clinical populations—including auditory neuropathy spectrum disorder, developmental dyslexia, and age-related hearing loss—and discusses evidence for experience-dependent plasticity and its implications for objective assessment and individualized auditory rehabilitation.

Methods

This study was conducted as a narrative (non-systematic) review with the objective of synthesizing and critically interpreting the existing literature to develop a hierarchical neurophysiological framework for auditory temporal processing. A comprehensive literature search was performed using PubMed/MEDLINE, Scopus, and Web of Science to identify relevant publications from January 1958 through December 2025. Google Scholar was used as a supplementary source to identify additional studies through citation tracking. The search strategy incorporated combinations of the following keywords: auditory temporal processing, electrophysiology, event-related potentials, auditory brainstem response, frequency-following response, auditory neuropathy, developmental dyslexia, and age-related hearing loss.

Studies were selected based on their relevance to the objectives of the review and the strength of their electrophysiological evidence. Eligible studies included peer-reviewed human investigations employing objective electrophysiological techniques to evaluate auditory temporal encoding or temporal processing deficits. Behavioral studies without electrophysiological measures, non-English publications, and animal studies lacking direct translational relevance to human auditory neurophysiology were excluded. Approximately 102 studies were ultimately synthesized to construct the proposed hierarchical framework. To maintain conceptual coherence,

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the review primarily focuses on clinical conditions for which substantial electrophysiological evidence is available, including auditory neuropathy spectrum disorder, developmental dyslexia, and age-related hearing loss. Other clinically relevant conditions, such as central auditory processing disorder and autism spectrum disorder, are briefly discussed as important directions for future research. The limitations of this narrative review are addressed in the final section of the manuscript.

Physiological mechanisms and hierarchical framework

The neural encoding of sound within the central auditory system depends on two complementary coding strategies: precise temporal coding (phase locking) and rate-based coding. Rather than operating independently, these mechanisms interact across different levels of the auditory pathway to encode the temporal, spectral, and spatial characteristics of acoustic signals.

Precise temporal coding refers to the synchronization of auditory neuronal firing to a specific phase of a periodic acoustic stimulus. This phase-locking mechanism is fundamental for encoding low-frequency pitch, temporal fine structure, sound timing, and binaural cues required for sound localization [14, 15]. Temporal synchrony is particularly robust within the auditory nerve, cochlear nucleus, and other brainstem nuclei, and is transmitted through topographically organized pathways to higher auditory centers, including the inferior colliculus and auditory cortex [14, 16]. Consequently, the integrity of subcortical temporal coding is essential for accurate speech perception, and disruption of neural synchrony may substantially impair speech understanding even in individuals with normal pure-tone hearing sensitivity [17–19].

Rate-based coding represents complementary acoustic information through changes in neuronal firing rates across tonotopically organized neural populations. This mechanism plays a critical role in encoding sound intensity and the spectral characteristics of complex acoustic stimuli, particularly at higher frequencies where phase locking becomes progressively less effective [20, 21].

A fundamental organizational principle of the auditory system is the hierarchical evolution of neural coding across successive processing stages. Rather than reflecting an abrupt transition, there is a gradual shift in the relative contribution of temporal and rate-based coding across the auditory hierarchy. Precise temporal coding predominates within the auditory nerve and brainstem, whereas the relative contribution of rate-based and higher-order integrative processing progressively increases at thalamic and cortical levels. Importantly, temporal coding is not eliminated within the cortex; instead, temporal synchrony and rate-based representations coexist, with their relative contributions depending on stimulus characteristics, neural population dynamics, and behavioral demands. Consequently, cortical auditory processing reflects an integrated interaction between these complementary coding strategies rather than a purely rate-coded system [14, 22].

Electrophysiological assessment across the auditory hierarchy

The complementary neural coding mechanisms described above provide the physiological basis for electrophysiological assessment of auditory temporal processing. Because ATP emerges through sequential processing across multiple neural levels, electrophysiological techniques offer objective measures of neural function throughout the auditory pathway and enable localization of dysfunction within the hierarchical system. Auditory evoked potentials provide an objective, non-invasive method for evaluating auditory pathway integrity with excellent temporal resolution [23]. Unlike behavioral assessments, these responses can be recorded across a wide range of ages and clinical populations with minimal dependence on language ability, attention, or cognitive status, making them valuable tools for both clinical diagnosis and auditory neuroscience research [24].

Subcortical electrophysiological measures

The ABR is the most widely used objective measure of brainstem auditory function and is routinely employed for threshold estimation and detection of retrocochlear disorders [25–31]. The response consists of a sequence of waves occurring within approximately 8–10 ms after stimulus onset. The principal neural generators of Waves I–V have been well established and include the distal auditory nerve (Wave I), proximal auditory nerve and cochlear nucleus (Wave II), superior olivary complex (Wave III), lateral lemniscus (Wave IV), and inferior colliculus (Wave V) [32–34]. Collectively, these waves reflect highly synchronized neural transmission through the auditory brainstem.

Beyond threshold estimation, ABR provides valuable information regarding neural synchrony. Reduced Wave I amplitude, prolonged interpeak latencies, or increased latency at high stimulus repetition rates may indicate neural

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dyssynchrony, impaired neural conduction, or degeneration of low-spontaneous-rate auditory nerve fibers [35–37]. Consequently, ABR remains one of the most informative objective measures for evaluating the integrity of subcortical auditory temporal processing.

The FFR complements ABR by assessing sustained neural encoding of the temporal characteristics of complex acoustic stimuli. FFR is a scalp-recorded response that reflects neural phase locking to both the temporal fine structure and envelope of periodic sounds. Although the response is generated predominantly within the auditory brainstem, accumulating evidence indicates additional contributions from the midbrain and, under specific stimulus conditions, cortical generators [38–40]. Because FFR directly indexes the fidelity of neural temporal encoding, it has become an important biomarker for investigating speech processing, auditory expertise, developmental disorders, and experience-dependent neuroplasticity. Recent studies have further demonstrated that FFR is sensitive to auditory training, highlighting its value for objectively monitoring plastic changes within the auditory system [41–45].

Cortical electrophysiological measures

Moving upward within the auditory hierarchy, the middle latency response reflects neural activity within the thalamo-cortical pathway approximately 10–50 ms after stimulus onset. The Na and Pa components primarily arise from interactions among the inferior colliculus, medial geniculate body, and primary auditory cortex, providing an objective assessment of early cortical activation [46–50].

The Late Latency Response (LLR), typically recorded between 50 and 250 ms, reflects cortical sensory processing of auditory stimuli. Among its components, the N1–P2 complex is the most robust and clinically useful. The N1 primarily reflects cortical detection of sound onset, whereas the P2 component is influenced by stimulus characteristics, attentional state, and long-term auditory experience, making it particularly sensitive to auditory learning and cortical plasticity [10, 51-53].

Beyond sensory responses, cortical event-related potentials provide insight into higher-order auditory processing. The MMN is an automatic, pre-attentive response elicited by deviant auditory stimuli and reflects auditory sensory memory together with early cortical change detection. Because MMN is highly sensitive to temporal and spectral deviations, it has become one of the most widely used electrophysiological markers of cortical auditory temporal processing [54, 55].

The P300 represents a later cognitive component associated with conscious stimulus evaluation, attentional resource allocation, and auditory discrimination. This positive potential, typically peaking around 300 ms over centroparietal scalp regions, is generated through distributed cortical networks involving frontal, temporal, and parietal regions [56, 57]. Accordingly, P300 provides an objective measure of top-down cognitive processing that complements the sensory information obtained from earlier cortical responses.

Electrophysiological profiles in clinical populations

Auditory neuropathy spectrum disorder

Auditory Neuropathy Spectrum Disorder (ANSD) is characterized by preserved cochlear outer hair cell function despite disrupted neural transmission along the auditory pathway. Clinically, the disorder is identified by the presence of Otoacoustic Emissions (OAEs) and/or Cochlear Microphonics (CM) in conjunction with an absent or severely abnormal ABR [58–62]. Within the proposed hierarchical framework, ANSD represents a prototypical disorder of subcortical temporal encoding, in which impaired neural synchrony compromises the faithful transmission of temporal information from the auditory nerve to higher auditory centers.

The consequences of this disrupted neural synchrony extend beyond the brainstem. Abnormal cortical electrophysiological responses, including delayed N1 latencies and elevated N1-P2 thresholds for temporal gap detection, indicate that impaired subcortical encoding adversely affects cortical temporal processing [63–65]. Interestingly, some individuals with ANSD exhibit relatively preserved cortical responses despite markedly abnormal brainstem activity, suggesting that compensatory cortical reorganization or adaptive neural plasticity may partially offset deficient ascending input [63, 66]. Nevertheless, the characteristic electrophysiological profile of ANSD reflects a breakdown of precise neural phase locking that propagates throughout the auditory hierarchy and contributes substantially to the severe speech-in-noise difficulties commonly experienced by affected individuals [17, 67].

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Age-related changes

Advancing age is associated with progressive deterioration of auditory temporal processing, reflected by abnormalities across multiple electrophysiological measures. Older adults commonly demonstrate poorer temporal resolution, including impaired gap detection and reduced sensitivity to amplitude modulation. These behavioral deficits are paralleled by reduced FFR amplitudes and prolonged ABR latencies, indicating diminished neural synchrony and slower subcortical processing [68–74]. Age-related peripheral hearing loss may further exacerbate these deficits by degrading the quality of afferent input reaching central auditory structures.

Cortical electrophysiological responses also undergo characteristic age-related alterations. Reduced MMN amplitudes suggest declining auditory sensory memory and less efficient automatic detection of acoustic changes, whereas relatively preserved P300 responses to temporal gap paradigms imply greater reliance on compensatory top-down attentional mechanisms [75–77]. Another consistent finding is enhanced cortical tracking of slow temporal envelopes in older listeners, which is thought to represent an adaptive strategy that compensates for reduced temporal fine-structure encoding by increasing dependence on envelope-based speech cues [18, 78–82].

Developmental dyslexia

Developmental dyslexia is widely recognized as a disorder in which impaired phonological processing is closely associated with deficits in auditory temporal processing. Current neurophysiological models, particularly the Temporal Sampling Framework and rapid auditory processing theory, propose that inefficient neural encoding of rapidly changing acoustic information contributes to the development of phonological deficits and reading difficulties [83, 84].

Unlike ANSD, individuals with developmental dyslexia generally exhibit normal ABR morphology and latency, indicating preserved gross brainstem synchrony. However, more sensitive electrophysiological measures reveal abnormalities in the precision of temporal encoding. At the subcortical level, reduced FFR fidelity to rapid formant transitions and temporal fine structure has been consistently reported, suggesting impaired neural representation of rapidly changing speech cues [85–88].

Cortical electrophysiological findings further support the presence of hierarchical dysfunction. Reduced or delayed MMN responses to subtle temporal contrasts indicate impaired automatic auditory discrimination, while alterations in the N1–P2 complex demonstrate reduced efficiency in processing rapid acoustic transitions and brief temporal gaps [89–92]. Importantly, these cortical abnormalities correlate with phonological awareness and reading performance, supporting the view that subtle instability in subcortical temporal encoding may cascade through the auditory hierarchy, ultimately increasing cognitive demands during speech perception and language processing [83, 93].

Synthesis and comparative analysis

Collectively, the electrophysiological findings across ANSD, age-related hearing loss, and developmental dyslexia demonstrate that auditory temporal processing deficits arise from disruption at different levels of the auditory hierarchy rather than from a single neural locus. Although all three conditions impair auditory temporal processing, they exhibit distinct electrophysiological signatures that reflect differences in the underlying mechanisms of neural encoding. As summarized in Table 1, ANSD is characterized primarily by impaired neural synchrony at the auditory nerve and brainstem, developmental dyslexia by reduced precision of temporal encoding despite relatively preserved gross brainstem conduction, and age-related hearing loss by a progressive hierarchical decline involving both subcortical degradation and compensatory cortical recruitment.

Importantly, abnormalities occurring at one level of the auditory pathway frequently influence neural processing at subsequent stages. Consequently, deficits in brainstem temporal encoding may alter cortical sensory and cognitive processing, whereas cortical compensatory mechanisms may partially mitigate degraded ascending input. These findings emphasize that auditory temporal processing depends on the coordinated interaction of distributed neural networks extending from the auditory nerve to the cerebral cortex. Therefore, a hierarchical electrophysiological approach that combines complementary brainstem and cortical measures provides a more comprehensive understanding of auditory temporal processing disorders than evaluation of any single response alone. Such an approach also offers a robust framework for diagnosis, monitoring disease progression, and guiding individualized rehabilitation strategies.

Experience-dependent plasticity and training-induced electrophysiological changes

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The hierarchical organization of auditory temporal processing further provides a valuable framework for understanding experience-dependent neural plasticity. Because electrophysiological responses can be recorded objectively at multiple levels of the auditory pathway, they offer unique biomarkers for monitoring how auditory training reshapes neural processing from the brainstem to the cortex.

Targeted auditory training induces measurable neurophysiological changes throughout the auditory hierarchy. At the cortical level, successful intervention is consistently associated with increased N1–P2 amplitudes, reflecting enhanced thalamo-cortical connectivity, improved neural synchrony, and more efficient cortical sensory encoding [94–96]. Importantly, these electrophysiological changes may precede measurable behavioral improvements, suggesting that reorganization of synchronous neural activity constitutes an essential neurophysiological foundation for subsequent perceptual gains [96, 97]. Auditory training has also been shown to shorten cortical response latencies and improve neural processing of speech in noise, with these objective changes correlating closely with enhanced speech perception performance [98, 99].

Experience-dependent plasticity is not limited to cortical structures. At the subcortical level, the FFR has emerged as a sensitive biomarker of brainstem plasticity. Auditory training enhances the fidelity of neural phase locking to both the temporal envelope and fine structure of speech, thereby improving temporal encoding and neural representation under adverse listening conditions [41, 42, 99]. These findings indicate that auditory training actively reshapes subcortical temporal coding rather than simply modifying higher-order cortical processing.

Higher-order cortical event-related potentials further complement this hierarchical assessment of plasticity. Increased MMN amplitudes following training indicate more robust pre-attentive auditory discrimination, whereas larger P300 amplitudes accompanied by shorter latencies reflect improved attentional resource allocation, cognitive processing efficiency, and neural synchronization [100–102]. Collectively, these electrophysiological changes demonstrate that auditory training strengthens neural processing across multiple hierarchical levels of the auditory system. Accordingly, objective electrophysiological measures provide powerful biomarkers for monitoring treatment efficacy and offer mechanistic insight into the neural adaptations that support successful auditory rehabilitation.

Conclusion

This review proposes a hierarchical neurophysiological framework for understanding auditory temporal processing by integrating electrophysiological evidence across successive levels of the auditory pathway, from the auditory nerve and brainstem to higher cortical networks. Rather than representing an abrupt transition, the available evidence supports a progressive shift in the relative contribution of temporal and rate-based neural coding across the auditory hierarchy, with complementary coding mechanisms operating throughout the auditory system.

Within this framework, objective electrophysiological measures provide complementary insights into distinct stages of auditory processing. Brainstem responses, including the Auditory Brainstem Response (ABR) and Frequency-Following Response (FFR), primarily reflect neural synchrony and temporal fidelity, whereas cortical auditory evoked potentials, including the N1–P2 complex, Mismatch Negativity (MMN), and P300, characterize progressively higher levels of sensory and cognitive processing. Together, these measures facilitate more precise localization of auditory temporal processing deficits in clinical conditions such as Auditory Neuropathy Spectrum Disorder, developmental dyslexia, and age-related hearing loss.

A major implication of this hierarchical model is that electrophysiological assessment extends beyond diagnosis to provide objective biomarkers of experience-dependent neural plasticity. Growing evidence indicates that auditory training can enhance both cortical processing and subcortical temporal encoding, supporting the use of electrophysiological measures for monitoring treatment outcomes and optimizing individualized rehabilitation strategies. Future research should focus on longitudinal validation, multimodal integration with advanced neuroimaging, and the development of standardized clinical protocols to facilitate routine implementation of hierarchical electrophysiological assessment in evidence-based auditory rehabilitation.

Limitations

This review was conducted as a narrative review and, therefore, did not follow a predefined systematic review or meta-analysis protocol. Consequently, study selection was based on relevance to the proposed conceptual framework rather than exhaustive protocol-driven identification, and no formal assessment of study quality or risk of bias was performed. The primary objective was to synthesize and critically interpret the available

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electrophysiological evidence to develop a coherent hierarchical model of auditory temporal processing. Accordingly, the conclusions should be interpreted as an evidence-informed conceptual synthesis rather than a quantitative summary of the literature.

Future directions

Translating advances in the electrophysiological assessment of auditory temporal processing into routine clinical practice will require coordinated progress across several complementary areas of research. A major priority is the development and validation of standardized protocols for recording and analyzing frequency-following responses and cortical auditory evoked potentials. Harmonized acquisition, processing, and reporting procedures across laboratories, clinical centers, and age groups are essential for improving measurement reliability, facilitating cross-study comparisons, and establishing robust normative databases suitable for clinical application.

Another important objective is the implementation of large-scale longitudinal studies to characterize the developmental and aging trajectories of auditory temporal processing. Such investigations will improve understanding of how electrophysiological responses evolve across the lifespan and provide normative benchmarks for the early detection of atypical neural processing associated with auditory processing disorders, neurodevelopmental conditions, and age-related auditory decline.

A more comprehensive understanding of the auditory processing hierarchy will also require integration of electrophysiological techniques with complementary neuroimaging modalities. Combining high-temporal-resolution electrophysiological measures with high-spatial-resolution techniques, such as functional magnetic resonance imaging and magnetoencephalography, will enable more accurate localization of the neural generators underlying auditory temporal processing and experience-dependent neuroplasticity. Simultaneous assessment of brainstem and cortical responses within the same individuals will further clarify how neural encoding at successive stages of the auditory pathway interacts under both normal and pathological conditions and how dysfunction at one level influences processing throughout the auditory hierarchy.

Future research should also strengthen the relationship between objective electrophysiological biomarkers and clinically meaningful functional outcomes. Establishing robust associations between neural measures and everyday listening abilities—including speech perception in noise, communication performance, attentional control, and working memory—will improve the ecological validity and diagnostic utility of electrophysiological assessment. Such evidence will facilitate the identification of biomarkers that not only characterize neural dysfunction but also predict functional performance and rehabilitation outcomes.

Ultimately, integrating hierarchical electrophysiological biomarkers into individualized intervention strategies represents an important step toward precision audiology. Auditory training programs tailored to each individual's neural profile and specific temporal coding deficits have the potential to optimize rehabilitation outcomes while providing objective measures for monitoring treatment-related neuroplasticity. Collectively, these advances will facilitate the routine clinical implementation of hierarchical electrophysiological assessment, enabling earlier diagnosis, more accurate prognostic evaluation, personalized rehabilitation, and evidence-based management of auditory temporal processing disorders.

Ethical Considerations

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Artificial intelligence disclosure

Artificial intelligence tools were utilized solely for language editing, grammatical correction, and improving textual clarity. All scientific content, literature synthesis, interpretation, and manuscript structure were developed independently by the authors.

Authors' contributions

FG: Conceptualization, study design, literature search, data extraction and synthesis, drafting the manuscript, and preparation of figures and tables; MEM: Conceptualization, study design, critical revision of the manuscript for intellectual content, supervision, and final approval of the version to be published.

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Conflict of interest

The authors have no conflicts of interest to declare.

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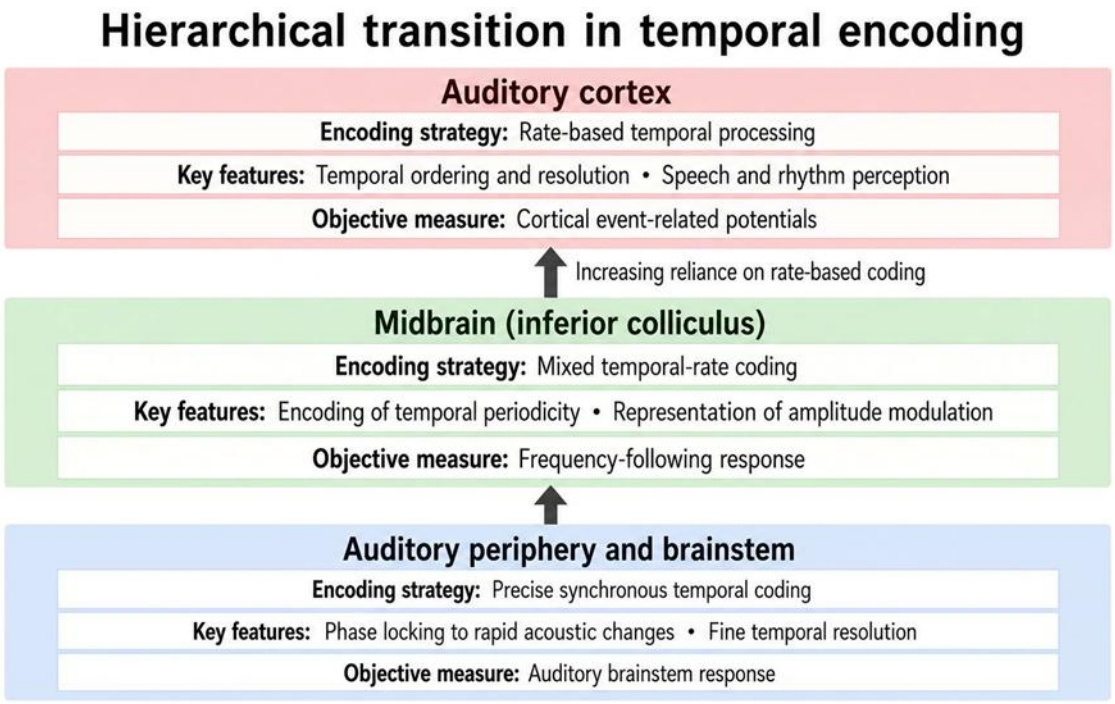

Figure 1. Hierarchical model of temporal encoding from brainstem to cortex

Table 1. Comparative electrophysiological profiles in auditory temporal processing disorders

Electrophysiological measure	Auditory neuropathy	Age-related decline	Developmental dyslexia
OAEs/CM	Preserved (intact OHC function)	Reduced/absent (with high-frequency loss)	Typically, normal
ABR	Absent/severely abnormal (impaired synchrony)	Present but delayed (peripheral loss)	Generally normal (latency/morphology)
FFR	Absent/grossly abnormal	Reduced amplitude and fidelity	Reduced consistency and fidelity to rapid transitions
MMN	Variable/delayed (upstream disruption)	Reduced amplitude (sensory memory decline)	Abnormal/reduced to temporal contrasts
N1-P2 complex	Delayed N1/elevated gap thresholds	Delayed latencies; altered amplitude	Deficits in processing rapid changes/gaps
P300	Variable (depends on detectability)	Preserved/enhanced (top-down compensation)	Altered (increased cognitive load)
Envelope tracking	Poor (upstream dyssynchrony)	Enhanced for slow modulation (<50 Hz)	Potential deficit in tracking syllabic rhythms

OAEs; otoacoustic emissions, CM; cochlear microphonic, OHC; outer hair cell, ABR; auditory brainstem response, FFR; frequency-following response, MMN; mismatch negativity

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