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NETWORK MODELING OF BROCA'S MOTOR APHASIA

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Summary. Lobasyuk B. A., Stoyanov A. N. **NETWORK MODELING OF BROCA'S MOTOR APHASIA. –** *The Odessa I. I. Mechnikov National University; The Odessa National Medical University; e-mail: blobasuk@gmail.com.* **Relevance.** Contemporary neuroscience approaches interpret aphasia as a disruption of dynamic network interactions between cortical and subcortical structures. In recent years, motor (Broca's) and sensory (Wernicke's) aphasias have increasingly been conceptualized as systemic disorders arising from failures in the connectivity of speech networks. However, a network-based model of motor aphasia symptoms has not yet been sufficiently described. **Objective.** To determine the probable mutual influences among the main symptoms of motor aphasia using multiple regression and correlation analysis, and to construct a network model of this speech disorder. **Materials and Methods.** At the stroke unit of Odesa Regional Hospital, from January 1, 2024, to May 12, 2025, 25 patients aged 40 to 60 years with aphasia, predominantly due to acute isolated ischemic stroke of the middle cerebral artery, were examined. The matrices of the initial data were constructed by coding the presence of a symptom as 1 and the absence of a symptom as 0. Twenty-five patients with motor aphasia symptoms were examined. In statistical analysis, binary matrices were constructed where symptom presence was coded as 1 and absence as 0. Each symptom was sequentially treated as a dependent variable (Y), with the others as predictors (X). Multiple linear regression was applied to determine directed mutual influences. Results were geometrically interpreted through polycyclic multigraphs. **Results. Anomia and oral comprehension:** bidirectional negative associations were found, with anomia exerting a stronger effect on comprehension than the reverse. **Anomia and speech motor production:** positive bidirectional influences were established, with motor impairments impacting anomia more strongly. **Paraphasia:** interacted with speech production and articulatory deficits through bidirectional negative links. Production difficulties had a stronger effect on paraphasia, while paraphasia more strongly influenced articulation. **Neologisms and comprehension:** showed bidirectional negative connections dominated by pathological neologisms. Thus, each group of symptoms formed a network in which the strength and direction of links reflected both pathogenetic mechanisms and potential compensatory strategies. **Conclusions. Network organization.** Symptoms of motor aphasia constitute an interconnected system uniting lexico-semantic, phonological, and motor components. **Central role of anomia.** Anomia exerts a key influence on speech comprehension and interacts with motor deficits, confirming its function as a lexical access hub. **Asymmetry of connections.** Negative bidirectional links (e.g., anomia ↔ comprehension) reflect the functional segregation of dorsal and ventral streams, while positive connections (anomia ↔ motor processes) point to shared dorsal nodes. **Motor–phonological loop.**

The strong impact of motor processes on anomia suggests that articulation may support lexical access. **Clinical significance.** The character and direction of links help specify lesion localization (e.g., left fronto-temporo-parietal overlap) and predict recovery strategies. **Methodological contribution.** The use of multiple regression and polycyclic multigraphs demonstrates the feasibility of describing aphasia as a systemic disorder rather than a set of isolated symptoms. Motor aphasia results from complex network interactions, with anomia as the central node and symptom dynamics determined by the balance between dorsal and ventral streams of speech processing. This approach opens new perspectives for diagnosis and individualized rehabilitation.

Key words: motor aphasia, anomia, paraphasia, network model, asymmetry of influences.

Реферат. Лобасюк Б. А., Стоянов О. М. **МЕРЕЖЕВЕ МОДЕЛЮВАННЯ МОТОРНОЇ АФАЗІЇ БРОКА. Актуальність.** Сучасні нейронаукові підходи інтерпретують афазію як порушення динамічних мережевих взаємодій між корковими та підкірковими структурами. В останні роки моторна (Брока) та сенсорна (Верніке) афазії все частіше концептуалізуються як системні розлади, що виникають внаслідок збоїв у зв'язності мовленнєвих мереж. Однак мережева модель симптомів моторної афазії ще не була достатньо описана. **Мета.** Визначити ймовірний взаємний вплив основних симптомів моторної афазії за допомогою множинного регресійного та кореляційного аналізу, а також побудувати мережеву модель цього мовленнєвого розладу. **Матеріали та методи.** У відділенні інсульту Одеської обласної лікарні з 1 січня 2024 року по 12 травня 2025 року було обстежено 25 пацієнтів віком від 40 до 60 років з афазією, переважно спричиненою гострим ізольованим ішемічним інсультом середньої мозкової артерії. Матриці вихідних даних були побудовані шляхом кодування наявності симптому як 1 та відсутності симптому як 0. Було обстежено двадцять п'ять пацієнтів із симптомами моторної афазії. У статистичному аналізі були побудовані бінарні матриці, де наявність симптому кодувалася як 1, а відсутність - як 0. Кожен симптом послідовно розглядався як залежна змінна (Y), а інші - як предиктори (X). Для визначення спрямованого взаємного впливу було застосовано множинну лінійну регресію. Результати були геометрично інтерпретовані за допомогою поліциклічних мультиграфів. Результати. Аномія та розуміння усного мовлення: виявлено двонаправлені негативні асоціації, причому аномія мала сильніший вплив на розуміння, ніж зворотний. Аномія та мовленнєва моторика: було встановлено позитивний двонаправлений вплив, причому моторні порушення сильніше впливали на аномію. Парафазія: взаємодіяла з мовленнєвою продукцією та артикуляційними дефіцитами через двонаправлені негативні зв'язки. Труднощі з мовленням сильніше впливали на парафазію, тоді як парафазія сильніше впливала на артикуляцію. Неологізми та розуміння: показали двонаправлені негативні зв'язки, в яких домінували патологічні неологізми. Таким чином, кожна група симптомів утворювала мережу, в якій сила та напрямок зв'язків відображали як патогенетичні механізми, так і потенційні компенсаторні стратегії. Висновки. 1. Організація мережі. Симптоми моторної афазії являють собою взаємопов'язану систему, що об'єднує лексико-семантичні, фонологічні та моторні компоненти. 2. Центральна роль аномії. Аномія має ключовий вплив на розуміння мовлення та взаємодіє з моторними дефіцитами, підтверджуючи свою функцію як центру лексичного доступу. 3. Асиметрія зв'язків. Негативні двонаправлені зв'язки (наприклад, аномія ↔ розуміння) відображають функціональну сегрегацію дорсального та вентрального потоків, тоді як позитивні зв'язки (аномія ↔ рухові процеси) вказують на спільні дорсальні вузли. 4. Моторно-фонологічна петля. Сильний вплив рухових процесів на аномію свідчить про те, що артикуляція може підтримувати лексичний доступ. 5. Клінічне значення. Характер і напрямок зв'язків допомагають визначити локалізацію ураження (наприклад, перекриття лівих лобно-скронево-тім'яних ділянок) та передбачити стратегії відновлення. 6. Методологічний внесок. Використання множинної регресії та поліциклічних мультиграфів демонструє можливість опису афазії як системного розладу, а не як набору ізольованих симптомів. **Висновок.** Моторна афазія є результатом складних мережевих взаємодій, де аномія є центральним вузлом, а динаміка симптомів визначається балансом між дорсальним та вентральним потоками обробки мовлення. Цей підхід відкриває нові перспективи для діагностики вказаної патології.

Ключові слова: моторна афазія, аномія, парафазія, мережева модель, асиметрія впливів.

Classical neuropsychology traditionally linked motor and sensory aphasias mainly to lesions in the Broca's and Wernicke's areas. In contrast, contemporary neuroscience approaches consider aphasia as the result of disrupted dynamic network interactions between various cortical and subcortical regions (Mesulam et al., 2015; Binder, 2015).

Motor aphasia (Broca's aphasia) is a classical form of aphasia described by Paul Broca in the 19th century. It arises from damage to the posterior part of the inferior frontal gyrus (Broca's area, Brodmann areas 44 and 45), predominantly in the left hemisphere of right-handed individuals (Broca, 1861; Ardila, 2010).

The Network Model of Broca's Motor Aphasia

The network model represents a modern direction in aphasiology and cognitive neuroscience. It interprets speech disorders not as the isolated impairment of "Broca's center" (a classical localizationist concept of the 19th–20th centuries), but as a consequence of dysfunction within a distributed speech network of the brain (Catani &Mesulam, 2008).

Core Ideas of the Network Model. Distributed speech network:Broca's aphasia is associated not only with Broca's area (posterior inferior frontal gyrus, BA 44/45) but also with a broader network involving premotor and supplementary motor areas, dorsolateral prefrontal cortex, insular cortex, subcortical structures (basal ganglia, thalamus), and white matter tracts (arcuate fasciculus, superior longitudinal fasciculus).

Mechanisms of speech disruption: The disorder arises from disconnections between semantic, lexical, and motor-articulatory nodes. This explains symptoms such as anomia (word-finding difficulties), agrammatism (deficit of syntactic structures), and non-fluent speech (slow, fragmented output) (Goodglass, 1993).

Network reorganization after damage: Following stroke or trauma, partial network reorganization occurs — the right hemisphere and preserved left-hemisphere regions compensate functionally. For this reason, modern aphasia models employ polycyclic multigraphs and methods of network analysis (graph theory, DTI, fMRI, EEG/ECoG).

Relation to the "Dual Stream" Model of Speech. Broca's aphasia is viewed as the result of disruption in the dorsal stream (articulatory–phonological pathway) that links parietal and temporal regions to motor speech centers. In contrast, the ventral semantic stream is often relatively preserved (Friederici, 2011).

Thus, the network model interprets Broca's aphasia not as a local lesion, but as a disconnection syndrome involving reorganization of a complex functional speech network.

Pulvermüller's Contribution: Neural Word Ensembles and Action–Perception Circuits

Pulvermüller (1999) proposed the concept of neuronal word ensembles, where each word corresponds to a distributed cortical network linking sensory, motor, and associative zones. Lexical and semantic representations are not localized to a single "center," but are distributed across the brain.

Later, Pulvermüller (2005) introduced the concept of action–perception circuits (APCs): language processes are tightly linked to motor systems. According to this model, speech acts emerge from distributed networks connecting motor areas (speech production) and auditory regions (perception), forming a functional unit for pronouncing words.

Example: perception of the word *"run"* activates the leg motor cortex.

This supports the hypothesis that language is embedded in sensorimotor systems, rather than isolated from them.

In later work, Pulvermüller (2018) expanded the APC theory, emphasizing that language exploits re-usable sensorimotor networks for understanding words and sentences. These networks form through learning and experience, enabling the linking of words to actions and objects.

Advances in Network-Based Aphasia Research

Network Control Theory (NCT):Wilmskoetter et al. (2022) examined how structural white matter connections influence speech recovery after stroke. They found that high controllability of frontal language regions predicted better speech recovery after 6 months, marking an important

step toward personalized rehabilitation forecasts.

Network-Based Statistics (NBS): Zhao et al. (2023) compared Broca's aphasia with anomic aphasia. Patients with Broca's aphasia showed weaker connections in premotor, motor, auditory, and sensory regions, while anomic aphasia involved fewer disruptions in different networks. NBS thus provides an objective tool for differentiating aphasia types.

Boes et al. (2015), using the method of lesion network mapping (LNM), in which damaged brain regions are "overlaid" on rs-fMRI data from healthy individuals, were the first to systematically demonstrate that the same symptom (e.g., hallucinations, aphasia) can arise from lesions in different locations if these regions belong to a common network. **Conclusion:** symptoms are better explained by networks rather than by isolated foci.

Hickok &Poeppel (2004) proposed the dual-stream model of speech: the **dorsal stream**subserves sensorimotor transformation (repetition, articulation), while the **ventral stream** underlies semantic comprehension. Sensory aphasia is interpreted as a disruption of the ventral stream (posterior temporal areas and their connections).

According to Mesulam et al. (2015), the classical "Wernicke's area" is not a single point, but rather a network: posterior superior temporal cortex, middle temporal cortex, and angular gyrus. Comprehension depends on the integration of these nodes.

Binder (2015) also considers "Wernicke's area" as a broader network, emphasizing the role of the middle temporal cortex and the angular gyrus in speech comprehension, rather than only the posterior superior temporal gyrus.

In a large-scale review including studies of more than 800 patients with aphasia, speech comprehension deficits were shown to be associated with lesions of the posterior middle temporal cortex and its connections. This supports the network-based approach (Fridriksson et al., 2018).

In the classical study of **transcortical sensory aphasia (TSA)** by Boatman et al. (2000), TSA was explained as a disconnection between the ventral stream and semantic regions, while sparing the arcuate fasciculus (hence the preserved repetition). A similar interpretation is provided by Shekari&Nozari (2023).

Reviews of the role of white matter in language functions have shown that sensory aphasia is associated with lesions of the **inferior longitudinal fasciculus (ILF)**, the **inferior fronto-occipital fasciculus (IFOF)**, and the **uncinate fasciculus (UF)**, all of which are critical for lexico-semantic connectivity.

fMRI studies of such lesions have demonstrated that speech comprehension depends on a network consisting of posterior temporal cortex, angular gyrus, and anterior temporal lobe, interconnected by the IFOF and ILF (Turken&Dronkers, 2011).

It has been found that damage to the **middle longitudinal fasciculus (MdLF)** disrupts access to semantic representations, leading to symptoms of sensory aphasia (Hamer et al., 2010).

Further evidence confirms that the MdLF is critical for semantic processes: its damage results in semantic paraphasias and impaired speech comprehension (Luo et al., 2019).

Analyses of the role of the **angular gyrus** in semantic processing also demonstrate that its lesion leads to semantic paraphasias and impaired comprehension.

Taken together, these studies support the view that both motor (Broca's) and sensory (Wernicke's) aphasias can be conceptualized as consequences of disrupted **dynamic network interactions** among diverse cortical and subcortical regions.

However, in the existing literature, the **network model of motor aphasia** remains underdeveloped. Therefore, the aim of our study was to construct a network model of motor aphasia.

Methods. At the stroke unit of Odesa Regional Hospital, from January 1, 2024, to May 12, 2025, 25 patients aged 40 to 60 years with aphasia, predominantly due to acute isolated ischemic stroke of the middle cerebral artery, were examined. The matrices of the initial data were constructed by coding the presence of a symptom as 1 and the absence of a symptom as 0.

Mutual, directed influences among symptom indicators were assessed using multiple linear regression and correlation analysis.

Levels of statistical significance were set at $p < 0.05$ and $p < 0.1$.

For mathematical modeling, each selected variable was in turn considered as the dependent variable (Y), while the remaining variables were considered as predictors (X). Using multiple

linear regression, directed influences were determined, yielding regression equations of the form:

Y'=a0+b1X1+b2X2+…+bnXn+e

where a0 is the intercept; b1,b2,…,bnb_1, b_2, …, b_nb1,b2,…,bn are regression coefficients reflecting the influence of the predictor variables X1,X2,…,Xn on the outcome.

The adequacy of regression coefficients was evaluated using their standard deviations, and the overall effectiveness of the regression was assessed by computing the multiple correlation coefficient (Manheim, D. J., & Rich, P. K., 1997). Geometrically, the regression equations were interpreted using polycyclic multigraphs (Diestel R., 2024.).

Own resear+ch and discussion. Table 1 presents the statistically significant regression coefficients reflecting the mutual directed influences of the symptom-indicators of motor aphasia. Figure 1 shows a polycyclic multigraph illustrating the relationships between the symptom-indicators identified in individuals suffering from motor aphasia.

Statistically significant regression coefficients reflecting the mutual directed influences of the symptom-indicators of motor aphasia.

Table 1

Indicators–Symptoms	**Influenceon**	**Influence from**	**Indicators–Symptoms**	**Influenceon**	**Influencefrom**
Anomia			Impaired (motor) speechproduction		
Problems with understanding spoken speech	-0.472	-0.811	Anomia	0.608	-0.0864
Impaired (motor) speechproduction	0.729	0.608	Paraphasia	-0.437	-0.864
Problems with understanding spoken speech			Pathologicalneologisms		
Anomia	-0.811	-0.472	Problems with understanding spoken speech	-0.410	-0.500
Speechnon-fluency	-0.511		Impaired/weakmuscular articulation		
Pathologicalneologisms	-0.500	-0.410	Paraphasia	-0.708	-0.349
Paraphasia					
Impaired (motor) speechproduction	-0.864	-0.437			
Impaired/weakmuscular articulation	-0.349	-0.708			

Bidirectional statistically significant negative mutual influences were observed between anomia and comprehension difficulties in spoken language, with the effect of anomia on comprehension exceeding the reverse influence. Conversely, bidirectional statistically significant positive influences were found between anomia and impaired (motor) speech production, with the effect of motor speech difficulties on anomia predominating.

Anomia arises from the division of processing streams (ventral vs. dorsal) combined with partial preservation of the ventral circuit. In some patients, where anomia is primarily due to "presegmental/lemmatic" access deficits and/or control impairments, the ventral pathway for comprehension may remain relatively intact. In a multivariate model controlling for other symptoms, this produces negative partial coefficients: the more pronounced the anomia (all else being equal), the smaller the additional contribution of deficits to comprehension—a typical pattern of suppressor effects and symptom collinearity in networks (Hickok, G., 2012; Fridriksson, J. et al., 2016).

Patients often exhibit compensatory strategies and resource redistribution. When a word

cannot be accessed, they tend to slow down, rely more on context and perceptual analysis, which may enhance input intelligibility and reduce comprehension errors—again, producing a negative partial effect. Evidence from attention and dual-task studies confirms that cognitive resource redistribution substantially alters comprehension outcomes. Consequently, the influence of anomia on comprehension is logically stronger than the reverse: strategic shifts originate from failed lexical access and secondarily affect speech reception (Zevgolatakou, E. et al., 2022).

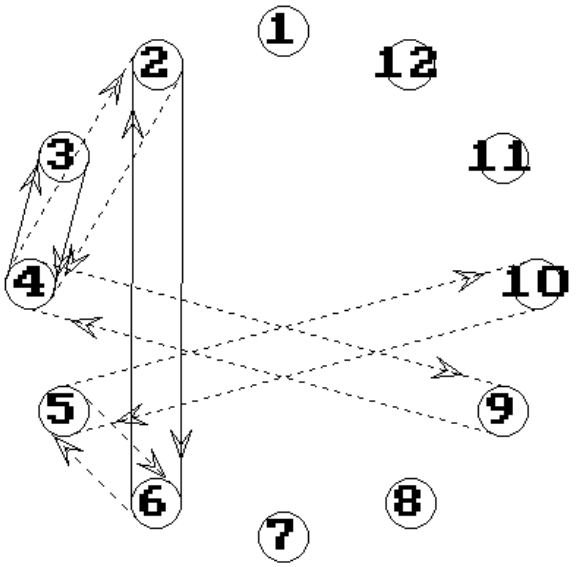

Fig. 1. Polycyclic multigraph illustrating the relational connections between symptom-indicators identified in individuals with Broca's aphasia.

Legend:1—Agrammatism, 2—Anomia, 3—Speech nonfluency, 4—Comprehension difficulties in spoken language, 5—Paraphasia, 6—Impaired (motor) speech production, 7—Awareness of speech deficit, 8—Reduced prosodic expressivity of speech deficit, 9—Pathological neologisms, 10—Impaired/weak muscular articulation, 11—Pauses in speech, 12—Compensation via articulatory gestures.

Solid lines indicate positive influences; dashed lines indicate negative influences.

Interactive models of lexical access emerged as an alternative to strictly sequential (modular) models of speech processing. Their core principle is bidirectional interaction among processing levels (phonemic, morphemic, lexical, semantic). Interactions between semantic and phonological representations, along with error monitoring, mean that a failure in word selection alters expectations/predictions and input processing strategies—again producing a negative "partial" sign in the directed graph.

Anomia – impaired (motor) speech production: Bidirectional positive influences, with the effect of production difficulties on anomia being stronger.

Dorsal pathway involvement: Both anomia (in some cases) and motor speech impairment share nodes in the dorsal stream (articulatory-phonological transformation, sensorimotor mapping). Therefore, when controlling for other nodes, the two symptoms mutually reinforce each other positively (Goodglass, H., & Wingfield, A., 1997).

Fluency as a hub node: Connected speech studies show that fluency deficits are closely linked to lexico-phonological assembly: greater difficulty in speech initiation/planning increases interruptions, repairs, and circumlocutions, which amplify anomia; conversely, pronounced anomia slows speech flow. The predominance of the production → anomia arc is explained by motor planning deficits masking or exacerbating word-finding difficulties more than lexical failures alone can disrupt the motor plan (Na, Y. et al., 2022).

Network (graph) perspective: In language networks, symptoms form clusters; fluency and lexical access features often cluster together, whereas comprehension aligns with the ventral

pathway. This pattern produces positive intra-cluster links and negative links across overlapping clusters **(**Ashaie, S., & Castro, C., 2021).Negative bidirectional relationships between anomia and comprehension do not represent a "therapeutic" effect, but rather reflect specific network effects when the ventral and dorsal streams are separated and/or during compensatory processes. Positive bidirectional relationships between anomia and motor speech production, as well as the predominance of the production → anomia arc, result from shared dorsal nodes (fluency, planning, articulation) and the amplification of lexical errors due to difficulties in initiating and repairing speech (Goodglass& Wingfield, 1997).

Oral comprehension is statistically significantly negatively affected by anomia, speech non-fluency, and pathological neologisms. As noted, anomia exerts a stronger influence on comprehension than the reverse. Similarly, pathological neologisms influence comprehension more than the opposite direction.

In fact, the negative effects of anomia, non-fluent speech, and pathological neologisms on oral comprehension can be explained by their disruption of normal lexical access and semantic integration mechanisms; they interfere with the construction of a proper semantic network for interpreting heard speech (Whitworth, Webster, & Howard, 2014).

Anomia primarily reflects a disruption in access to lexical items; it not only hampers speech production but also impairs the reverse use of the lexical store during speech recognition (comprehension). Therefore, its impact on comprehension is stronger than vice versa (Goodglass& Wingfield, 1997).

Pathological neologisms distort the internal lexicon, generating incorrect lexemes that integrate into cognitive networks and hinder adequate speech recognition (Nickels, 2001; Dell, Schwartz, Martin, Saffran, & Gagnon, 1997).

Speech non-fluency indicates dysfunction in speech programming and control, which also affects "online" perception processes: if the patient cannot produce fluent utterances, the dynamics of predicting others' speech are disrupted.

Thus, the stronger influence of anomia and pathological neologisms on comprehension (compared to reverse effects) can be interpreted within a network model of aphasia: damage to nodes responsible for lexical-semantic access produces cascading effects, including on speech perception.

Statistically significant negative bidirectional interactions were observed between phonemic paraphasia and both impaired (motor) speech production and weak/disrupted muscular articulation. Motor speech production exerted a stronger influence on paraphasia than the reverse, whereas paraphasia had a stronger influence on muscular articulation than vice versa.

These bidirectional relationships can be explained by overlapping speech processing levels. Paraphasias (especially phonemic) are traditionally associated with disruptions at the level of phonological representation and/or access, while impaired motor production is linked to deficits in planning/programming motor commands (apraxia of speech) or peripheral articulatory loss (dysarthria). These levels are not isolated: in normal speech, flow from semantics affects lexical selection, which in turn influences phonology, ultimately impacting motor execution. This flow is interactive, and errors at one level can propagate to others, resulting in statistically significant bidirectional influences in patient groups (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997).

Why motor production influences paraphasia more than vice versa:

If articulation programming/coordination (speech apraxia) is primarily impaired or there is pronounced motor dysfunction, systematic and repeated phonetic/phonemic errors emerge when attempting to articulate phonemic plans, manifesting as phonemic paraphasias. In other words, failures in "translating phonemes into articulation" generate observable paraphasias faster and more consistently than the reverse pathway, which explains why the "motor → paraphasia" effect appears stronger in statistics. This is well supported by studies examining the phonetic nature of phonemic paraphasias and the role of conversion into the articulatory plan (Kurowski et al., 2015).

Why paraphasia influences muscular articulation more than vice versa: Paraphasias, particularly those reflecting unstable phonological/lexical selection (semantic or phonemic errors), lead to repeated attempts to match and adjust articulatory programs, increasing load on the motor system (repetitions, compensations, reassignment of motor commands), which can manifest as impaired coordination or "oscillation" of articulation. Additionally, lesions causing

paraphasias (e.g., parietal–perisylvian areas) often involve neighboring motor/corticobulbar networks, resulting in an asymmetric "paraphasia → articulation" effect (Chen, Q., & Kawashima, H..2024).

Role of interactive models and feedback:

Modern models (interactive two-step models, SLAM, etc.) integrate lexical-phonological and motor-articulatory components, explaining both the existence of bidirectional effects and their asymmetry depending on the "location" and "depth" of the primary deficit. The SLAM model directly demonstrates how adding a motor (articulatory) subsystem changes the distribution of errors and their directional influence (Walker et al., 2016).

Clinically, speech apraxia/articulatory disorders, phonemic paraphasias, and other symptoms often co-occur with lesions in the left fronto-temporo-parietal region; differences in the strength of directional influences may reflect specific lesion sites (e.g., predominant involvement of motor programming vs. phonological networks) and patients' compensatory or restorative strategies (Conterno et al., 2021).

Motor speech production difficulties were statistically significantly positively influenced by anomia and statistically significantly negatively influenced by paraphasia. At the same time, the influence of motor speech production difficulties on anomia and paraphasia was greater than the reverse influences.

The observed positive effect of anomia on motor speech production difficulties can be explained by the fact that anomia (word-finding difficulties) increases motor speech challenges, as the search for a word activates more competing lexico-phonological nodes (Schwartz et al., 2006; Nickels, 2001). This creates additional cognitive load on speech motor planning, resulting in slowing, pauses, and "stumbling" speech. Therefore, the influence is in a positive direction: the stronger the anomia, the greater the difficulty in speech production.

Semantic paraphasia negatively affects motor speech production difficulties. Paraphasia involves "word substitution" (selection errors, often semantic). If a patient more frequently "chooses the wrong word" but pronounces it without marked motor pauses, speech production may appear less "difficult" from a motor perspective. In other words, paraphasia reduces the likelihood of speech "getting stuck," following an "easier path" — incorrect but more motorically automated. Hence, the influence is negative: an increase in paraphasias effectively "reduces" the observable degree of motor difficulties (speech flows more smoothly, though with errors) (Levelt et al., 1999; Dell et al., 1997).

Asymmetry of influences is observed (motor speech production has a stronger effect on anomia/paraphasia than vice versa). Motor difficulties block the speech flow itself, which leads to: increased anomia (words are difficult not only to find but also to articulate); higher likelihood of paraphasias (the system seeks bypass options). In other words, the motor "bottleneck" systematically determines distortions at other levels of the speech process.

In network model terms: the motor node has a higher influence weight on lexico-semantic nodes than the reverse. Thus, the resulting model of symptom interactions fully aligns with the network (interactive) model of speech: anomia burdens motor production, paraphasia "reduces" it via a bypass route, and motor difficulties have systemic priority and stronger influence on other levels (Dell et al., 1997; Nickels, 2001).

Pathological neologisms and oral comprehension difficulties were associated with bidirectional, statistically significant negative mutual influences, with pathological neologisms predominating over comprehension problems.

The relationship between pathological neologisms and comprehension difficulties, characterized by bidirectional statistically significant negative mutual influences, can be explained by several key neuropsychological and neurolinguistic aspects:

Disruptions in neurolinguistic processes: Pathological neologisms often arise in aphasias, particularly anomic and sensory forms, where word-formation and semantic integration processes are impaired. This can lead to the creation of meaningless or distorted words, hindering speech comprehension. At the same time, oral comprehension difficulties may be linked to deficits in auditory and semantic processing, further impairing adequate speech perception and interpretation.

Cognitive and psycholinguistic mechanisms: Studies show that in aphasias and other speech disorders, both semantic and phonological networks of the mental lexicon degrade. This

can lead to catastrophic failures in conceptual representation, which in turn affects the ability to perceive and produce speech.

Mutual influence and predominance of pathological neologisms: According to some studies, pathological neologisms may be more pronounced than oral comprehension problems, especially with left-hemisphere lesions responsible for language function. This may be because speech generation deficits (e.g., creating neologisms) are more noticeable and easily detectable than comprehension difficulties, which can be less obvious (Chaika, E., 1990; Kircher, T., Bröhl, H., Meier, F., Engelen, J., 2018).

Bidirectional statistically significant negative influences were also found between impaired/weak muscular articulation and paraphasia, with paraphasia predominating over impaired/weak articulation.

These bidirectional statistically significant negative influences between impaired/weak muscular articulation and paraphasia, with paraphasia predominating, can be explained by neurophysiological and neuropsychological mechanisms. Paraphasia is a speech disorder characterized by the loss of meaning and grammatical structure, manifesting as substitutions of correct sounds, letters, syllables, or words with others that do not conform to language norms. It is a symptom of various speech and neurological disorders, such as aphasia, alalia, and different forms of dementia.

Articulation impairments, in turn, are linked to disrupted coordination of the muscles involved in speech, which can be caused by various conditions, including strokes, brain injuries, tumors, and other neurological disorders. Articulation difficulties may affect a patient's ability to pronounce words correctly, which can in turn contribute to the emergence of paraphasias.

The predominance of paraphasia over impaired articulation may indicate a more pronounced disruption of cognitive and neuropsychological functions responsible for speech formation and reproduction. This may be associated with lesion localization in specific brain regions involved in language, such as Broca's and Wernicke's areas.

The conducted analysis of mutual influences among speech disorder symptoms demonstrates a complex, multi-level network of interactions between lexical-semantic, phonological, and motor components of speech. Anomia exerts a stronger influence on oral language comprehension than vice versa, reflecting its key role in accessing lexical items and forming semantic representations. Negative partial coefficients indicate both symptom collinearity and suppressor effects arising within the division of the ventral and dorsal speech processing streams.

Positive bidirectional connections between anomia and impaired motor speech production point to shared dorsal nodes involved in planning, articulation, and speech fluency, with the influence of motor difficulties on anomia exceeding the reverse, highlighting the systemic prominence of the "bottleneck" of motor production in the hierarchy of speech processes.

Paraphasias show bidirectional negative effects with motor production and articulation, with asymmetry in these effects reflecting the interactive nature of lexical-phonological and motor levels. Muscle articulation deficits and pathological neologisms impact speech comprehension by disrupting normal lexical-semantic integration mechanisms, with pathological neologisms exerting a stronger effect than comprehension difficulties, emphasizing their dominant cognitive influence.

Thus, the observed patterns are fully consistent with a network-based interactive model of aphasias: symptoms cluster according to processing levels, within which positive connections reflect exacerbation of deficits, and between clusters, negative partial effects arise due to compensation and redistribution of cognitive resources.

This structural perspective explains the directionality and strength of symptom influences, their asymmetry, and the cascading consequences of damage on productive and receptive speech. Overall, the findings emphasize that lexical-semantic, phonological, and motor impairments interact dynamically and contextually, forming complex patterns of speech deficits, which has direct implications for diagnosis, prognosis, and rehabilitation planning in aphasia.

Conclusions

1. **Network nature.** The symptoms of motor aphasia form an interconnected system that integrates lexico-semantic, phonological, and motor components.

2. **Central role of anomia.** Anomia exerts a key influence on speech comprehension and interacts with motor impairments, confirming its importance as a critical node of lexical access.

3. **Asymmetry of connections.** Negative bidirectional influences (e.g., anomia ↔ comprehension) reflect the division of functions between the dorsal and ventral streams, whereas positive connections (anomia ↔ motor functions) point to the involvement of shared dorsal nodes.

4. **Motor–phonological loop.** The strong impact of motor processes on anomia indicates that articulation can support lexical access.

5. **Clinical significance.** The nature and direction of connections help refine the localization of lesions (e.g., left fronto-temporo-parietal overlap) and predict recovery strategies.

6. **Methodological contribution.** The use of multiple regression and polycyclic multigraph analysis demonstrates that aphasia can be described as a systemic disorder rather than a collection of isolated symptoms.

Motor aphasia results from complex network interactions, with anomia acting as the leading node. The dynamics of symptoms are determined by the balance between dorsal and ventral pathways of speech processing. This approach opens new perspectives for diagnosis and individualized rehabilitation.

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