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Speech Perseveration in Elderly: A Neurobehavioral Analytic Reconceptualization

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Abstract

Perseverative speech is a well documented clinical phenomenon among elderly, characterized by the unintentional, verbatim repetition of previously emitted verbal responses, often occurring within minutes of the initial utterance. Individuals who exhibit this pattern repeat phrases with identical wording, prosody, and intonation, and typically demonstrate no awareness that the repetition has occurred. Unlike repetitive manding, anxiety driven repetition, or memory loss question asking, perseverative speech is not sensitive to social consequences; it is not influenced by correction or redirection, and not accompanied by metacognitive commentary (autoclitic tacts) such as "Did I already say this?" This phenomenon is also commonly observed in neurological conditions involving executive dysfunction, but it also appears in older adults without clear traumatic or degenerative diagnoses. Traditional or radical behaviorism offers limited tools for analyzing this form of repetition. The standard functional analytic framework—attention, escape, tangible, and automatic reinforcement—presumes that verbal behavior is shaped and maintained by environmental contingencies. However, perseverative speech often occurs independent of reinforcement history, independent of audience presence, and independent of antecedent manipulations. Neurocognitive models, while more mechanistically detailed, are also weak and poor guides for behavioral assessment. Given the limitations of both approaches, an integrative behavioral–neurological model is warranted. Such a model must preserve the observable, environmental focus of behavior analysis while incorporating the mechanistic insights of neuropsychology regarding inhibition, residual activation, and stimulus control updating. The proposed model conceptualizes perseverative speech as a multi stage failure of verbal control. Five stages of speech deconstruction explain the verbatim nature, identical intonation, short interval recurrence, and independence from reinforcement that define perseverative speech. Implications for applied research are discussed regarding clinical analogues for treatment.

Keywords: Linguistics, Aging, Speech perseveration, Speech looping, Interbehaviorism, Applied behavior analysis, Gerontology, Neuropsychology

1. Introduction

Perseverative speech represents a clinically recognizable yet theoretically underdeveloped form of verbal repetition. It is defined by the *unintentional, verbatim re production of previously emitted utterances*, often with identical prosody, timing, and syntactic structure [1-3]. Unlike repetitive manding, anxiety driven reassurance seeking, or memory related question repetition, perseverative speech is *not socially mediated, not sensitive to correction, and not accompanied by metacognitive awareness* of having repeated oneself. The phenomenon is most commonly associated with neurological conditions involving executive dysfunction, but it is also observed in older adults without clear traumatic, degenerative pathology, medication misuse, or substance abuse [4-7]. Despite its prevalence across diagnostic categories, perseverative speech has remained conceptually marginalized, in part because it sits at the intersection of two explanatory traditions—behavior analysis and neurocognitive science—neither of which fully accounts for its defining features.

Across the last decade, the literature addressing repetitive verbal behavior in dementia and aging has remained largely descriptive, educational, or clinically advisory rather than empirical. The available sources—ranging from caregiver oriented guides to dementia support organizations—consistently acknowledge that older adults may exhibit repetitive speech, repeated questions, or perseverative verbal routines, yet none provide a formal behavioral analysis or experimental treatment model specific to verbatim speech perseveration. Instead, these materials frame

perseveration as a general symptom of cognitive decline and offer broad management strategies such as reassurance, distraction, environmental simplification, or emotional validation. While these recommendations are clinically intuitive, they lack mechanistic grounding and do not differentiate among the distinct forms of repetition that occur in dementia, such as memory driven repetition, anxiety driven repetition, and true *perseverative speech looping*.

Notably absent from the literature is any experimental research—single case designs, randomized trials, or controlled studies—testing interventions specifically aimed at reducing verbatim speech looping in older adults without traumatic brain injury. The few studies that address repetitive verbal behavior in dementia tend to focus on repeated questioning, agitation, or general behavioral disturbances, not the delayed, identical recurrence of a prior utterance that defines perseverative speech [8-10]. Moreover, no published work in the last ten years offers a behavior analytic functional analysis of perseverative speech as a distinct response class. Without such analysis, existing recommendations remain broad and nonspecific, making it difficult for clinicians to design interventions that target the underlying mechanisms rather than the surface behavior.

This absence of empirical models underscores the need for a mechanistically grounded framework capable of explaining and guiding treatment for speech perseveration in older adults. This paper first addresses limitations in the behavior analytic, interbehavioral, and neurocognitive domains. Second, it introduces an integrative or *unified (multi-stage) neurobehavioral model*. The integrative multi stage model fills this gap by conceptualizing perseverative speech as a cascade of control failures—including residual activation, stimulus control updating deficits, inhibition breakdowns, response selection failures, and impaired self monitoring. Unlike existing descriptive accounts, this model identifies *specific behavioral and neurocognitive processes* that can be targeted through structured environmental supports, competing response generation, paced speech routines, and externalized monitoring aids. In doing so, it provides the first evidence informed, theoretically coherent basis for developing behavioral interventions tailored to the unique features of perseverative speech. Third, discussion follows about the model's value for applied research and direct clinical implications for behaviorally treating speech perseveration.

2. Limitations of the Behavior Analytic Model

Behavior analytic models struggle because perseverative speech does not behave like an operant or respondent. It does not vary with attention, escape, deprivation, or extinction. It does not show the shaping effects of reinforcement history or higher order conditioning. And, it does not produce the autoclitic commentary expected when speakers detect socially inappropriate repetition. Attempts to classify it as automatically reinforced vocal stereotypy or repetitive manding fail to explain the delayed, verbatim, and motorically identical nature of the repetitions.

Traditional or radical behaviorism offers limited tools for analyzing this form of repetition. The standard functional analytic framework—attention, escape, tangible, and automatic reinforcement—presumes that verbal behavior is shaped and maintained by environmental contingencies [11]. However, perseverative speech often occurs independent of reinforcement history, independent of audience presence, and independent of antecedent manipulations. The behavior does not vary with attention, does not diminish with extinction, and does not increase under deprivation. Moreover, the absence of autoclitic behavior or self monitoring contradicts the expectation that speakers learn to avoid socially inappropriate repetition through community reinforcement. Radical behaviorism also lacks mechanisms for explaining why the repeated utterance is identical in prosody and timing, or why the repetition emerges after a short temporal delay rather than immediately. As a result, purely behavioral accounts tend to misclassify perseveration as stereotypy, repetitive manding, or automatically reinforced speech—none of which capture the defining features of the phenomenon. For example, Skinner described self-editing and autoclitic control as necessary for avoiding repetition [12]. But this presumed the speaker recognized ("had awareness of") looping repetitions. Similar presumptions inherently flawed the conclusion of verbal behavior becoming insensitive to consequences when autoclitics fail [13, 14].

Speech diminishment studied in autism research moved the needle closer to reasons why repeated utterances are identical in prosody, timing, and response topography. Fisher, Rodrigue, & Owen , for example, showed perseverative and echoic speech were *attention maintained* [13]. Differential reinforcement (DR) of non perseverative and on topic speech reduced perseveration, with on topic DR producing better conversational turn taking. Conversational flow replaced repetitive identical utterances. Similarly, Kuntz, Santos & Kennedy employed a treatment package of Differential Reinforcement of Alternative Behaviors (DRA) + extinction + prompting to decrease perseverative speech and increase appropriate verbalizations [15, 16]. But in each of these cases, as with the majority of ABA language-modification studies, emphasis is less on etiology and more on symptom elimination.

One exception was Hussian's premier attempt to frame geriatric problems in behavioral concepts [17]. Despite etiological analyses in more concrete and measurable terms, repetitive verbal behavior was attributed to cognitive decline in dementia. Hussian's definitions were broad, not functional, symptom-oriented, not mechanistic, and grouped with other repetitive behaviors (repeated questions, repetitive requests). He still improperly defined verbatim recurrences, prosodic identity, and delayed re-evocation of verbal behaviors in traditional neurocognitive explanations. In short, his description of perseveration did not differentiate it from other repetitive verbal behaviors or specify the control processes involved.

3. Limitations of the Interbehavioral Model

Kantor's interbehavioral psychology treats verbal behavior as an

event field; a dynamic interaction among organismic variables, stimulus functions, historical factors, and setting conditions [18,19]. In this view, speech is not a discrete response, but a *transactional* episode shaped by the entire interbehavioral field. Kantor's linguistic analysis emphasizes how verbal episodes emerge from contextual functions, organismic dispositions, and stimulus functions, rather than from reinforcement contingencies or internal cognitive modules [20,21]. Perseverative speech, therefore, reflects a failure of field transition where the organism-stimulus field does not re-organize to support a new verbal episode [16, 20, 22, 23].

Kantor also explicitly rejected internal cognitive or physiological explanations, arguing that such constructs reintroduce dualism and obscure the functional nature of behavior [24, 25]. The model proposed below, by contrast, integrates contemporary findings on working memory updating, executive inhibition, and error monitoring, treating these as organismic variables that participate in the interbehavioral field. This integration allows the new model to explain phenomena that Kantor could only describe at a contextual level—such as why the repeated verbal unit retains identical prosody, timing, and topography, or why the It integrates neurocognitive processes, which Kantor explicitly avoided. Kantor rejected physiological or cognitive "inner causes," whereas the integrative model uses neurocognitive constructs (e.g., working memory updating, inhibitory control) to explain why the interbehavioral field fails to shift to a new verbal episode

4. Limitations of the Neurocognitive Model

Neurocognitive models offer mechanistic constructs—such as inhibition failure, working memory decay, and set shifting deficits—but these accounts often remain too broad or abstract to guide functional assessment. They describe the cognitive architecture of perseveration but do not specify how internal failures translate into the *observable topography and temporal patterning* of the repeated utterance. As a result, perseverative speech remains theoretically orphaned and often rejected by behavior analysts studying verbal behavior [26-28]. Classic neuropsychological taxonomies distinguish recurrent, stuck in set, and continuous perseveration, attributing them to failures in inhibition failure, set shifting, or working memory decay, and frontal-striatal dysfunction [29-30].

Perseverative errors in verbal fluency tasks, for example, are typically symptomatic of Alzheimer's disease and are driven by executive function decline, especially inhibition and switching. Inhibition allegedly is the brain's ability to stop or suppress a response that is no longer appropriate. In dementia—especially Alzheimer's and frontotemporal disorders—the frontal lobes and frontal–subcortical circuits weaken. When inhibition fails, the brain cannot turn off the motor verbal program that produced the previous utterance [32]. Switching (or set-shifting) describes the ability to move from one idea to another and generate new responses instead of repeating old verbal responses. Effective transfer between ideas depends on the executive control networks in the dorsolateral prefrontal cortex, without which verbal behavior (a) stays locked into the previous topics, (b) cannot generate new responses, and (c) defaults to the last activated utterance [2].

Both inhibition and switching as constructs are scientifically limited when evaluated from a functional analytic standpoint. Both terms refer to *inferred internal processes*, not observable events, and are therefore too abstract to specify the environmental contingencies or response classes that actually govern behavior. As Stuss and Alexander noted, inhibition and set shifting are *performance level inferences* drawn from task outcomes rather than discrete, measurable mechanisms [33]. Their broadness further reduces analytic precision: "inhibition" is used to explain everything from stopping a motor act to suppressing a thought, while "switching" collapses topic shifts, task shifts, and response set changes into a single label [34]. Because these constructs do not identify the antecedent conditions, competing responses, or stimulus control dynamics involved in conversational behavior, they cannot predict when perseveration will occur or how it can be altered. Even comprehensive neurocognitive reviews acknowledge that executive function deficits correlate with perseveration but do not specify the functional relations needed for intervention [29,32]. As a result, inhibition and switching function largely as descriptive summaries of impaired performance rather than precise scientific tools. A functional analysis requires finer grained, observable mechanisms—such as residual activation, stimulus control updating, response selection competition, and monitoring failures—that can be manipulated directly in treatment.

5. Unified Neurobehavioral Analytical Model

To address these limitations, we propose a multi stage *Neurobehavioral Analytical Model (NAM)* that synthesizes the strengths of both traditions. This model conceptualizes perseverative speech as a *stepwise failure of verbal control*. It begins with the creation of a strongly activated motor–verbal response unit and culminates in its unintended re evocation when competing responses fail to reach threshold activation. The framework integrates behavior analytic concepts such as stimulus control, response competition, and residual response strength with neurocognitive mechanisms involving inhibition, working memory updating, and error monitoring. By mapping each stage of the failure process onto both observable behavior and known neural systems, the model provides a coherent explanation for the verbatim nature, delayed recurrence, and consequence insensitive profile of perseverative speech. It also offers a functional vocabulary that is compatible with both behavioral assessment and neuropsychological testing, bridging a longstanding conceptual divide.

The NAM conceptualizes perseverative speech as a progressive, multi stage failure of verbal control across seven interconnected control processes. In *Stage 1*, an initial verbal response is generated, forming a strongly activated motor–verbal program. *Stage 2* reflects the slow decay of this activation, leaving the response available for re evocation. *Stage 3* involves a failure of stimulus control updating,

in which the original controlling stimulus does not deactivate and new environmental cues fail to evoke alternative responses. *Stage 4* represents an inhibition failure, preventing suppression of the previously activated motor–verbal program. In *Stage 5*, response selection breaks down as competing verbal responses fail to reach activation threshold, causing the system to default to the last strongly activated unit. *Stage 6* captures the verbatim re evocation of the prior motor–verbal program, producing a repeated utterance with identical wording, prosody, and timing. *Stage 7* reflects impaired self monitoring, preventing detection or correction of the repetition. Together, these stages provide a mechanistic account of why perseverative speech is delayed, verbatim, consequence insensitive, and unaccompanied by metacognitive awareness. This stepwise model explains the *verbatim nature, identical intonation, short interval recurrence, and independence from reinforcement* that define perseverative speech (see Table 1).

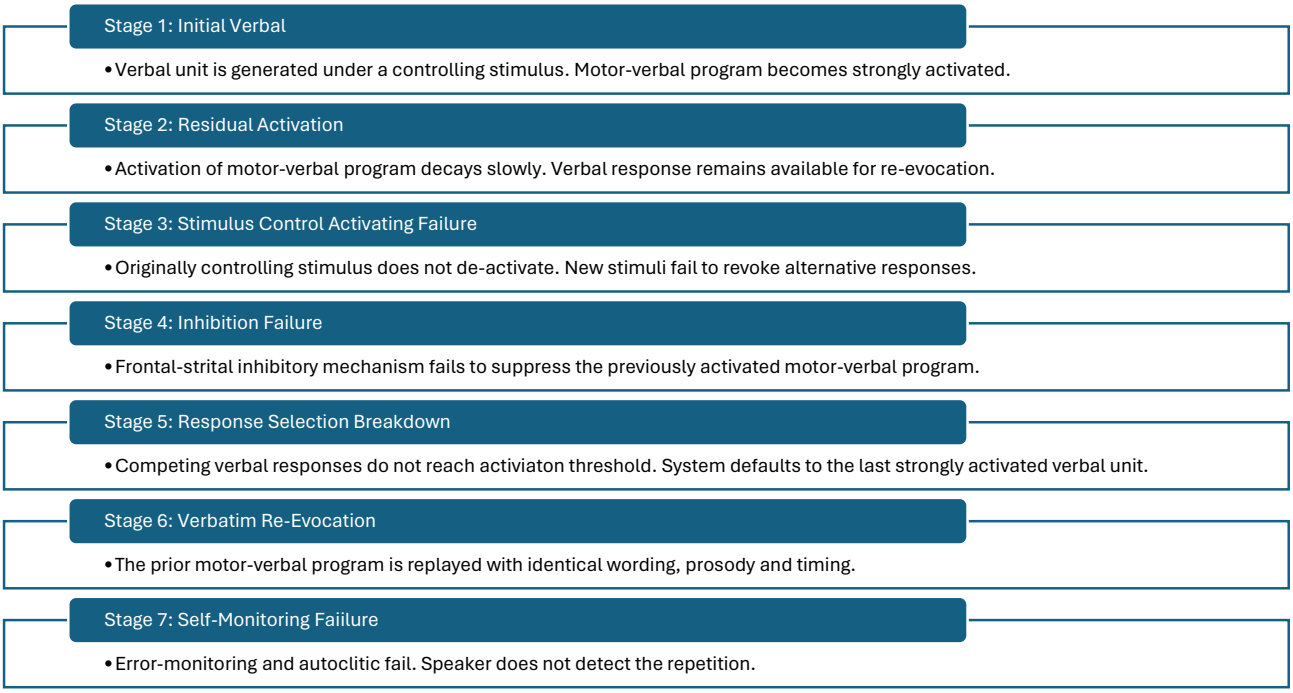

Table 1: Schematic description of the proposed multi stage model of perseverative speech. The diagram illustrates how residual activation, failures in stimulus control updating and inhibition, breakdowns in response selection, and impaired self monitoring interact to produce the delayed, verbatim recurrence of a previously emitted utterance.

Transactionally, elderly pass through a series of *setup conditions* under which a verbal response becomes unusually likely to recur. These early stages involve the creation of a strongly activated motor–verbal program, the slow decay of that activation due to reduced inhibitory tone, and a failure of stimulus control updating that prevents new environmental cues from evoking alternative responses. At this point, the prior utterance remains disproportionately accessible, while competing responses remain weakly activated. Behaviorally, this appears as subtle "stickiness," slowed topic transitions, or difficulty generating new content. Neurologically, these stages reflect inefficient working memory updating, persistent activation in frontal–striatal circuits, and reduced flexibility in prefrontal set shifting systems. Tables 2 and 3 more clearly illustrate how perseverative speech emerges not from a single deficit but from a cascading interaction of activation dynamics, stimulus control failures, inhibitory breakdowns, and monitoring impairments. Stages 1–3 create the conditions under which a prior utterance remains abnormally available, while Stages 4–7 determine how that availability translates into an observable, consequence insensitive speech loop. This linkage underscores the model's central claim: Perseverative speech is best understood as a multi level control failure, rather than an operant behavior or a unitary executive function deficit.

Multi Stage Behavioral–Neurocognitive Model of Perseverative Speech, Stages 1-3

Domain	1. Initial Verbal Response	2. Residual Activation	3. Stimulus Control Updating Failure
Behavioral Output	A coherent verbal response is produced; speech appears normal and context appropriate.	Speaker transitions away from the initial utterance, but the prior response remains subtly "ready" for reuse.	New conversational turns fail to take hold; speaker shows difficulty shifting topics or generating new content.
Stimulus Control	Response is governed by a clear controlling stimulus (question, prompt, conversational cue).	Original stimulus begins to fade but does not fully deactivate; weak competing stimuli emerge.	Original stimulus continues to exert disproportionate control; new environmental cues fail to evoke alternative responses.
Response Dynamics	Motor–verbal program is encoded as a unified sequence; strong activation trace is established.	Activation decays slowly, leaving the prior response highly accessible relative to new responses.	Response competition becomes imbalanced; alternative responses fail to gain sufficient activation to replace the prior one.
Neurocognitive Mechanisms	Left inferior frontal gyrus, supplemental motor area, and basal ganglia encode the motor–verbal sequence; working memory holds the utterance online.	Slow decay of activation reflects reduced inhibitory tone and inefficient working memory updating.	Dorsolateral prefrontal cortex and parietal networks fail to shift cognitive sets; frontal–striatal loops remain locked onto the prior response.
Speech Characteristics	Fluent, context appropriate utterance with normal prosody and timing.	Subtle signs of "stickiness" may appear—slowed transitions, reduced generativity, or lingering phrasing.	Speech becomes rigid or non progressive; attempts at topic shift may stall or revert toward the prior utterance.

Table 2: Behavioral and neurocognitive characteristics of the first three stages of the proposed multi stage model of perseverative speech. The table outlines how initial response encoding, residual activation, and failures in stimulus control updating influence observable speech patterns, response dynamics, and underlying neural mechanisms. These early stages describe how a verbal response becomes strongly activated, remains accessible due to slow decay, and persists as a dominant response when new stimuli fail to shift control.

Multi Stage Behavioral–Neurocognitive Model of Perseverative Speech, Stages 4-7

Domain	4. Inhibition Failure	5. Response Selection Breakdown	6. Verbatim Re-evocation	7. Self-M Failure onitoring
Behavioral Output	Previously emitted phrase remains available; speaker shows difficulty suppressing recently used verbal units.	No new utterance gains enough activation to compete; speech becomes "stalled" or non generative.	Prior utterance is reproduced with identical wording, prosody, and timing.	Speaker does not comment on or correct the repetition; no autoclitic markers appear.
Stimulus Control	Original controlling stimulus continues to exert influence despite context change.	Environmental cues fail to evoke alternative responses; topic shifts do not occur.	Speech is driven by residual activation rather than current stimuli.	Speaker does not detect mismatch between current context and repeated utterance.
Response Dynamics	Suppression of prior motor–verbal program is impaired; response remains primed.	Competing responses fail to reach threshold activation; response field collapses to a single dominant option.	Motor–verbal program is replayed as a unit, producing a verbatim loop.	No competing response is generated to override or interrupt the loop.

Neuro-cognitive Mechanism	Frontal–striatal inhibitory circuits underperform; reduced gating of prior responses.	Dorsolateral prefrontal cortex fails to activate new response sets; weak top down selection.	Supplemental Motor Area and left inferior frontal gyrus re activate the previously encoded motor sequence.	Anterior cingulate cortex fails to signal error or novelty; monitoring is blunted.
Speech Characteristics	Subtle hesitations or "stuckness" before repetition; difficulty initiating new content.	Speech becomes sparse or rigid; pauses precede the recurrence.	Utterance is reproduced exactly, including rhythm, pitch contour, and timing.	Speaker continues conversation as if no repetition occurred; no awareness of looping.

Table 3: Behavioral and neurocognitive characteristics of the later stages of the proposed multi stage model of perseverative speech. The table details how inhibition failure, response selection breakdown, verbatim re evocation, and impaired self monitoring interact to produce the delayed, identical recurrence of a prior utterance. These stages account for the consequence insensitive, topographically invariant, and unrecognized nature of perseverative speech.

Consider this clinical vignette. Mrs. L, a 67 year old woman with mild Alzheimer's disease, is riding in the passenger seat while her adult son drives her to a doctor's appointment. As they pull out of the driveway, Mrs. L looks back toward the house and asks, "Did you turn off the stove before we left?" Her son reassures her: "Yes, Mom, I checked it." A few minutes passed. The car radio plays softly, and traffic moves steadily. Despite these new environmental cues, Mrs. L's attention remains anchored to the earlier thought. The internal activation of the "stove" utterance does not fade. She glances back toward the house again, even though it is no longer visible. Her son begins talking about the appointment, but she does not shift with him. Instead, she quietly repeats, "Did you turn off the stove before we left?" He answers again, gently. She nods but does not appear fully reassured. Several minutes later, with no new trigger and no sign of anxiety, she repeats the question a third time—same words, same tone, same rhythm.

This example does not present repeated questioning under motivating operators of fear or memory loss. It is *true perseverative speech*: a delayed, involuntary, identical recurrence of a prior utterance caused by *residual activation, stimulus control updating failure, inhibition breakdown, and response selection failure, culminating in monitoring failure and perseverative output*. Table 4 illustrates how each stage of the seven stage behavioral–neurocognitive model of perseverative speech corresponds to specific, observable behaviors in a 67 year old woman with Alzheimer's disease. The table demonstrates the progression from an initially appropriate utterance through residual activation, failures in stimulus control updating, inhibition, response selection, and monitoring, culminating in a delayed, identical recurrence of the original utterance. The mapping highlights how internal control process breakdowns manifest as real world conversational perseveration, distinguishing true perseverative speech from repeated questioning or anxiety driven repetition.

Seven Stages Mapped to Observable Behavior in the Vignette

Stage	Guided Link	What Happens Internally	Observable Behavior in Mrs. L
1. Initial Utterance	Initial utterance	Normal activation of a verbal response	Asks appropriately: "Did you turn off the stove before we left?"
2. Residual Activation	Residual activation	Motor verbal program stays active too long	Continues thinking about the stove even after reassurance
3. Stimulus Control Updating Failure	Stimulus control updating	New cues fail to shift attention	Does not shift to radio, conversation, or car environment
4. Inhibition Failure	Inhibition failure	Cannot suppress the now irrelevant response	Repeats the question despite knowing she asked it already
5. Response Selection Breakdown	Response selection	Competing responses cannot be generated	Cannot move to new topics; old utterance "wins"
6. Monitoring Failure	Monitoring failure	Cannot detect that she already said it	Shows no awareness of repetition; no self correction
7. Perseverative Output	Perseverative output	Old motor verbal program re fires identically	Repeats: "Did you turn off the stove before we left?" with same prosody and timing

Table 4: Summarizes how each stage of the seven stage behavioral–neurocognitive model corresponds to specific observable behaviors in an older adult exhibiting true perseverative speech.

6. Implications for Applied Research and Clinical Treatment

A key contribution of the NAM is its ability to explain why repeated utterances are *identical in prosody and timing*, a feature that neither behavioral nor neurocognitive accounts have adequately addressed. By framing the repetition as the *re-evocation of a previously activated motor–verbal program*, the model provides a mechanistic explanation for the precision of the recurrence. This perspective also clarifies why repetition often emerges after a short delay rather than immediately: the system defaults to the last strongly activated response only when *no competing verbal response achieves sufficient activation*. This default to prior mechanism aligns with known properties of frontal–striatal circuits while remaining fully compatible with behavior analytic concepts such as residual response strength and response competition. In this way, the model bridges the conceptual divide between the two disciplines, offering a framework that is both mechanistically grounded and behaviorally interpretable.

The model also reframes perseverative speech as a phenomenon that is *not maintained by reinforcement, but rather one that emerges from control failures distributed across multiple cognitive and behavioral systems*. This distinction has important implications for assessment and treatment. Functional analyses that rely on manipulating consequences are unlikely to reveal meaningful control variables, whereas assessments that examine stimulus control, competing response availability, and cognitive load may be more informative. Likewise, neuropsychological assessments that focus solely on global executive function scores may overlook the specific interaction of processes—activation, inhibition, updating, and monitoring—that jointly produce the behavior. By specifying these components, the model provides a roadmap for more targeted assessment and treatment strategies that can identify which stage(s) of the control chain are most impaired in a given individual.

From an applied treatment perspective, the model generates a range of testable predictions. For example, increasing the salience of competing verbal stimuli should reduce the likelihood of perseveration, whereas increasing cognitive load or reducing environmental structure should increase perseverative speech. Experimental manipulations can be designed to isolate each stage of the model: tasks that tax inhibition may increase recurrence frequency; tasks that manipulate stimulus control updating may alter the timing of recurrence; and tasks that enhance self monitoring may reduce the probability of uncorrected repetition. These predictions create opportunities for translational research that links laboratory based executive function paradigms with real world verbal behavior, enabling more precise characterization of perseverative speech across clinical populations.

An example of a behavioral treatment under the NAM model consists of designing procedures that change activation, cue control, and response competition, rather than trying to extinguish the speech repetition [35]. Stage-linked behavioral procedures follow seven steps:

Steps 1 and 2: Use *stimulus fading* in reverse, similar to similar fading, prompt fading, and backward chaining [36, 37]. Fade the response by pacing and constraining it. That way, its activation decays more quickly. The trainer paces the conversation or deliberately pauses before the next question or topic. This combines with topic anchoring. Use written or pictorial topic cards. When a new topic starts, physically shift to a new card. Guiding to new stimuli replaces corrective feedback of "You already said that."

Step 3: Introduce new stimuli to exert control over the old cue. This is identical to programming discriminative stimuli for topic shifts, like teaching multiple components [38, 39]. Here, discrimination training is for conversation says, "Now we're going to talk about ___" plus show a card or object tied to the new topic [40]. After briefly discussing the topic, use explicit closure signals such as "We're done talking about X," paired with a consistent gesture (closing a folder, turning a card, changing the stimuli). Pairing objects or gestures with prompts to a new subject both serves a discriminative stimulus (establishing operation) for new speech behavior and simultaneously shapes or reinforces completion (not repetition) of the last statement made by the elderly (i.e., chaining or errorless learning; [41]).

Steps 4 and 5: The goal is inhibition failure and response selection-breakdown. This procedure is analogous to response-class expansion and prompting alternative responses. In this way, the trainer builds a competing response class [42, 43]. Trainer first guides choices for responding, "Do you want to talk about your garden or grandkids?" Trainer then offers scripted options for common situations. For example, "When someone asks how you are, you can say, I'm okay, or just taking it day by day.'" Then, trainers prompt generativity. Trainer uses sentence stems to activate new content, such as, "What is one thing you remember about today?"

Step 6: The process is decreasing verbatim re-evocation by interrupting the speech loop without punishment or argument. This soft pattern-break involves a new concrete cue prompting other verbal behavior. First, the trainer gently interrupts the repeated verbal pattern (e.g., places a hand on the table, points to a topic card) right as the repetition starts. Second, trainers use a tap, metronome, or slow-speech prompt to delay initiation of the verbatim response and allow for alternative response(s) to activate. Third, trainers use embedded direction: "You just said that-let's add something new," followed immediately by a concrete prompt ("what else happened in the situation?").

Step 7. This last step corrects self-monitoring failures. This is functionally analogous to research demonstrating applications of elderly who develop or refine self-recording, error correction in speech and communication, memory-based self-monitoring, and metacognitive prompting [44, 45]. Externalized monitoring, at first, replaces the elderly relying on impaired internal systems of self-monitoring. Here are the subtests. First, using external monitoring aids, the trainer puts a simple card on the table and

points to it, such as "We already talked about this." Collaborative reframing follows, "We've covered that-let's move to the next card together." Second, for cognitively higher functioning elders, conversation logs are helpful, such as "Today we talked about (1) the doctor's appointment, (2) lunch with your sister, (3) grandkids' birthday, and (4) physical pains in your shoulder. Third, practice script of conversation logs builds awareness of verbal statements the elderly recently engaged in. This "awareness" is a repertoire-altering effect that also teaches basic autoclitics (tacts, mands) to later self-monitor and self-stop re-evocation of verbatim speech responses [44, 46-51]. In later occurrences, the elder might precede or self-interrupt a verbatim statement with, "I think I told you this, but," or "did I say this already?" Table 5 summarizes the sequential steps (1-7) of training.

Behavioral Treatment Protocol for Speech Perseveration Based on the Integrative Multi Stage Model

Step	Primary goal	Key procedures	Example clinician/caregiver behaviors
1. Functional–mechanistic assessment	Identify whether speech fits perseveration and which stages are most impaired.	Observe conversations; note delay, verbatim quality, response to correction, triggers; map to stages (activation, updating, inhibition, monitoring).	"I'll watch for when the repetition happens, how similar it is, and whether it changes when people respond differently."
2. Caregiver orientation and reframing	Reduce blame and ineffective correction; align expectations with the model.	Explain control failure model; emphasize that scolding/ arguing is unhelpful; agree on neutral cues and language.	"When she repeats, instead of saying 'You already said that,' we'll gently guide her to something new."
3. Structure conversational environment	Reduce residual activation and strengthen topic shift cues (Stages 1–3).	Use topic cards; clear "start" and "end" phrases; move/flip cards; slow pacing; limit long, open ended questioning.	"Now we're talking about your garden" (points to card). "We're done with your garden now" (flips card, pauses).
4. Build competing responses	Provide easy alternative responses to outcompete the prior utterance (Stages 4–5).	Create short scripts; use choice prompts; use sentence stems; rehearse during calm times.	"When I ask how you are, you can say, 'I'm okay, just taking it day by day.' Let's practice that together."
5. Pattern interruption and redirection	Gently interrupt loops and shift to new or expanded content (Stage 6).	Neutral interrupt cue (tap, gesture); collaborative phrase; immediate prompt for new detail or new topic; avoid confrontation.	(Loop starts) tap "You just said that—let's add something new. What else happened?" (or shifts to new topic card).
6. Externalized monitoring supports	Compensate for impaired self monitoring with simple external cues (Stage 7).	Use "we already talked about this" card or symbol; caregiver taps/shows it; guide to new topic/script; optional visible list of topics covered.	"We've already talked about the doctor today" (points to list), "Let's look at what's next—lunch."
7. Ongoing evaluation and adjustment	Monitor effectiveness and refine the plan.	Track loop frequency, repetitions per loop, caregiver distress; identify most helpful supports; adjust complexity and structure.	"Since we added topic cards, the loops are shorter. Let's keep those and add more practice with scripts."

Table 5: Outlines each intervention step, its clinical goal, the specific procedures used, and examples of caregiver or clinician behaviors. The protocol emphasizes environmental structuring, strengthened stimulus control, competing response generation, gentle pattern interruption, and externalized monitoring supports—reflecting the model's focus on activation dynamics, updating failures, inhibition breakdowns, and impaired self monitoring rather than operant reinforcement contingencies.

7. Discussion

The present model offers a unified account of persevering speech by integrating behavioral principles with neurocognitive mechanisms and addressing longstanding gaps in both explanatory traditions. Traditional behavior analytic frameworks have struggled to characterize perseverative speech because the behavior does not conform to operant principles. It is insensitive to consequences, unaffected by extinction, and topographically invariant across repetitions. Attempts to classify it as automatically reinforced vocal stereotypy or repetitive manding fail to explain its delayed recurrence, verbatim reproduction, and absence of autoclitic self monitoring. Neurocognitive models, while rich in mechanistic constructs such as inhibition failure and working memory decay, have historically lacked the behavioral specificity needed to map internal deficits onto the observable temporal and structural properties of the repeated utterance. The integrative model proposed here resolves these limitations by conceptualizing perseverative speech as a multi stage failure of verbal control. Seven procedural steps implement residual activation, stimulus control updating failures, interruption of inhibitory deficits, and repair of impaired self monitoring to prevent repetitive speech looping patterns.

Overall, the integrative model provides a coherent, mechanistically grounded, and behaviorally precise account of perseverative speech that resolves longstanding limitations in both behavior analytic and neurocognitive explanations. By articulating the specific stages through which verbal control fails, the model offers a foundation for more targeted assessment, more effective intervention, and more rigorous empirical investigation. It positions perseverative speech not as an anomaly that falls between disciplines, but as a phenomenon that can be understood only through the combined explanatory power of both. This synthesis opens the door to a new generation of applied research and clinical practice that treats perseveration not as an intractable byproduct of executive dysfunction, but as a systematic, analyzable, and modifiable behavior pattern.

8. Future Directions

The absence of empirically tested interventions for speech perseveration in older adults highlights an immediate need for systematic, mechanism driven research. The integrative multi stage model offers a natural scaffold for such work by specifying discrete control processes—residual activation, stimulus control updating, inhibition and response selection, and self monitoring—that can be manipulated experimentally. Future studies should move beyond global descriptions of "repetitive speech" and instead design targeted tests of specific stages in the model, using both behavioral and neurocognitive measures.

A first line of research could employ single case experimental designs (e.g., multiple baseline across participants, settings, or caregivers) to evaluate the treatment protocol derived from the model. For example, researchers could sequentially introduce components such as topic cards (stimulus control), scripted alternatives (competing responses), and neutral pattern interruption cues (Stage 6) while measuring changes in (a) frequency of perseverative loops, (b) number of repetitions per loop, and (c) caregiver rated distress. Component analyses could identify which procedures are necessary and sufficient for clinically meaningful change, and whether different individuals benefit from different stage targeted strategies.

A second line of work should focus on laboratory style analogue tasks that isolate specific mechanisms. For instance, tasks that manipulate cognitive load and topic shift salience could test predictions about residual activation and stimulus control updating (Stages 1–3), while Go/No Go or stop signal paradigms embedded in verbal tasks could probe inhibition and response selection (Stages 4–5). By pairing these tasks with naturalistic conversation samples, researchers could examine whether performance on specific executive function measures predicts the severity or pattern of perseverative speech, thereby validating the model's proposed control processes.

Third, future research should explore population heterogeneity within older adults. Comparative studies could examine whether individuals with Alzheimer's disease, vascular dementia, frontotemporal dementia, or mild cognitive impairment show distinct profiles of stage impairment (e.g., more pronounced updating failures vs. monitoring deficits). Such work could support a more nuanced taxonomy of perseverative phenomena and inform personalized intervention planning, where treatment packages are tailored to the dominant failure points in the control chain.

Fourth, there is a need for caregiver focused intervention studies that evaluate not only changes in perseverative speech but also caregiver burden, communication satisfaction, and adherence. Randomized or quasi experimental trials could compare the integrative, stage based protocol to usual care or generic communication training. Outcomes might include reductions in perceived frustration, improvements in perceived communication quality, and feasibility of implementing environmental and conversational supports in home and long term care settings.

Finally, future work should consider multimodal assessment, integrating behavioral observation, caregiver report, and, where feasible, neuroimaging or electrophysiological measures. Although the model is designed to be clinically useful without advanced technology, converging evidence from neural markers of inhibition, monitoring, and set shifting would strengthen its validity and may reveal biomarkers of treatment response. Over time, such research could support the development of evidence based clinical guidelines for managing speech perseveration in older adults. Progress in applied and clinical research might transform the current landscape from descriptive advice to mechanistically informed, empirically supported practice.

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