Auditory and Vestibular Research

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

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Highlights

ABR and FFR show brainstem's precise temporal coding.
Cortex relies on rate-based and cognitive coding .
Training induces plasticity measurable by Event-Related Potentials .

Abstract

Background and Aim: Auditory temporal processing (ATP), the ability to perceive acoustic changes over time, is fundamental for communication. Deficits in ATP underlie various auditory processing disorders. This review focuses on the electrophysiological correlates of ATP deficits in key clinical populations, including auditory neuropathy, dyslexia, and age-related hearing loss. The aim was to establish a hierarchical neurophysiological framework for understanding these deficits, from the brainstem to the cortex, using objective electrophysiological measures.

Recent Findings: Evidence reveals a systematic shift in neural encoding. The brainstem employs precise temporal coding, measured by the Auditory Brainstem Response (ABR) and Frequency-Following Response (FFR). In contrast, higher cortical centers utilize rate-based and cognitive coding for periodicity, captured by cortical Event-Related Potentials (ERPs) like the N1-P2 complex and Mismatch Negativity (MMN). This framework allows for the objective localization of dysfunction, such as neural dyssynchrony in auditory neuropathy. Critically, the system shows experience-dependent plasticity; targeted auditory training can normalize cortical responses and enhance subcortical encoding.

Conclusion: Electrophysiological measures provide a critical, non-invasive window into ATP. They offer validated biomarkers for diagnosing disorders, objectively validating rehabilitation efficacy, and guiding personalized intervention. Future translation into clinical practice requires standardized protocols and integrated multi-level assessments.

Keywords: Auditory temporal processing, electrophysiology, event-related potentials, central auditory processing, auditory training

Introduction

Auditory temporal processing (ATP) is a fundamental component of central auditory processing, defined as the ability to perceive acoustic changes over time and is crucial for deriving meaning from sound [1, 2]. It is intrinsically linked to speech perception, as comprehending speech requires the ability to recognize the temporal distinctions between different phonemes and syllables [3, 4]. Deficits in this domain lead to difficulties in processing rapid speech, differentiating phonemes, and appreciating music and rhythm [5, 6]. Furthermore, auditory temporal resolution is essential for assessing speech clarity and supports effective language development in children [7, 8]. While this process encompasses several sub-processes, it is primarily assessed through its core components of temporal ordering and resolution using specialized behavioral and electrophysiological tools [9, 10].

Temporal processing is vital for recognizing the acoustic characteristics of speech, such as prosody, accent, and rhythm. Temporal resolution is critical as most speech sound patterns rely on millisecond time differences [11]. Temporal ordering pertains to the processing of auditory stimuli in their sequence of occurrence. The integrity of

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these functions can be evaluated through both standardized behavioral tests and objective electrophysiological measures [12, 13].
This review synthesizes current evidence on the electrophysiological correlates of auditory temporal processing, tracing its hierarchical organization from brainstem to cortex. As illustrated in **Figure 1**, neural encoding transitions from precise phase-locking mechanisms in the brainstem (indexed by Auditory Brainstem Response (ABR) and Frequency-Following Response (FFR) to rate-based and cognitive periodicity coding at the cortical level (indexed by Mismatch Negativity (MMN) and P300). We evaluated the utility of these objective measures in characterizing temporal processing deficits in clinical populations such as auditory neuropathy, dyslexia, and age-related hearing loss. Furthermore, we examined the neurophysiological evidence for experience-dependent plasticity, exploring how targeted auditory training induces measurable functional changes within this hierarchical system.

Methods

This article constitutes a **narrative (non-systematic) review** designed to synthesize, critique, and interpret existing literature to construct a coherent hierarchical framework for auditory temporal processing. A comprehensive search was conducted across PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar for articles published between January 1958 and December 2025. Keywords included: "auditory temporal processing," "electrophysiology," "event-related potentials," "auditory brainstem response," "frequency-following response," "auditory neuropathy," "dyslexia," and "age-related hearing loss."
Approximately 350 articles were initially screened. Inclusion criteria required peer-reviewed human studies utilizing objective electrophysiological measures to investigate temporal encoding or clinical temporal processing deficits. Exclusion criteria removed purely behavioral studies, non-English publications, and animal models not directly informing human neurophysiology. Approximately 103 studies met criteria and were synthesized. The review focuses on populations with robust, established electrophysiological evidence (neuropathy, aging, dyslexia) to maintain conceptual coherence within the hierarchical framework. Broader conditions such as general Auditory Processing Disorder (APD) and Autism Spectrum Disorder, while relevant, are noted as important targets for future hierarchical electrophysiological research. Limitations are addressed at the end of the manuscript.

Physiological mechanisms and hierarchical framework

The neural encoding of sound in the central auditory system relies on two primary, complementary mechanisms: precise temporal coding (phase locking) and rate-based (place) coding.
Precise phase locking refers to the reliable synchronization of auditory neuron action potentials to a specific phase of a periodic sound. This temporal coding mechanism is fundamental for encoding low-frequency pitch, sound timing, and binaural cues for sound localization [14, 15]. The auditory periphery and cochlear nucleus rely heavily on this precise synchronous coding, which projects topographically to the inferior colliculus and cortex [14, 16]. The integrity of this brainstem processing is critical, as its deterioration severely impairs speech comprehension, even with normal pure-tone sensitivity [17–19].
Rate-based coding is a complementary strategy where sound features are represented by the firing rate of neurons organized within a tonotopic map. It is crucial for representing sound intensity and the spectral content of complex sounds, especially at higher frequencies where phase locking diminishes [20, 21]. A fundamental organizational principle of the central auditory system is a hierarchical shift from precise temporal coding in the brainstem to rate-based periodicity coding in higher cortical centers [14, 22]. While rate coding dominates in the cerebral cortex, synchronous temporal processing remains active, supporting an integrated hierarchical model.

Electrophysiological techniques for assessment

Electrophysiological techniques provide an objective method for assessing the central auditory pathway with high temporal precision. Auditory evoked potentials serve as valuable clinical tools for neurologic and audiological diagnosis [23]. Unlike behavioral tests, these objective measures can be reliably recorded regardless of a patient's age, cognitive state, or attention level [24].

Auditory brainstem response:

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The ABR is a standard subcortical evoked potential used clinically for estimating auditory thresholds and screening for retrocochlear pathologies [25–31]. It comprises a sequence of waves (I–VII) occurring within 8–10 ms post-stimulus. Waves I–V have distinct, well-mapped neural generators: Wave I (distal auditory nerve), Wave II (proximal nerve/cochlear nucleus), Wave III (superior olivary complex), Wave IV (lateral lemniscus), and Wave V (inferior colliculus) [32–34]. The ABR fundamentally represents synchronized neural activity from the auditory brainstem. Specific patterns, such as reduced Wave I amplitude or prolonged latency at high repetition rates, indicate neural dyssynchrony or loss of low-spontaneous-rate fibers [35–37].

Frequency-following response:
The FFR is a scalp-recorded potential that reflects neural phase-locking to a sound's temporal structure. It originates from coordinated activity in the auditory nerve and brainstem neurons, capable of tracking frequencies up to approximately 2000 Hz [38, 39]. Recordable with montages highlighting pontine or midbrain generators, the FFR directly captures sustained neural phase-locking to the fine structure and envelope of periodic sounds [40]. Recent research focuses on the FFR's ability to index experience-dependent plasticity in brainstem temporal processing [41–45].

Middle and late latency responses:
The MLR (10–50 ms) assesses thalamo-cortical circuitry, with Na and Pa waves originating from the inferior colliculus, thalamus, and primary auditory cortex [46–50]. Later, the ALLR (50–250 ms) reflects cortical sensory coding. The N1-P2 complex is the most consistent component. The N1 is a transient response to sound onset, while the P2 is influenced by attention, stimulus features, and long-term experience [10, 51, 52]. Cortical Event-Related Potentials (ERPs) are powerfully modulated by attention and cognitive state, linking subcortical encoding to perception, though they exhibit higher inter-subject variability than brainstem responses [10, 53].
Mismatch negativity and P300: The MMN is an automatic, pre-attentive ERP component elicited by deviant auditory stimuli, reflecting sensory memory and early cortical change detection. It is highly sensitive to temporal and spectral contrasts and routinely used to index early cortical temporal processing deficits [54, 55]. The P300 component assesses conscious recognition, attention, and stimulus discrimination. This positive wave, peaking around 300 ms at central-parietal sites, is primarily generated by non-auditory frontal and temporal cortices [56, 57], serving as a marker of top-down cognitive allocation during temporal tasks.

Clinical electrophysiological profiles

Auditory neuropathy spectrum disorder
Auditory neuropathy (AN) represents a dissociation between normal cochlear outer hair cell function and disrupted neural transmission. Diagnosis relies on preserved otoacoustic emissions (OAEs)/cochlear microphonics (CM) alongside an absent or severely abnormal ABR [58–63]. Within the hierarchical framework, AN exemplifies a primary failure of brainstem temporal encoding. This neural dyssynchrony profoundly disrupts central temporal resolution, evidenced by abnormal cortical responses including delayed N1 waves and elevated thresholds for N1-P2 complexes to temporal gaps [64–66]. Notably, some studies report near-normal cortical potentials in certain AN patient, suggesting compensatory cortical reorganization despite upstream dyssynchrony [64, 67]. The electrophysiological signature confirms a breakdown in precise subcortical phase-locking that propagates upstream, severely impacting speech-in-noise comprehension [17, 68].

Age-related changes in auditory temporal processing
Age-related declines in ATP are evident across multiple electrophysiological measures. Elderly individuals show impairments in gap detection and amplitude modulation perception, with physiological correlates including reduced FFR amplitudes and delayed ABR latencies [69–75]. These deficits are compounded by peripheral hearing loss, which degrades subcortical input. At the cortical level, aging alters automatic and cognitive processing: MMN is typically reduced, suggesting impaired auditory sensory memory, while preserved P300 responses to temporal gaps indicate compensatory top-down processing [76–78]. A particularly notable finding is the age-related enhancement of cortical envelope tracking for slower modulation rates, likely reflecting compensatory reliance on envelope cues to offset degraded temporal fine structure processing [18, 79–83].

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Developmental disorders: dyslexia and specific language impairment

In developmental dyslexia, a core deficit in phonological processing is strongly linked to anomalies in auditory temporal processing, primarily explained by the Temporal Sampling Framework and rapid temporal processing theory [84, 85]. Unlike AN, ABR morphology and latencies are generally normal in dyslexia, confirming intact brainstem gross synchrony but highlighting a specific deficit in the precision of rapid temporal encoding [86, 87]. At the subcortical level, individuals frequently show reduced FFR fidelity to rapid formant transitions and temporal fine structure [88, 89]. At the cortical level, MMN is often abnormal or reduced in response to subtle temporal contrasts, indicating impaired pre-attentive change detection [90, 91]. Cortical ERPs (N1, P2) reveal deficits in processing rapid rate changes and brief temporal gaps, directly correlating with phonological awareness [92, 93]. This profile supports a hierarchical disruption wherein subcortical timing instability impairs automatic cortical discrimination, increasing cognitive load during speech perception [84, 94].

Synthesis and comparative analysis

The disorders of AN, age-related decline, and developmental dyslexia all implicate ATP deficits but exhibit distinct electrophysiological profiles within the auditory hierarchy. **Table 1** summarizes these comparative profiles, aligning each condition with specific neural encoding failures and compensatory mechanisms.

Experience-dependent plasticity and electrophysiological training markers

Targeted auditory training drives significant experience-dependent plasticity, objectively measurable through hierarchical electrophysiological markers. Rather than relying on general rehabilitation principles, this section focuses on how training modifies neural encoding at specific levels of the auditory pathway.

At the cortical level, successful auditory training is frequently accompanied by significant increases in N1-P2 amplitude, reflecting enhanced thalamo-cortical connectivity and neural synchrony [95–97]. Critically, these neural changes can precede perceptual improvements, suggesting that plasticity at the level of synchronous neural ensemble firing establishes a necessary precondition for behavioral gains [97, 98]. Training also normalizes cortical response latencies and enhances subcortical processing of speech in noise, with neurophysiological improvements directly correlating with better behavioral speech perception [99, 100].

Subcortical plasticity is indexed by the FFR. Studies demonstrate that auditory training refines brainstem temporal encoding, enhancing the precision of phase-locking to speech envelopes and improving signal-to-noise representation [41, 42, 100]. This confirms that training-induced plasticity is not confined to higher cortical centers but actively reshapes subcortical temporal coding. Endogenous cortical potentials like the P300 and MMN also serve as biomarkers for training efficacy. Post-training, MMN amplitude increases signify more robust pre-attentive sound discrimination, while P300 shows increased amplitude and decreased latency, reflecting improved neural synchronization and attentional resource allocation [101–103]. These objective electrophysiological shifts validate auditory training as a mechanism for restoring hierarchical temporal processing, offering direct windows into plastic changes that underpin clinical rehabilitation.

Conclusion

This review establishes a coherent hierarchical neurophysiological framework for auditory temporal processing, tracing the transformation of temporal information from the brainstem to the cortex. The evidence demonstrates a systematic shift in neural encoding strategies: the auditory periphery and brainstem rely on precise temporal coding (Auditory Brainstem Response, Frequency-Following Response), while higher cortical centers increasingly utilize rate-based and cognitive coding (Mismatch Negativity, N1-P2, P300). Electrophysiological measures allow for the specific identification of neural dyssynchrony in auditory neuropathy, degraded subcortical timing in aging, and impaired rapid temporal encoding in dyslexia. Furthermore, targeted auditory training induces measurable functional improvements, objectively demonstrated by the normalization of cortical responses and refined subcortical encoding. This positions electrophysiology not only as a diagnostic tool but also as a critical biomarker for validating rehabilitation efficacy and guiding personalized intervention.

Limitations

As a narrative review, this work does not follow a formal systematic review or meta-analysis protocol. Therefore, the study selection process may be subject to author bias, and the strength of evidence is not quantitatively graded. The aim is conceptual integration and theory-building within the proposed hierarchical model rather than an

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exhaustive, protocol-driven aggregation of all available data. The conclusions are interpretive, drawn from a critical analysis of the assembled literature.

Future Directions

To translate these findings into clinical practice, future work must focus on:

1. Developing validated, uniform protocols for FFR and ERP acquisition across clinics and age groups to ensure reliability and comparability.
2. Conducting longitudinal studies to track the development and decline of ATP over time and establish normative electrophysiological trajectories.
3. Integrating high-temporal-resolution electrophysiology with high-spatial-resolution neuroimaging to better localize neural sources underlying deficits and plasticity.
4. Simultaneously assessing brainstem and cortical activity in the same individuals to obtain a holistic view of the auditory processing hierarchy.
5. Precisely quantifying how electrophysiological deficits relate to real-world listening challenges and cognitive factors like attention and memory.
6. Using individual neural profiles to tailor auditory training regimens, targeting specific deficits in temporal coding for more effective intervention.

By addressing these directions, the field can move toward routine electrophysiological assessment of the ATP hierarchy in diagnosing auditory processing disorders, monitoring therapeutic progress, and delivering evidence-based, personalized rehabilitation.

Authors' Contribution

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

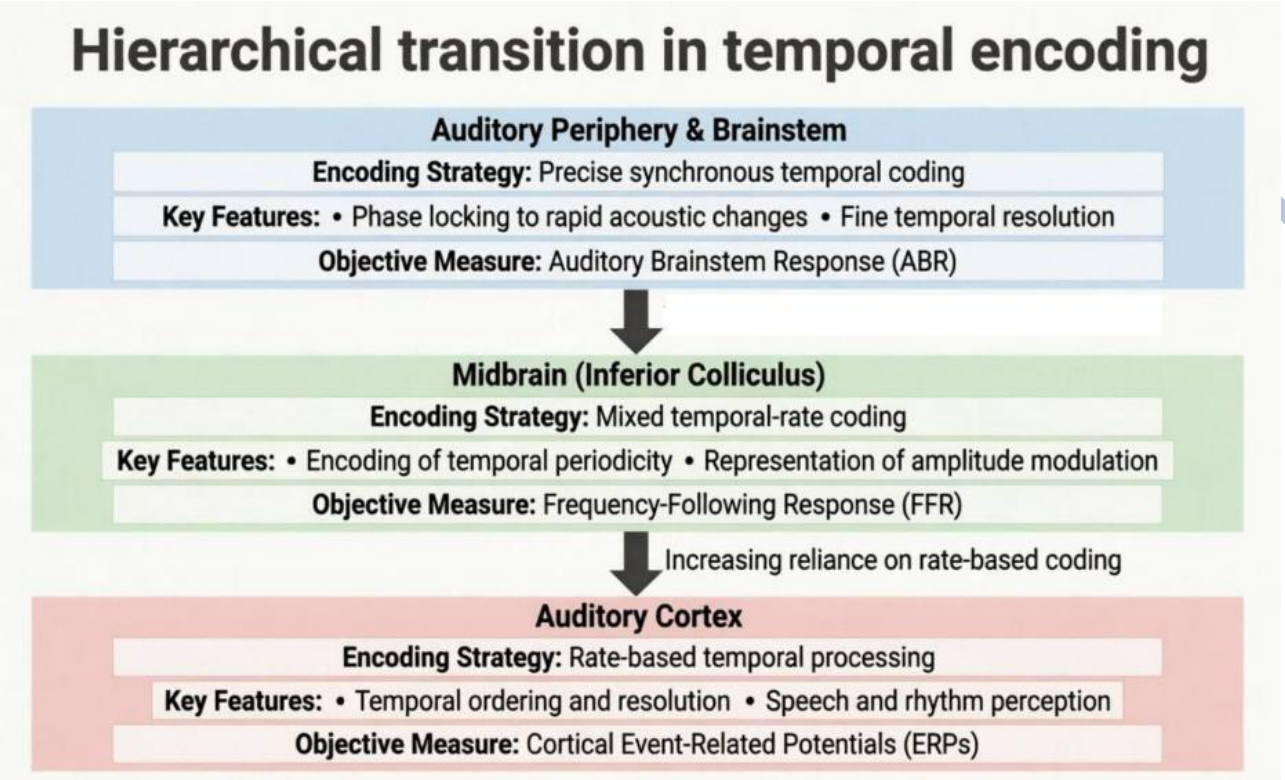

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Table 1. Comparative electrophysiological profiles in auditory temporal processing disorders

Electrophysiological Measure	Auditory Neuropathy (AN)	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 & Fidelity	Reduced Consistency & 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
Core Electrophysiological Deficit	Primary failure of neural phase-locking (Brainstem)	Degraded peripheral input + cortical compensation	Imprecise subcortical temporal encoding → cortical instability

Abbreviations: ABR = Auditory Brainstem Response; FFR = Frequency-Following Response; MMN = Mismatch Negativity; OAEs = Otoacoustic Emissions; CM = Cochlear Microphonic; OHC = Outer Hair Cell.

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