Human Arenas
https://doi.org/10.1007/s42087-024-00430-5

RESEARCH

On the Revival of Neurocognitive Modeling: Testing Old Ideas with New Techniques

Ardi Roelofs[1]

Received: 27 March 2024 / Revised: 13 July 2024 / Accepted: 25 July 2024

Abstract

This article describes how synergy between cognitive psychology, computer science, and artificial intelligence led to a revival of 19th-century neurocognitive modeling in the form of 20th-century neurocognitive computational modeling. Scientific evidence about the mind and its relationship to the brain began to accumulate about two centuries ago. Neurocognitive modeling based on this evidence rose to prominence in the last quarter of the 19th century, with prominent examples being the models of Wernicke and Wundt, presented as diagrams. This work lost its influence after the First World War, but came back to life after the Second. In the 1950s, researchers began developing cognitive models, which became neurocognitive in the 1960s. Moreover, with the rise of computer science and artificial intelligence, modeling became computational. Today's neurocognitive models, realized as diagrams or computer programs, revive the early modeling of Wernicke and Wundt in several respects. While predictions were derived and tested qualitatively from the early models, today they can be derived mathematically in computer simulations and statistically evaluated for quantitative agreement with data sets. I describe how recently 20th-century techniques have been used to test 19th-century ideas about attentional control (Wundt), choice in go/no-go tasks (Donders vs. Wundt), the role of the arcuate fasciculus in speech repetition (Wernicke), and focal behavioral symptoms in neurodegeneration (Wernicke vs. Pick).

Keywords Computational modeling · Neurocognitive · WEAVER++/ARC · Wernicke · Wundt

---

✉ Ardi Roelofs
Ardi.Roelofs@donders.ru.nl

[1] Donders Institute for Brain, Cognition and Behaviour, Centre for Cognition, Radboud University, Thomas van Aquinostraat 4, Nijmegen 6525 GD, The Netherlands

Introduction

While people have speculated about the mind for thousands of years, it was only about two centuries ago that they began to scientifically investigate the mind and its relationship to the brain (e.g., Boring, 1950; Leahey, 2017). Moreover, in the second half of the 19th century, researchers began to develop models that linked cognitive processes to structures and processes of the brain, today called *neurocognitive modeling*. This approach was largely abandoned after the First World War, but revived after the Second. Moreover, with the rise of modern cognitive psychology, computer science, and artificial intelligence in the 1950s, the neurocognitive modeling became *computational* by implementing the models as computer programs. The modeling concerned a revival in the 20th century and not an initial beginning, as is sometimes suggested. For example, in their critically acclaimed book *The Organisation of Mind*, Shallice and Cooper (2011) stated: "Sixty years ago, virtually nothing scientific was known about the general organisation of the mind. … It was in the period 1950–70 that the first major developments occurred" (pp. 2 and 4). However, Shallice and Cooper were wrong by 150 years: They mistook a revival for the beginning. In this article, I briefly describe the 19th-century beginning and the 20th-century revival, and how recently, modern techniques have been used to test early ideas.

A Scientific Beginning 200 Years ago

In the early 19th century, researchers began to use experimental methods to study physiological processes in the brain (e.g., Benjamin, 2024; Boring, 1950; Brysbaert & Rastle, 2021; Leahey, 2017). A hotly debated issue concerned whether mental faculties are precisely localized in the brain, as Franz Joseph Gall (1758–1828) claimed for the cerebral cortex, or distributed holistically, as maintained by Pierre Flourens (1794–1867). To experimentally test between these views, Flourens lesioned specific parts of the brains of animals such as pigeons, rabbits, and cats to study the effect on perception, memory, and movement. The experiments, reported in Flourens (1824), revealed functional specialization. For example, brain stem lesions led to a loss of muscle contractions (*contractions musculaires*), lesions to the cerebellum resulted in a loss of coordinated movements (*mouvemens coordonnés*), and lesioning of the cerebral cortex disrupted higher-level functions, such as voluntary movement (*mouvemens dits volontaires*). However, Flourens was unable to find specific cortical regions for these higher-level functions, which led him to conclude that they are distributed across the entire cerebral cortex, refuting Gall's (1798) phrenological claims. Later discoveries showed, however, that Flourens' cortical holism was wrong not only about animals (Ferrier, 1886; Fritsch & Hitzig, 1870) but also about people. In particular, in the 1860s, Paul Broca (1824–1880) discovered that damage to the left inferior frontal gyrus rather than the entire cortex of patients impaired the ability to produce spoken language, which refuted Flourens' cortical holism. Broca (1861) argued that the localization of one mental faculty, such as his localization of the ability to produce articulated language, is sufficient to establish the truth of the localization principle.

Another important scientific discovery concerned the speed of nerve conduction and mental processing. While Johannes Müller (1801–1858), one of the founders of 19th-century physiology, claimed that nerve conduction proceeds at immeasurable speed (Müller, 1835), his student Hermann Helmholtz (1821–1894) found evidence that the velocity is about 26 m per second in the frog (Helmholtz, 1850), refuting Müller's claim. In the 1860s, a good friend of Helmholtz, the Dutchman Frans Donders (1818–1889), obtained evidence that mental processing speed is finite and measurable. His experiments were conducted in 1865 and published three years later (Donders, 1868) in a journal founded by Müller. In critical experiments, Donders measured the time it took to repeat heard spoken syllables. He used a subtraction method that would also play a crucial role in the rise of modern neuroimaging in the 1990s (Posner & Raichle, 1994). Donders estimated the durations of perceptual discrimination and choice by measuring reaction times for tasks that differed in whether or not they included these processes. Subtracting reaction times for the repetition of spoken syllables in these tasks provided estimates of discrimination and choice durations in milliseconds.

The two groundbreaking discoveries about mental localization and speed laid the foundation for a neurocognitive modeling approach to the mind that linked cognitive functioning to structures and processes of the brain. This approach reached a high point in 1874, exactly 150 years ago, in books of Carl Wernicke (1848–1905) and Wilhelm Wundt (1832–1920). Wernicke (1874) advanced a neurocognitive model of spoken word production, repetition, and comprehension that accounted for the results from "experiments of nature", such as Broca's discovery and his own findings on aphasia. Wernicke called his work "a psychological study on an anatomical basis" in the title of his book. Wundt (1874) proclaimed a new domain of science, which he called *physiological psychology*, in which the neurocognitive approach featured prominently. In the book, Wundt also laid the foundation for his later neurocognitive model of attentional control, which he linked to the prefrontal cortex. Wundt's model was designed to improve on Wernicke's model by incorporating attentional control.

Overview of this Article

In the remainder of this article, I first describe the 19th-century neurocognitive modeling of Wernicke and Wundt, and then its revival in the form of 20th-century neurocognitive computational modeling. Next, I describe how 19th-century ideas have been tested with 20th-century techniques. A first test that I discuss concerned whether Wundt's attentional control model actually works on tasks for which it was designed, such as naming tasks involving distraction. A second test concerned Wundt's (1874) criticism of Donders' (1868) subtractions, which was about the involvement of choice, a simple act of attentional control, in one of Donders' speech repetition tasks. In this task, participants heard one of five syllables and only had to repeat a prespecified syllable. Unlike Donders, Wundt claimed that this task involves a choice, namely whether or not to respond. A third test concerned the role of a major white-matter fiber tract in the brain, the arcuate fasciculus, in speech repetition, on which Wernicke (1874, 1906) and later Geschwind (1970) had not reached agreement. A fourth test concerned focal symptoms in neurodegeneration, the possibility of which was denied by Wernicke (1874), but confirmed by Pick (1892), and only recently became the subject of neurocognitive modeling.

Wernicke's Neurocognitive Model

After studying medicine, Wernicke worked most of his academic career in Breslau (then in the German Empire, now called Wrocław, in Poland), where he became professor of neurology and psychiatry in 1885. In 1874, as an assistant physician, he published his model in a book entitled *Der aphasische Symptomencomplex: Eine psychologische Studie auf anatomischer Basis* (*The Aphasic Symptom Complex: A Psychological Study on Anatomical Basis*). Figure 1 illustrates Wernicke's model, adapting a graph from a reprint of his 1874 book in Wernicke (1893). The core of the model consists of psychological reflexes, which concern learned associations between memory images: auditory images (A), movement images (M), and concepts (C) consisting of sensory images, such as visual images (V) and tactile images (T).

Wernicke (1874) assumed that a child first learns concepts, later learns to associate auditory images for words with movement images for the repetition of them (supposedly learned during babbling), and still later learns to connect the auditory images to the concepts to support comprehension, and the concepts to movement images to support concept-driven production. According to Wernicke, the auditory images for words are stored in the left superior temporal gyrus, the movement images in left inferior frontal gyrus (i.e., Broca's area), and the sensory images for concepts in corresponding cortical areas for their sensory processing, like visual images in occipital cortex and tactile images in the postcentral gyrus.

Wernicke had designed his model to explain classic aphasia syndromes consisting of patterns of impaired and spared word production, repetition, and comprehension. Production concerns saying words to express meaning, repetition concerns saying heard words or pseu-

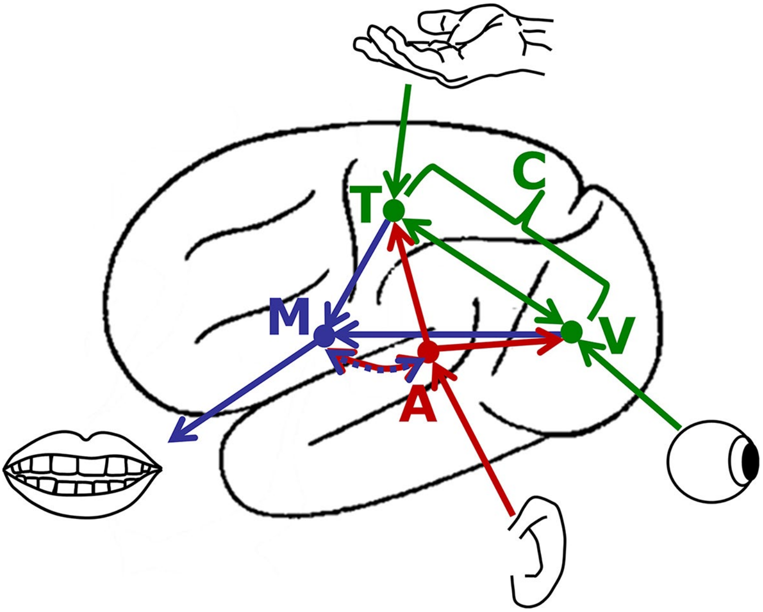
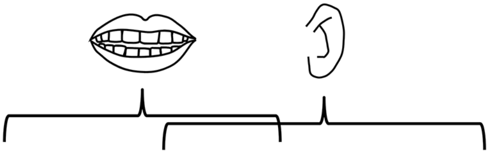


Fig. 1 Illustration of Wernicke's neurocognitive model for word production, repetition, and comprehension. Drawing of the brain taken from Wernicke (1893) and network created by the author based on Wernicke (1874, 1893). The eye, hand, ear, and mouth have been added to indicate the perceptual and movement organs. C=concept, T=tactile, V=visual, A=auditory, M=movement

dowords, and comprehension concerns understanding the meaning of heard words. Syndromes distinguished by Wernicke (1886), and their explanation by his model, are shown in Fig. 2. The syndromes and the basics of the explanation are still largely valid (e.g., Kemmerer, 2022).

In motor aphasia, now called Broca's aphasia, the movement images (M) are taken to be disrupted, due to damage to the left inferior frontal gyrus, which impairs word production and repetition while sparing comprehension. In sensory aphasia, now called Wernicke's aphasia, the auditory images (A) are disrupted, due to damage to the left superior temporal gyrus, which impairs word comprehension and repetition, while sparing word production, apart from frequent errors (*Paraphasien*). According to Wernicke (1874), these errors occur because the auditory images no longer sufficiently constrain the selection of movement images via the direct connection between A and M, assuming activation reverberates between them (Lichtheim, 1885). In transcortical motor aphasia, the connection between concepts and movement images is disrupted, which impairs production while comprehension and repetition remain spared. In transcortical sensory aphasia, the connection between auditory images and concepts is disrupted, which impairs comprehension while repetition and production remain spared, except that errors occur in word production. Rather than assuming that there is reverberation of activation between movement and auditory images, Wernicke (1886) explained these errors by assuming that movement images are activated both directly by concepts and indirectly via auditory images. Therefore, if the connection between concepts and auditory images is disrupted, the auditory images can no longer constrain the selection of the movement images in word production. Finally, in conduction aphasia, the connection between A and M is disrupted, causing errors in production and repetition (now accomplished via concepts, as Lichtheim argued in his 1885 article) while comprehension is spared. Wernicke (1906) made it clear that a test of the intactness of the connection between auditory and movement images requires repetition of *pseudowords*, which are similar to real words but do not actually occur in the language. This is because a real word, but not a pseudoword, can also be repeated by having the auditory image activate the concept, which then activates the movement image (Lichtheim's argument).



Aphasia type	Production	Repetition	Comprehension	Disruption
Motor (Broca's)	X	X	√	~~M~~
Sensory (Wernicke's)	√ x	X	X	~~A~~
Transcortical motor	X	√	√	C ~~—~~ M
Transcortical sensory	√ x	√	X	A ~~—~~ C
Conduction	√ x	√ x	√	A ~~—~~ M

√ = spared, X = impaired

Fig. 2 Classic aphasia syndromes and their explanation according to Wernicke (1874, 1886)

Based on his own postmortem brain dissections, Wernicke (1874) claimed that the connections between the auditory images in the superior temporal gyrus and the movement images in the inferior frontal gyrus were supported by a fiber path passing through the insula, which is a part of the cerebral cortex that is folded deeply into the lateral sulcus. However, Wernicke's contemporaries Dejerine and von Monakow pointed to a role of the arcuate fasciculus based on their own postmortem brain dissections (e.g., Dejerine, 1895; von Monakow, 1897). I refer to Roelofs (2024) for a detailed discussion.

Although Wernicke's model had a major immediate impact, it lost its influence after the First World War. The model was revived half a century later by Geschwind (1965, 1970, 1972), with a legacy of unresolved issues. These included the anatomical underpinning of the connection between the auditory and movement images, on which the views of Wernicke and Geschwind differed, and the sufficiency of the psychological reflexes to explain language performance, which was questioned by Wundt (1900).

Wundt's Neurocognitive Model

After studying medicine and being Helmholtz' assistant for a few years, Wundt worked most of his academic career in Leipzig (then in the German Empire), where he became professor of philosophy in 1875. In 1879, Wundt started a laboratory for experimental psychology, the first of its kind in the world. With his monumental two-volume *Die Sprache* (*Language*), published in 1900, he provided a synthesis of 19th-century psycholinguistics and laid the foundation for modern developments (Levelt, 2013). In a discussion of Wernicke's model, Wundt argued that something important is missing, because it is based on psychological reflexes only. Consider the following conversation between Wernicke and his patient Seidel (from Wernicke, 1874). Wernicke: "Is your name Seidel?" Seidel: "Yes". Wernicke's model implies that when hearing the question, the auditory images of the words in the question, including "Seidel", are activated, and the auditory images activate the corresponding movement images. This predicts that the patient would repeat the question, or part of it, like "Seidel", as patients with transcortical aphasia often do (Kemmerer, 2022). Instead, patient Seidel answered the question appropriately. Thus, something controls the processing, which is not part of the model. Wundt's model addressed this issue.

In his book entitled *Grundzüge der physiologischen Psychologie* (*Principles of Physiological Psychology*), Wundt (1874) argued that correct responding occurs because the "laws of association, too, are entirely subject to the control of attention" (p. 793)[1], which "expresses itself not only in the elicitation of certain movements, but also in the perception of sense impressions and the reproduction of ideas" (p. 830)[2]. He linked attentional control to the frontal lobes. From the second edition of his *Grundzüge* onward, published in 1880, Wundt presented a diagram that formalized his neurocognitive ideas about attentional control, which he referred to as *apperception*, illustrated in Fig. 3 (from Wundt, 1902).

Although forgotten today, Wundt's model was well known at the time. For example, Ribot, the first professor of experimental psychology in France (Nicolas & Murray, 1999), promoted Wundt's work since the 1870s. In the various editions of *La Psychologie Alle-*

---

[1] "Auch die Associationsgesetze sind ganz und gar der Herrschaft der Aufmerksamkeit unterworfen" (p. 793).

[2] "… äussert sich nun nicht bloss in der Hervorrufung bestimmter Bewegungen, sondern auch in der Auffassung der Sinneseindrücke und der reproducirten Vorstellungen" (p. 830).

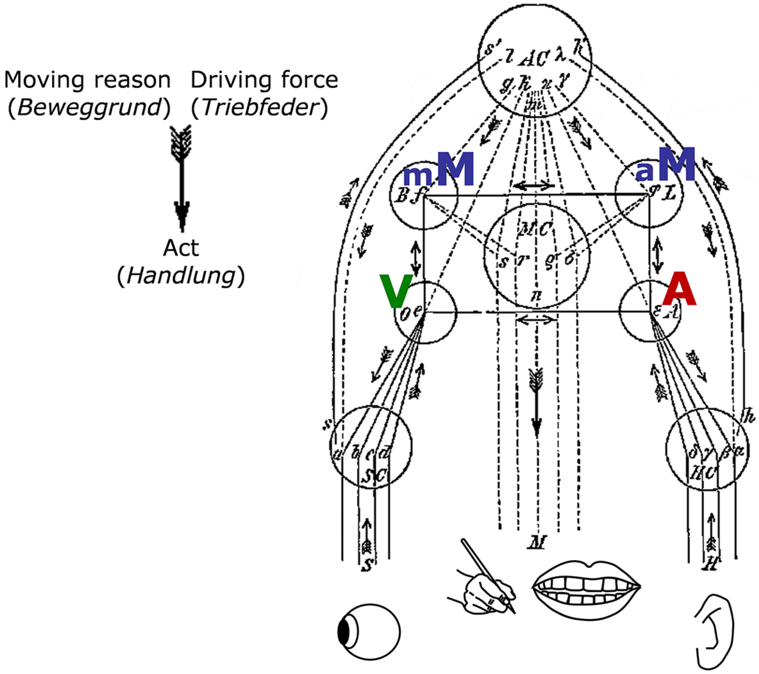


Fig. 3 Illustration of **(left)** a motivated act and **(right)** the network of Wundt's neurocognitive model for spoken and written word production, comprehension, and repetition. Network taken from Wundt (1902) and the remainder added by the author based on Wundt (1896, 1902). The eye, hand, mouth, and ear have been added to indicate the organs of perception and movement. V = visual, mM = manual movement, aM = articulatory movement, A = auditory

mande Contemporaine (*Contemporary German Psychology*), from the second edition in 1885 to the seventh in 1909, he described Wundt's neurocognitive model of apperception in detail (Ribot, 1885, 1909).

Wundt's model assumes that auditory and movement images for spoken words are represented in superior temporal and inferior frontal areas respectively, as in Wernicke's model, and visual and movement images for written words in occipitotemporal and midfrontal areas, respectively. An important addition by Wundt was a center for attentional control, the apperception center (AC), which he assumed to be located in prefrontal cortex. Attentional control was viewed as a mental act, conditioned by a moving reason and a driving force (Wundt, 1896), somewhat similar to modern IF-THEN production rules. One attentional control act was hypothesized to be inhibition. The motivated acts mediated both attention-controlled and automatic mental processes. Furthermore, Wundt assumed that motivated acts could be chained, with one act providing the motive for the next.

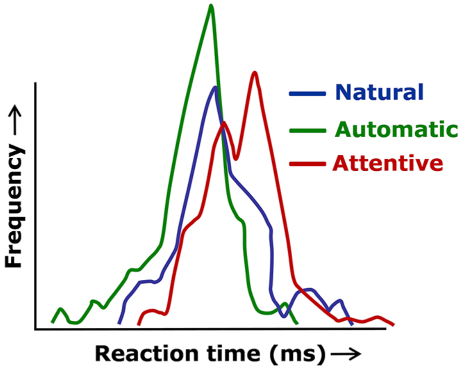
Elsewhere (Roelofs, 2021), I have argued that Wundt's view about attentional inhibition fits well with modern evidence that alpha activity in the brain underpins gating through inhibition (e.g., Jensen & Mazaheri, 2010). Attentional inhibition can explain why the patient of Wernicke said "Yes" and not "Seidel". Selective responding when answering a question, as in the Seidel example, is achieved by inhibiting the connection between the auditory image and the movement image, or between the movement image and the articulation organs, for the inappropriate response “Seidel” and other words, so that the correct answer “Yes” can be produced. Attentional inhibition also explains correct performance on Donders’ go/no-go task, in which participants heard one of five syllables and only had to repeat a prespecified syllable. Initially, all movement images are inhibited, and after the decision to respond, the movement image of the prespecified syllable is released (e.g., Jongman et al., 2020; Piai et al., 2015).

Wundt tested his model in reaction time experiments, inspired by the work of Donders (1868). In a famous photo, taken in 1912, Wundt pretends to be involved in such a reaction time experiment. In the background hangs a poster with important reaction time data. One of the two graphs on the poster is displayed in Fig. 4, showing reaction time distributions in three conditions (Roelofs, 2021).

Wundt (1903) explained the reaction time curves as follows. Suppose that during a series of trials a participant is asked to press a button when hearing a sound. According to Wundt, the first few hundred trials will lead to a *natural* distribution of reaction times, which is the result of a mixture of *automatic* and *attentive* responses. If the participant performs a few hundred more trials, the corresponding reaction time distribution is shifted to the left relative to the natural distribution, indicating that more responses have become automatic. When the participant is instructed to respond attentively, the distribution of reaction times for these trials is shifted to the right relative to the natural distribution. According to Wundt, this shows that apperception involves additional processing, which increases reaction time. Wundt did not have the means to formally analyze the reaction time distributions, which we can do today (e.g., Luce, 1986).

Wundt (1874) had begun to use interference and a stimulus-onset asynchrony (SOA) manipulation as a way to study apperception. In particular, he conducted an experiment in which he examined the influence of a distracting sound (*störender Klang*, p. 748) on the reaction time of manually responding to a target sound (*Haupteindruck*). By manipulating the SOA (the exact SOA was not reported), the time course of the interference could be examined. Wundt observed that reaction time was longer with distractor preexposure (*vorher*) than with postexposure (*nachher*) or simultaneous presentation (*gleichzeitig*). A century later, Glaser and Glaser (1982) and Glaser and Düngelhoff (1984) studied attentional control by examining the SOA curves of interference from distractor words during color and picture naming in color-word Stroop and picture-word interference experiments, which I discuss later. Stroop’s (1935) task was inspired by work in Wundt’s laboratory by

Fig. 4 Reaction time distributions on Wundt’s poster (after Wundt, 1903)



Cattell (1885), who conducted groundbreaking studies in the 1880s examining how long it takes to name pictures and colors and to read aloud words.

The Revival of Neurocognitive Modeling

The early neurocognitive modeling approach of Wernicke and Wundt lost influence after the First World War, but underwent a revival after the Second. During the interwar period, Wernicke's localization view of the mind was replaced by a holistic view (for reviews see Levelt, 2013; Tesak & Code, 2008). This view is exemplified by Goldstein (1939) and the work of Gestalt psychologists (reviewed by Ash, 1995). Wundt's work was mainly forgotten (e.g., Blumenthal, 1975; Brysbaert & Rastle, 2021; Leahey, 1979). In the 1950s, Donald Broadbent (1926–1993) proposed a cognitive filter model of attention (Broadbent, 1958), unaware of Wundt's model. In the 1960s, Norman Geschwind (1926–1984) revived Wernicke's neurocognitive model (Geschwind, 1965, 1970, 1972). From the 1980s onward, as a result of the rise of computer science and artificial intelligence (e.g., Lachman et al., 1979; Meyer & Kornblum, 1993), computational models were developed. These included psycholinguistic models of naming, repetition, comprehension, and reading (e.g., Coltheart et al., 2001; Dell et al., 1997; McClelland & Rumelhart, 1981; Seidenberg & McClelland, 1989; Ueno et al., 2011).

Yet attentional control mechanisms were still lacking in the modern psycholinguistic models. Like Wernicke's model, the modern models were designed to perform two or more tasks, such as picture naming and speech repetition (Dell et al., 1997), naming, repetition, and word comprehension (Ueno et al., 2011), and reading aloud and reading for comprehension (Coltheart et al., 2001; Seidenberg & McClelland, 1989). However, there were no attentional control mechanisms within the models that mediated selective processing for one task or the other, or that resolved interference when multiple stimuli were present, such as hearing or seeing a word while naming a picture. Geschwind (1970, 1972) revived Wernicke's model without including attentional control mechanisms. To my knowledge, the WEAVER++ model (e.g., Roelofs, 2003) and its neurocognitive version WEAVER++/ARC (e.g., Roelofs, 2014) are still the only computationally implemented psycholinguistic models that include attentional control mechanisms. They combine ideas from the work of Wernicke and Wundt with modern insights in a computational form.

Computationally Testing Wundt's Neurocognitive Model

Wundt's neurocognitive model, illustrated in Fig. 3, had been evaluated qualitatively but never quantitatively. Thus, the question remained whether its assumptions are warranted. To assess the model more formally, I created a possible computational implementation of Wundt's model (Roelofs, 2021), called Wundt 2.0. I tested the model on the SOA curves of interference in color-word Stroop and picture-word interference experiments, in which gating by inhibition is predicted to play an important role.

In the version of the Stroop task used by Glaser and Glaser (1982), participants named colored rectangles and tried to ignore superimposed distractors, which were incongruent or congruent color words, or neutral *x*'s. For example, they said "red" to a red colored rectangle while trying to ignore the superimposed word *green* (incongruent) or *red* (congruent), or *x*'s (neutral baseline). The words and *x*'s were presented at SOAs ranging from 400 ms preexposure (i.e., −400 ms) to 400 ms postexposure. Other participants read aloud the words while trying to ignore incongruent or congruent colored rectangles or a neutral empty frame. Glaser and Düngelhoff (1984) did the same using pictures and words. For example, participants said "cat" to a picture of a cat while trying to ignore the word *dog* (incongruent) or *cat* (congruent), or *x*'s (neutral baseline), and other participants read aloud the words while ignoring incongruent or congruent pictures or an empty picture frame. The results for color-word Stroop and picture-word interference were the same. Color and picture naming were delayed in the incongruent condition relative to the neutral *x*'s, with interference peaking around zero SOA. Congruent words facilitated color and picture naming relative to *x*'s at the preexposure SOAs. Word reading was not affected by incongruent or congruent colors or pictures at any SOA.

Computer simulations showed that the Wundt 2.0 model captures the findings on color-word Stroop and picture-word interference (Roelofs, 2021). This is illustrated for picture-word interference in Fig. 5. Varying the assumptions of the model in the simulations revealed that attentional inhibition of distractor perception was crucial for obtaining a fit between model and data, with interference peaking around zero SOA in picture naming and no effect in word reading. Moreover, without perceptual inhibition, the word would be read aloud rather than the picture named in the picture naming task.

The simulations provided evidence that Wundt's attentional control model, originally developed in the 19th century and tested in the modern Wundt 2.0 version, actually performs well on tasks for which it was designed, such as naming tasks that involve distraction. Moreover, Wundt's model turned out to do a better job than most modern models at explaining the time course of interference (see Roelofs, 2021, for discussion). A model that performed as well as Wundt 2.0 is the WEAVER++ model, which embodies some of the same ideas as Wundt's model.

The WEAVER++ Model

The WEAVER++ model is a computationally implemented model that has been developed over the past thirty years at the Max Planck Institute for Psycholinguistics and the Donders Institute for Brain, Cognition and Behaviour of Radboud University in Nijmegen (e.g., Levelt et al., 1999; Roelofs, 1992, 1997, 2003, 2008, 2014, 2022, 2023a, 2024). The model has several features derived from Levelt's (1989) comprehensive processing theory of speaking. The acronym WEAVER++ stands for Word Encoding by Activation and Verification, involving associative memory and procedural knowledge, respectively. The ++ (i.e., the increment operator in the programming language C) indicates that the model is an incremental extension of WEAVER, which was a model of word-form encoding (Roelofs, 1997) complementing a nameless model of lemma retrieval (Roelofs, 1992). The model combines elements from cognitive psychology (i.e., type of theory and experimental paradigm used to test it), artificial intelligence (i.e., relational network and IF-THEN rules), and computer science (i.e., the C language in which the model was programmed).

The WEAVER++ model was developed by me without knowledge of Wundt's ideas about attentional control. It was not until around 2008 that I discovered Wundt's work (e.g., Roe-

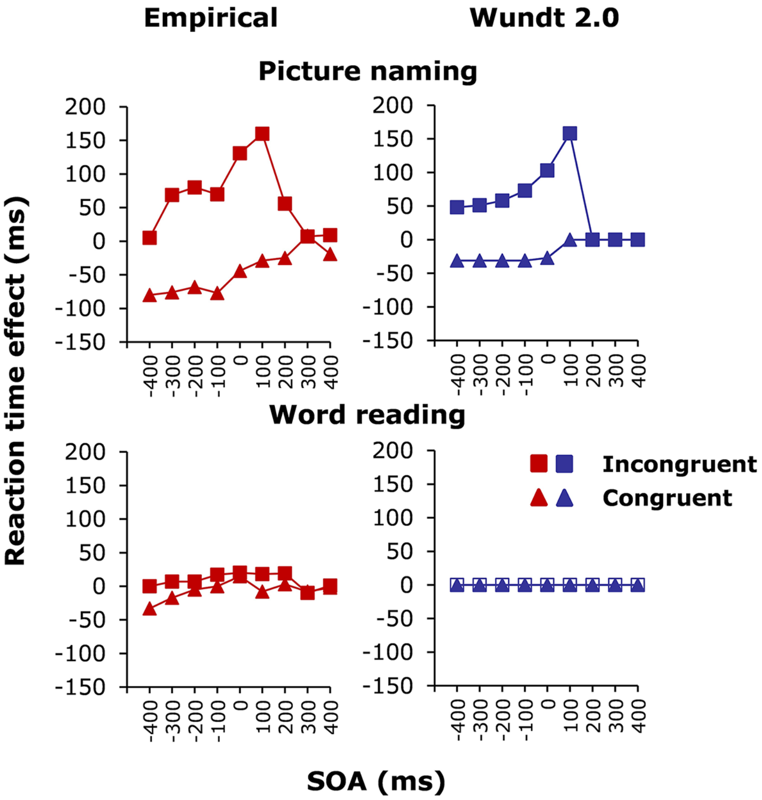


Fig. 5 Stimulus onset asynchrony (SOA) curves for the effect of **(top panels)** incongruent and congruent distractor words relative to neutral *x*'s in picture naming, and **(bottom panels)** incongruent and congruent pictures relative to an empty frame in word reading, as observed **(left panels)** empirically by Glaser and Düngelhoff (1984) and **(right panels)** in Wundt 2.0 simulations (Roelofs, 2021). ms = milliseconds

lofs, 2008), and saw the similarity with my own modeling. In particular, Wundt's distinction between an activation network and motivated acts corresponds to WEAVER++'s distinction between a spreading activation network and IF-THEN production rules. Another similarity is the central role of attentional control, which in both Wundt's model and WEAVER++ is supported by procedural knowledge.

The WEAVER++ model assumes that word production proceeds from conceptualizing (e.g., conceptual identification of a pictured object) via lemma retrieval (lexical selection) to word-form encoding and articulation, the latter also being involved in speech repetition and reading aloud. The model assumes a declarative network and procedural IF-THEN rules. Figure 6 illustrates part of the network with concepts and lemmas, and an IF-THEN rule. The associative network is accessed by spreading activation, while IF-THEN rules select from the activated nodes those nodes that meet the goals and task requirements specified in working memory (e.g., naming an object). Rules enhance the activation of target concepts (shown in Fig. 6) and block out irrelevant perceived words (not shown in the figure), thereby realizing an inhibitory filter as in Wundt's model.

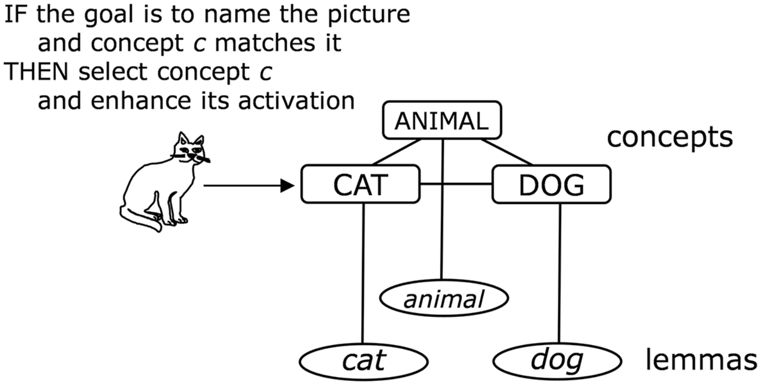


Fig. 6 Part of the network and an IF-THEN rule of the WEAVER++ model

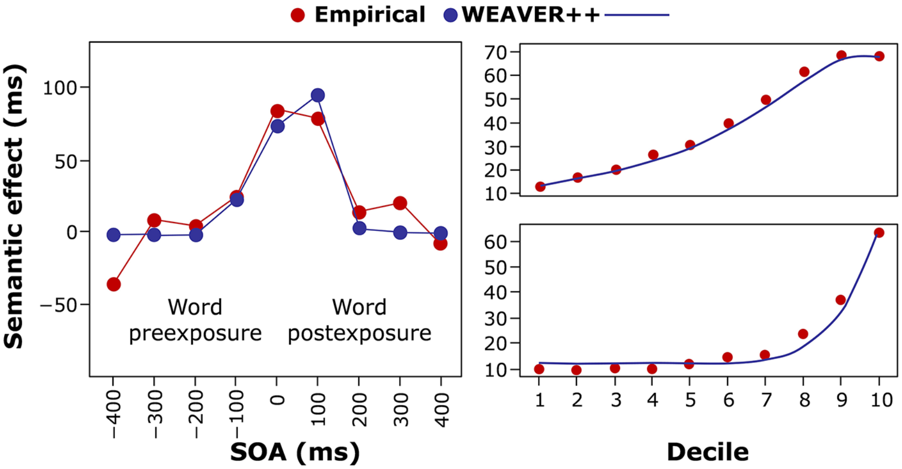


Fig. 7 **(left panel)** The semantic effect of distractor words in picture naming as a function of stimulus onset asynchrony (SOA) observed empirically by Glaser and Düngelhoff (1984) and in WEAVER++ simulations (Roelofs, 1992). The semantic effect across the reaction time distribution observed empirically **(right, top panel)** by Roelofs and Piai (2017) and **(right, bottom panel)** by Scaltritti et al. (2015) and **(right, both panels)** in WEAVER++ simulations (San José et al., 2021)

Simulations with the model published in Roelofs (1992) focused on several findings about picture-word interference, including the time course of interference, which concerns the effect of distractor words presented at different SOAs (as earlier discussed for Wundt 2.0). In particular, the model addressed *semantic* effects, which concern the difference in reaction time between picture naming in the context of semantically related and unrelated distractor words. For example, participants said "cat" to a picture of a cat while trying to ignore the word *dog* (semantically related) or *tree* (unrelated). The left panel of Fig. 7 shows the classic data of Glaser and Düngelhoff (1984) and WEAVER++ simulation results, which fit the real data.

Thirty years later, in 2021, WEAVER++ simulations concerned the entire reaction time distribution of the semantic interference. The right panels of Fig. 7 show how the semantic effect at zero SOA varied empirically across the entire reaction time distribution, from decile to decile, in studies of Roelofs and Piai (2017), shown in the top panel, and Scaltritti et al. (2015), shown in the bottom panel, and in WEAVER++ simulations (San José et al., 2021). The model captures the difference in distribution of the semantic effect between the two studies by assuming different rates of attentional lapses, concerning temporary failures to block out the distractor word or to maintain the task goal in working memory. Wundt (1874) investigated interference via SOA manipulation and later tested his theory of attentional control by qualitatively examining reaction time distributions (Wundt, 1903). The modern empirical studies and the simulation results demonstrate the increase in precision obtained over the past 150 years.

Choice in Go/no-go Tasks (Donders vs. Wundt)

A few years ago, the WEAVER++ model was used to address an old controversy between Donders and Wundt about the involvement of choice in the go/no-go task. After studying medicine, Donders worked most of his academic career in Utrecht (in The Netherlands), where he became a professor in 1848, first in medicine and later also in physiology. In 1865, he measured reaction times for spoken syllable repetition using three tasks: simple, choice, and go/no-go. In the simple task, a student of Donders pronounced a predetermined syllable such as *ki* and Donders repeated it, which was assumed to consist of hearing the syllable and articulating it. In the choice task, the student pronounced one of five syllables and Donders repeated the syllable heard, which was taken to additionally consist of discrimination between the five syllables and choosing the appropriate movement program. In the go/no-go task, the student pronounced one of five syllables and Donders only repeated a predetermined syllable, for example *ki*. This was thought to involve discrimination, but not choice, as the movement program was known in advance. Donders obtained the choice duration by subtracting the go/no-go reaction time from the choice reaction time, yielding 47 milliseconds, and the discrimination duration by subtracting the simple reaction time from the go/no-go reaction time, yielding 36 milliseconds. Wundt (1874) argued, however, that Donders' subtractions were inaccurate because the go/no-go task also involves a choice, namely whether to respond or not, which is an act of attentional control. This would imply that subtracting the go/no-go reaction time from the choice reaction time underestimates the choice duration.

In early 2018, I discovered unpublished data in a handwritten laboratory notebook of Donders. The notebook is in the archives of the University Museum Utrecht (Donders, 1865). The data in the notebook showed that there was no difference between go/no-go and choice reaction times when his students repeated the syllables, indicating that they had more difficulty than Donders himself in making the decision to go. If the go/no-go task involves a choice, and this choice takes considerable time for some participants, then the reaction times for the go/no-go and choice tasks may be similar for them, as observed for the students. To further examine the issue, I replicated the experiment of Donders together with my daughter Sterre (as Donders had done with his daughter Marie in some experiments), using all the original stimulus lists of Donders (Roelofs, 2018), which were also in his handwritten notebook. My daughter spoke the syllables and I repeated them. We replicated Donders' own pattern of reaction times, but with a smaller choice than discrimination duration, while these were comparable for Donders, suggesting that the go/no-go decision was more difficult for me than for Donders.

Computer simulations using the form encoding part of WEAVER++ provided evidence that individual differences in go/no-go cost may underlie the difference in reaction time patterns between Donders and his students. Figure 8 shows the empirically observed patterns and simulation results. When the attentional control cost is negligible, the reaction time pattern of Donders himself is obtained (simple < go/no-go < choice), and when it is more substantial, the model yields the pattern of his students (simple < go/no-go = choice). The model explains 93% of the variance in the real data.

Dondersian Subtraction in Modern Neuroimaging

Donders' subtraction technique has been crucial for the rise of modern neuroimaging. Since the 1960s, local blood flow in the human brain could be measured, but the tasks used were far too complex to localize subprocesses, such as imagining yourself walking through a city versus resting. Psychologist Posner and neurologist Raichle got the crucial insight that Donders' subtraction logic combined with simple tasks was needed. This approach allowed for the precise localization of simple mental processes, leading to a boom in neuroimaging studies in the early 1990s (see Raichle, 1998, for a brief history). Several of these studies are reviewed by Posner and Raichle in their book *Images of Mind*, published in 1994. In the early years of functional neuroimaging, positron emission tomography (PET) was used, whereas later studies more often employed functional magnetic resonance imaging (fMRI). PET uses radioactive tracer to measure local blood flow or energy metabolism, and fMRI measures the blood oxygen-level-dependent (BOLD) response that reflects local ratios of oxyhemoglobin versus deoxyhemoglobin, indexing neuronal activity.

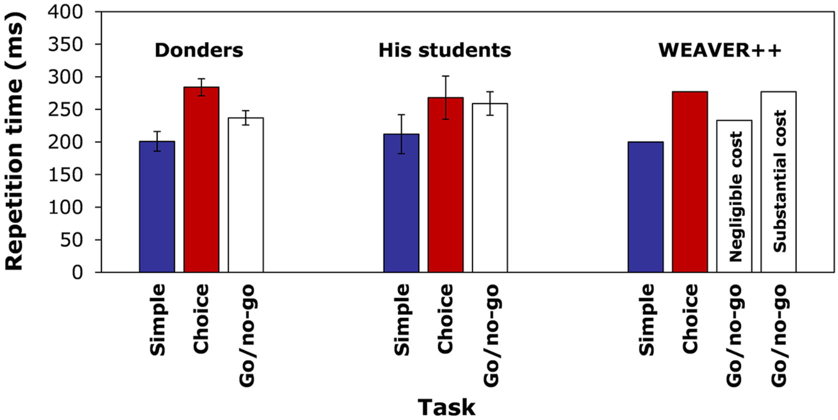


Fig. 8 Reaction time patterns for the simple, choice, and go/no-go tasks of **(left)** Donders and **(middle)** his students, and **(right)** in WEAVER++ simulations by Roelofs (2018) depending on the magnitude of the go/no-go cost (negligible vs. substantial)

In a now classic study (Petersen et al., 1988), Petersen, Posner, and Raichle, together with other colleagues, subtracted the PET image with brain activation obtained when participants looked passively at a fixation cross (+) from the PET image obtained when, in addition, they passively listened to words (e.g., the word *hammer*). This led to a localization of the perception of spoken words in the left superior temporal gyrus. Next, the PET image for passively listening to words (e.g., the word *hammer*) was subtracted from the PET image for actively repeating the heard words (i.e., say "hammer" to *hammer*). This led to a localization of the articulatory planning of words in left premotor and motor areas in frontal cortex. Finally, the PET image for repeating the words (e.g., say "hammer" to *hammer*) was subtracted from the PET image for saying a verb expressing a use for the noun (e.g., say "hit" to *hammer*). In this task, participants do not produce the strongest association but the association that is appropriate to the instruction, requiring attentional control. This led to a localization of attentional control in the frontal lobes, including left lateral prefrontal cortex and the anterior cingulate cortex. Thus, the PET imaging study supported Wundt's claim about the frontal location of attentional control. The attentional control is needed to sequence the processes in verb generation and to prevent inadvertent repetition of the spoken noun (as in the earlier Seidel example, to comprehend and respond instead of repeating a word), which would be a predominant response.

The WEAVER++/ARC Model

In more recent years, a neurocognitive version of WEAVER++ has been developed, called WEAVER++/ARC, with ARC standing for Arcuate Repetition and Conversation. The hypothesized functional neuroanatomy is illustrated in Fig. 9, which was initially based on a meta-analysis of neuroimaging studies in the spoken word production and listening literature (Indefrey & Levelt, 2004), and later confirmed by targeted studies conducted during the past twenty years (e.g., Kemmerer, 2022). The associative network that realizes declarative knowledge is thought to be represented in temporal and inferior frontal areas of the human

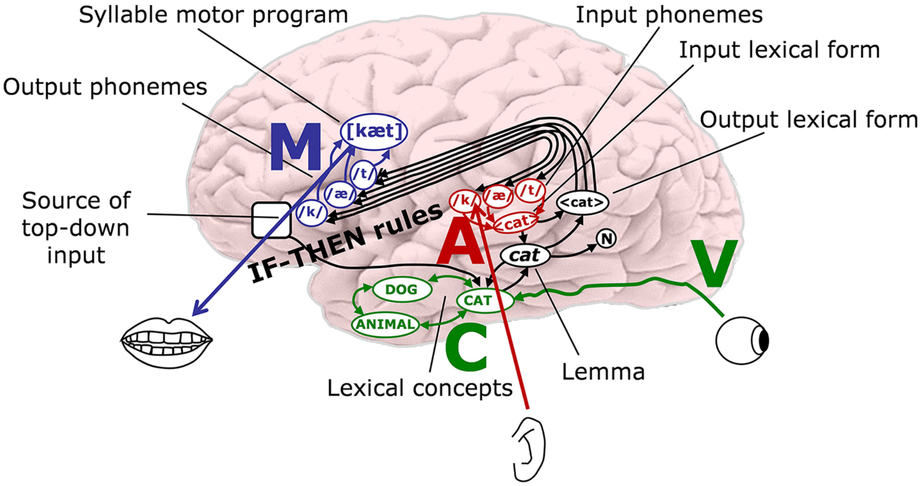


Fig. 9 Illustration of the neurocognitive WEAVER++/ARC model. See the text for an explanation

brain, including Wernicke’s area and Broca’s area. The system of IF-THEN rules that realize procedural knowledge is represented in the basal ganglia, thalamus, and frontal cortex, including Broca’s area. Eichenbaum (2012) described the brain systems for declarative and procedural memory. The associative network is accessed by spreading activation, while IF-THEN rules select nodes that meet the goals and task requirements specified in working memory (e.g., naming an object), associated with dorsolateral frontal cortex. Attentional control is exerted from frontal cortex mediated by the procedural system.

The network contains input phonemes (e.g., /k/, /æ/, and /t/) and input lexical forms (e.g., < cat>), roughly corresponding to the A nodes of Wernicke’s model, thought to be represented bilaterally in the middle to posterior superior temporal gyrus and superior temporal sulcus. Output phonemes (e.g., /k/, /æ/, and /t/) and motor programs for syllables (e.g., [kæt]), roughly corresponding to the M nodes of Wernicke’s model, are thought to be represented in left posterior inferior frontal gyrus (i.e., Broca’s area) and the ventral precentral gyrus. Concept nodes (C) are represented bilaterally in the anterior temporal lobes, separate of modality-specific features, such as visual features (V) in occipital and inferior temporal cortex. Lemma nodes (e.g., *cat*) are represented in left middle temporal cortex, linked to syntactic properties (N = noun). The arcuate fasciculus is thought to mediate two processing streams: a lexical stream for conceptually guided production, enabled by connections from output lexical forms to output phonemes, and a nonlexical stream for phoneme-based repetition (e.g., for novel words, pseudowords), enabled by connections from input to output phonemes. Furthermore, output phonemes activate input phonemes, which supports self-monitoring.

Note that Donders’ syllable repetition task requires the nonlexical connections. This was assumed in the previously described computer simulations of Donders’ (1868) classic study. Contrary to Wernicke’s (1874) assumption, the arcuate fasciculus is thought to mediate the repetition in the model.

Role of the Arcuate Fasciculus (Wernicke)

Wernicke (1874) assumed that repetition is mediated by a fiber path running through the insula rather than by the arcuate fasciculus. Support for the assumption in WEAVER++/ARC that the arcuate fasciculus mediates lexical and nonlexical phonological connections comes from a recent study by Janssen et al. (2023) that combined fMRI with diffusion-weighted imaging and probabilistic tractography. Participants performed verb generation (e.g., saying “hit” in response to the heard word *hammer*) and pseudoword repetition (e.g., saying “tokber” to the heard pseudoword *tobker*). The results are shown in Fig. 10. Both tasks activated the inferior frontal gyrus. Furthermore, pseudoword repetition activated the superior temporal gyrus more than verb generation. During the latter, other temporal areas, including the middle temporal gyrus, and other frontal areas were activated more than during pseudoword repetition. The tractography revealed that one subtract of the arcuate fasciculus running from the superior temporal gyrus to the inferior frontal gyrus was specifically engaged in pseudoword repetition, which requires the nonlexical connections. In contrast, another subtract of the arcuate fasciculus running from the left middle temporal gyrus to the inferior frontal gyrus was specifically engaged in verb generation, which requires the lexical connections.

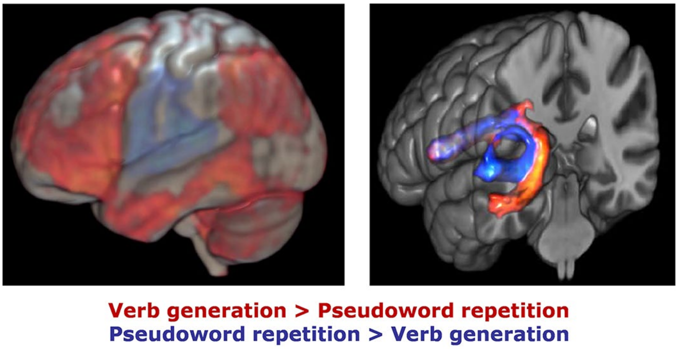


Fig. 10 Functional activation and tractography results for verb generation and pseudoword repetition averaged across 50 participants. **(left)** Activation for verb generation>pseudoword repetition (red) and pseudoword repetition>verb generation (blue). **(right)** Arcuate fasciculus subtracts subserving verb generation (red) and repetition (blue). Adapted from Janssen et al. (2023)

Simulations have shown that the WEAVER++/ARC model accounts for the results of lesion-deficit analyses linking word production, repetition, and comprehension impairments to damaged brain areas and fiber tracts, including the arcuate fasciculus. The model has been applied to both poststroke aphasia syndromes (Roelofs, 2014), as originally described by Wernicke (1874, 1886, see Fig. 2), and impairments due to neurodegenerative diseases (Roelofs, 2022, 2023a).

A century after it was proposed, Geschwind (1965, 1970, 1972) revived Wernicke's model. In his version of the model, Geschwind proposed that the arcuate fasciculus supports both concept-driven word production (e.g., engaged in spontaneous speech, picture naming, or verb generation) and speech repetition (e.g., engaged in syllable, word, or pseudoword repetition), while Wernicke (1874) had assumed that repetition is mediated by fibers that run through the insula. Different from WEAVER++/ARC, Geschwind assumed that word production and repetition are mediated by exactly the same arcuate fibers, which explains why repetition and production deficits are correlated after damage. However, explaining dissociations poses a challenge to Geschwind's model. For example, in the modern literature, Selnes et al. (2002) reported a patient with extensive damage to the arcuate fasciculus, who presented with impaired spontaneous speech and naming, but relatively spared repetition. Elsewhere, I have argued that patient data provide evidence for double dissociation (Roelofs, 2024).

According to the WEAVER++/ARC model, both picture naming and word repetition are supported by the arcuate fasciculus, thus damage to this fiber tract is expected to impair both naming and repetition. However, double dissociation between naming and repetition may also occur. Disruption of the connections between output lexical forms and phonemes should impair naming more than repetition, whereas disruption of the connections between input and output phonemes is expected to impair repetition more than naming. In my reanalysis of a study in patients with arcuate fasciculus damage reported by Marchina et al. (2011), the predicted correlation and double dissociation were observed (Roelofs, 2024).

The top panels of Fig. 11 show that damage to the arcuate fasciculus causes naming and repetition deficits, with more damage leading to poorer performance (solid trendlines). The figure also shows that the WEAVER++/ARC simulations reveal the same (dashed trendlines). Moreover, the bottom panel shows that naming and repetition performance are correlated in these patients (solid trendline), but double dissociation is also observed (circled data points for patients 1 and 5). With 31% damage of the arcuate fasciculus, Patient 1 scored 95% correct on picture naming but only 60% correct on word repetition, while with 55% damage, Patient 5 scored 100% correct on word repetition but only 28% correct on picture naming. The figure shows that WEAVER++/ARC captures these findings (dashed trendline, solid circles).

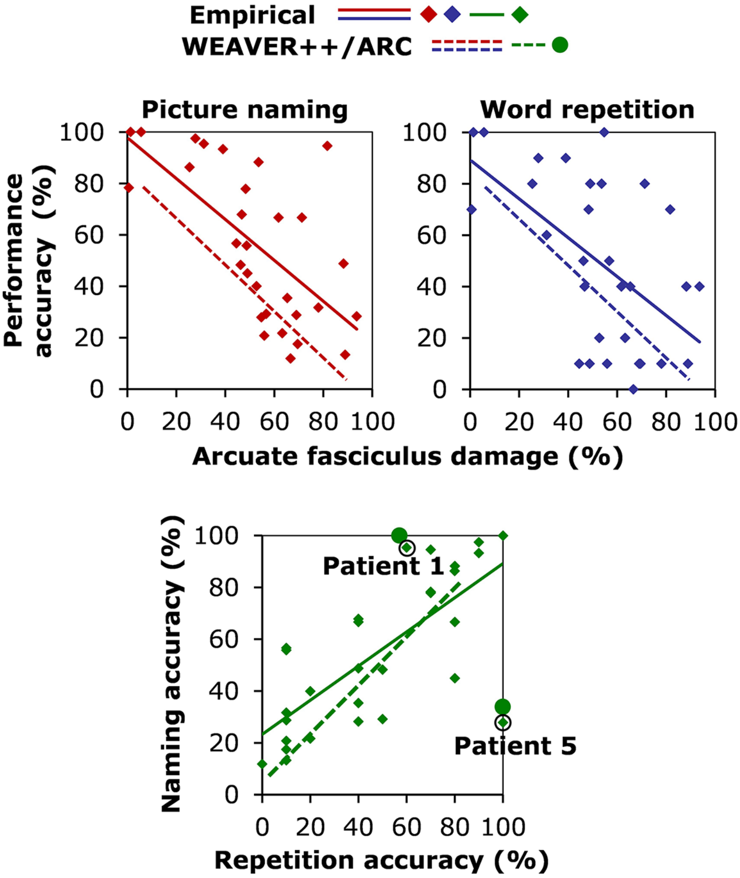


Fig. 11 **(top panels)** Picture naming and word repetition accuracy as a function of arcuate fasciculus damage and **(bottom panel)** the relationship between naming and repetition accuracy, as observed empirically in 30 patients (diamonds and solid trendlines in all panels) and in WEAVER++/ARC simulations (dashed trendlines in all panels). The circled data points for patients 1 and 5 in the bottom panel indicate a double dissociation and the solid circles denote WEAVER++/ARC simulations. The patient data are from Marchina et al. (2011)

Focal Symptoms in Neurodegeneration (Wernicke vs. Pick)

Language disorders can also result from neurodegeneration, as Pick (1892, 1904) showed. After studying medicine, Arnold Pick (1851–1924) worked most of his academic career in Prague (then in the Austro-Hungarian Empire), where he became professor of psychiatry in 1886. Wernicke (1874) claimed that brain atrophy cannot cause focal symptoms as observed in the classic aphasia syndromes due to stroke (illustrated in Fig. 2). However, Pick refuted this claim by documenting in increasing detail several cases with focal language symptoms caused by circumscribed atrophy of the left temporal lobe, frontal lobe, or both. To explain why the atrophy is circumscribed and leads to focal symptoms, Pick (1908) put forward a functional network account, which was soon forgotten.

Oblivious to Pick's seminal studies, research since the 1970s has rediscovered that focal behavioral symptoms may arise from neurodegeneration, and has obtained support for Pick's (1908) forgotten functional account of the distribution of atrophy and the focal symptoms (Roelofs, 2023b). For example, Warrington (1975) reported that neurodegeneration can specifically affect the content of declarative semantic memory, such as knowing that a cat is an animal and has a tail. It is now clear that this loss of conceptual knowledge is observed in frontotemporal degeneration, which typically gives rise to semantic and behavioral syndromes (Gorno-Tempini et al., 2011; Rascovsky et al., 2011). The conceptual deficit is primary in the semantic syndrome and secondary in the behavioral syndrome, which is characterized by personality and behavioral changes (e.g., apathy, disinhibition). In both syndromes, the loss of knowledge is modality general, as it affects not only the ability to recognize and name objects in seeing them, but also in other modalities, such as touch (Patterson et al., 2007). In the semantic syndrome, also called semantic dementia, neurodegeneration affects the anterior temporal lobes bilaterally, while in the behavioral syndrome the frontal lobes are affected and the anterior temporal lobes to a lesser extent. In both syndromes, picture naming is more impaired than word comprehension, and the impairment is worse in the semantic than in the behavioral syndrome.

Pick (1908) hypothesized that neurodegenerative diseases target and spread through functional networks, which are localized. While stroke destroys brain areas regardless of function, neurodegeneration is function specific. Pick's explanation was forgotten for almost a century, but recently reproposed and empirically supported by evidence from network-sensitive neuroimaging (e.g., Seeley et al., 2009), making it now one of the best explanations available.

Implementing Pick's ideas, the WEAVER++/ARC model was applied to the picture naming and word comprehension performance observed by Snowden and colleagues (2019) in a sample of 100 patients with frontotemporal degeneration, 30 diagnosed with semantic dementia and 70 with the behavioral syndrome. When naming, patients pronounced the name of the object seen in each picture (e.g., a cat, say "cat"). Word comprehension was assessed using a word-to-picture matching test with the same items as the naming test. Patients had to match a printed word (e.g., *cat*) by referring to one of four semantically related pictures (the cat, not the dog or another animal). Computer simulations were run to see if the model could account for the impairment assuming a reduction in the activation capacity of the conceptual network.

Figure 12 shows the model-predicted performance accuracy plotted against the empirically observed accuracy for each patient, indicated by dots, separately for semantic demen-

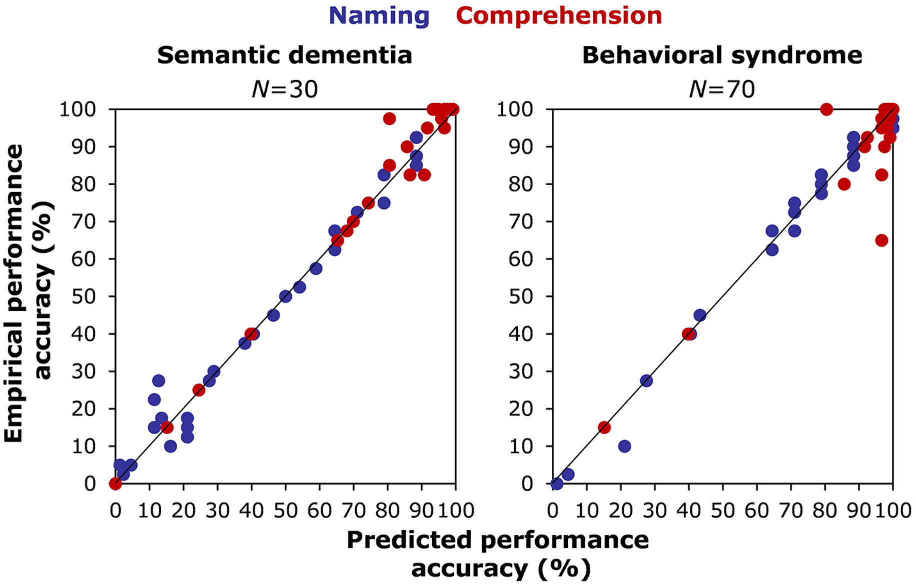


Fig. 12 Naming and comprehension accuracy for patients with **(left)** semantic dementia and **(right)** behavioral variant frontotemporal dementia. For each patient, denoted by dots, the performance accuracy predicted by WEAVER++/ARC is plotted against the empirical performance accuracy

tia and the behavioral syndrome. The simulations showed that the model accounts for 97% of the variance of the individual naming and comprehension accuracies of the 100 patients. The capacity reduction in the model for each of the patients correlated with the amount of neurodegeneration in the anterior temporal lobes, but not in other brain areas (see Roelofs, 2023a).

According to an alternative view, concepts consist of widely distributed modality-specific features without a central node, as advocated by Snowden et al. (2019) themselves and originally proposed by Wernicke (1874). For modality-general loss of conceptual knowledge to occur, several modality-specific representations or connections between them must be disrupted simultaneously. This alternative view of concepts does not explain why the impairment results from degeneration of the anterior temporal lobes and not from degeneration of widespread areas encoding modality-specific features or connections between them. Furthermore, it does not explain the evidence that the conceptual disorder in semantic dementia occurs across all input modalities (i.e., not only vision, but also touch and other modalities).

Summary

I have described how scientific evidence about the mind and its relationship to the brain began to accumulate two centuries ago, beginning with pioneering experiments by Flourens and later continued by the work of Broca, Helmholtz, and Donders. Neurocognitive modeling based on this evidence reached a high peak in the last quarter of the 19th century with the models of Wernicke and Wundt, which were presented as diagrams. The neurocognitive approach was largely abandoned after the First World War, but was revived after the Second. In the 1950s, researchers began developing cognitive models, such as Broadbent's attention model, which became neurocognitive in the 1960s. In particular, Geschwind revived Wernicke's model. Moreover, thanks to the rise of computer science and artificial intelligence, modeling became computational, with the models being realized as computer programs. Today's neurocognitive modeling, realized as diagrams or computer programs, revives the early modeling approach of Wernicke and Wundt in several respects. While predictions were derived and tested qualitatively from early models, today they can be derived mathematically in computer simulations and statistically evaluated for quantitative agreement with empirical data. I described how 20th century techniques have been used to test 19th century ideas about attentional control (Wundt), choice in go/no-go tasks (Donders vs. Wundt), the role of the arcuate fasciculus in speech repetition (Wernicke), and focal behavioral symptoms in neurodegeneration (Wernicke vs. Pick).

Author Contributions A.R. conducted the study, wrote the manuscript text, prepared all figures, and reviewed the manuscript.

Data Availability No datasets were generated or analysed during the current study.

Declarations

Competing Interests The authors declare no competing interests.

References

Ash, M. G. (1995). *Gestalt psychology in German culture, 1890–1967: Holism and the quest for objectivity*. Cambridge University Press.

Benjamin, L. T. (2024). *A brief history of modern psychology* (4th ed.). Blackwell Publishing.

Blumenthal, A. L. (1975). A reappraisal of Wilhelm Wundt. *American Psychologist*, *30*(11), 1081–1088. https://doi.org/10.1037/0003-066X.30.11.1081

Boring, E. G. (1950). *A history of experimental psychology* (2nd ed.). Appleton-Century-Crofts.

Broadbent, D. (1958). *Perception and communication*. Pergamon.

Broca, P. (1861). Remarques sur le siége de la faculté du langage articulé, suivies d'une observation d'aphémie (perte de la parole) [Remarks on the seat of the faculty of articulated language, following an observation of aphemia (loss of speech)]. *Bulletin de la Société Anatomique*, *6*, 330–357.

Brysbaert, M., & Rastle, K. (2021). *Historical and conceptual issues in psychology* (3rd ed.). Pearson.

Cattell, J. M. (1885). Ueber die Zeit der Erkennung und Benennung von Schriftzeichen, Bildern und Farben [On the time to recognize and name characters, pictures, and colors]. *Philosophische Studien*, *2*, 635–650. https://vlp.mpiwg-berlin.mpg.de/library/data/lit4142

Coltheart, M., Rastle, K., Perry, C., Langdon, R., & Ziegler, J. (2001). DRC: A dual route cascaded model of visual word recognition and reading aloud. *Psychological Review*, *108*(1), 204–256. https://doi.org/10.1037/0033-295X.108.1.204

Dejerine, J. (1895). *Anatomie des centres nerveux* [Anatomy of nerve centers]. Rueff. https://wellcomecollection.org/works/nmkhgfhe

Dell, G. S., Schwartz, M. F., Martin, N., Saffran, E. M., & Gagnon, D. A. (1997). Lexical access in aphasic and nonaphasic speakers. *Psychological Review*, *104*(4), 801–838. https://doi.org/10.1037/0033-295X.104.4.801

Donders, F. C. (1865). *Snelheid der werking in 't zenuwstelsel* [Speed of processing in the nerve system]. *University Museum Utrecht (Archive Do07.77)*. https://vlp.mpiwg-berlin.mpg.de/library/data/lit19826

Donders, F. C. (1868). Die Schnelligkeit psychischer Processe [The speed of mental processes]. *Archiv für Anatomie, Physiologie und wissenschaftliche Medicin*, *6*, 657–681. https://vlp.mpiwg-berlin.mpg.de/library/data/lit21223/

Eichenbaum, H. (2012). *The cognitive neuroscience of memory: An introduction* (2nd ed.). Oxford University Press.

Ferrier, D. (1886). *The functions of the brain* (2nd ed.). Smith, Elder, & Co. https://wellcomecollection.org/works/udbcq2f8

Flourens, P. (1824). *Recherches expérimentales sur les propriétés et les fonctions du système nerveux, dans les animaux vertébrés* [Experimental research on the properties and functions of the nervous system in vertebrate animals]. Crevot. https://wellcomecollection.org/works/b8edvn28

Fritsch, G., & Hitzig, E. (1870). Ueber die elektrische Erregbarkeit des Grosshirns [On the electrical excitability of the cerebrum]. *Archiv für Anatomie, Physiologie und wissenschaftliche Medicin*, *37*, 300–332.

Gall, F. J. (1798). Schreiben über seinen bereits geendigten Prodromus über die Verrichtungen des Gehirns der Menschen und der Thiere an Herrn Jos. Fr. von Retzer [Writing to Mr. Jos. Fr. von Retzer about his already completed preliminary publication on the functions of the brains of humans and animals]. *Der neue teutsche Merkur*, *3*(12), 311–332.

Geschwind, N. (1965). Disconnexion syndromes in animals and man: I. *Brain*, *88*(2), 237–294. https://doi.org/10.1093/brain/88.2.237

Geschwind, N. (1970). The organization of language and the brain. *Science*, *170*(3961), 940–944. https://doi.org/10.1126/science.170.3961.940

Geschwind, N. (1972). Language and the brain. *Scientific American*, *226*(4), 76–83. https://doi.org/10.1038/scientificamerican0472-76

Glaser, W. R., & Düngelhoff, F. J. (1984). The time course of picture-word interference. *Journal of Experimental Psychology: Human Perception and Performance*, *10*(5), 640–654. https://doi.org/10.1037/0096-1523.10.5.640

Glaser, M. O., & Glaser, W. R. (1982). Time course analysis of the Stroop phenomenon. *Journal of Experimental Psychology: Human Perception and Performance*, *8*(6), 875–894. https://doi.org/10.1037/0096-1523.8.6.875

Goldstein, K. (1939). *The organism: A holistic approach to biology derived from pathological data in man*. American book Company. https://archive.org/details/organismholistic00gold

Gorno-Tempini, M. L., Hillis, A. E., Weintraub, S., Kertesz, A., Mendez, M., Cappa, S. F., Ogar, J. M., Rohrer, J. D., Black, S., Boeve, B. F., Manes, F., Dronkers, N. F., Vandenberghe, R., Rascovsky, K., Patterson, K., Miller, B. L., Knopman, D. S., Hodges, J. R., Mesulam, M. M., & Grossman, M. (2011). Classification of primary progressive aphasia and its variants. *Neurology*, *76*(11), 1006–1014. https://doi.org/10.1212/WNL.0b013e31821103e6

Helmholtz, H. (1850). Ueber die Methoden, kleinste Zeittheile zu messen, und ihre Anwendung für physiologische Zwecke [About the methods of measuring the smallest parts of time and their application for physiological purposes]. *Koenigsberger naturwissenschaftliche Unterhaltungen*, *2*, 169–189. http://vlp.mpiwg-berlin.mpg.de/references?id=lit29040

Indefrey, P., & Levelt, W. J. (2004). The spatial and temporal signatures of word production components. *Cognition*, *92*(1–2), 101–144. https://doi.org/10.1016/j.cognition.2002.06.001

Janssen, N., Kessels, R. P. C., Mars, R. B., Llera, A., Beckmann, C. F., & Roelofs, A. (2023). Dissociating the functional roles of arcuate fasciculus subtracts in speech production. *Cerebral Cortex*, *33*(6), 2539–2547. https://doi.org/10.1093/cercor/bhac224

Jensen, O., & Mazaheri, A. (2010). Shaping functional architecture by oscillatory alpha activity: Gating by inhibition. *Frontiers in Human Neuroscience*, *4*, Article 186. https://doi.org/10.3389/fnhum.2010.00186

Jongman, S., Roelofs, A., & Lewis, A. (2020). Attention for speaking: Pre-stimulus motor-cortical alpha power predicts picture naming latencies. *Journal of Cognitive Neuroscience*, *32*, 747–761. https://doi.org/10.1162/jocn_a_01513

Kemmerer, D. (2022). *Cognitive neuroscience of language* (2nd ed.). Psychology Press.

Lachman, R., Lachman, J. L., & Butterfield, E. C. (1979). *Cognitive psychology and information processing: An introduction*. Erlbaum.

Leahey, T. H. (1979). Something old, something new: Attention in Wundt and modern cognitive psychology. *Journal of the History of the Behavioral Sciences*, *15*(3), 242–252. https://doi.org/10.1002/1520-6696(197907)15:3%3C242::aid-jhbs2300150305%3E3.0.co;2-o

Leahey, T. H. (2017). *A history of psychology: From antiquity to modernity* (8th ed.). Routledge.

Levelt, W. J. M. (1989). *Speaking: From intention to articulation*. The MIT Press.

Levelt, W. J. M. (2013). *A history of psycholinguistics: The pre-Chomskyan era*. Oxford University Press.

Levelt, W. J. M., Roelofs, A., & Meyer, A. S. (1999). A theory of lexical access in speech production. *Behavioral and Brain Sciences*, *22*(1), 1–38. https://doi.org/10.1017/S0140525X99001776

Lichtheim, L. (1885). On aphasia. *Brain*, *7*(4), 433–484.

Luce, R. D. (1986). *Response times: Their role in inferring elementary mental organization*. Oxford University Press.

Marchina, S., Zhu, L. L., Norton, A., Zipse, L., Wan, C. Y., & Schlaug, G. (2011). Impairment of speech production predicted by lesion load of the left arcuate fasciculus. *Stroke*, *42*(8), 2251–2256. https://doi.org/10.1161/STROKEAHA.110.606103

McClelland, J. L., & Rumelhart, D. E. (1981). An interactive activation model of context effects in letter perception: I. An account of basic findings. *Psychological Review*, *88*(5), 375–407. https://doi.org/10.1037/0033-295X.88.5.375

Meyer, D. E., & Kornblum, S. (1993). (Eds.). *Attention and performance XIV: Synergies in experimental psychology, artificial intelligence, and cognitive neuroscience*. The MIT Press.

Müller, J. (1835). *Handbuch der Physiologie des Menschen für Vorlesungen* [Handbook of human physiology for lectures] (2nd ed., Vol. 1). Hölscher. https://wellcomecollection.org/works/xa7t8w6p/items?canvas=7

Nicolas, S., & Murray, D. J. (1999). Théodule Ribot (1839–1916), founder of French psychology: A biographical introduction. *History of Psychology*, *2*(4), 277–301. https://doi.org/10.1037/1093-4510.2.4.277

Patterson, K., Nestor, P. J., & Rogers, T. T. (2007). Where do you know what you know? The representation of semantic knowledge in the human brain. *Nature Reviews: Neuroscience*, *8*(12), 976–987. https://doi.org/10.1038/nrn2277

Petersen, S. E., Fox, P. T., Posner, M. I., Mintun, M., & Raichle, M. E. (1988). Positron emission tomographic studies of the cortical anatomy of single-word processing. *Nature*, *331*(6157), 585–589. https://doi.org/10.1038/331585a0

Piai, V., Roelofs, A., Rommers, J., Dahlslätt, K., & Maris, E. (2015). Withholding planned speech is reflected in synchronized beta-band oscillations. *Frontiers in Human Neuroscience*, *9*, 549. https://doi.org/10.3389/fnhum.2015.00549

Pick, A. (1892). Ueber die Beziehungen der senilen Hirnatrophie zur Aphasie [On the relationships between senile brain atrophy and aphasia]. *Prager medicinische Wochenschrift*, *17*(16), 165–167.

Pick, A. (1904). Zur Symptomatologie der linksseitigen Schläfenlappenatrophie [On the symptomatology of left-sided temporal lobe atrophy]. *Monatsschrift für Psychiatrie und Neurologie*, *16*, 378–388. https://doi.org/10.1159/000219215

Pick, A. (1908). Die umschriebene senile Hirnatrophie als Gegenstand klinischer und anatomischer Forschung [Circumscribed senile cerebral atrophy as an object of clinical and anatomical research]. In A. Pick (Ed.), *Arbeiten aus der deutschen psychiatrischen Universitäts-Klinik in Prag* (pp. 20–28). Karger.

Posner, M. I., & Raichle, M. E. (1994). *Images of mind*. Scientific American Library.

Raichle, M. E. (1998). Behind the scenes of functional brain imaging: A historical and physiological perspective. *Proceedings of the National Academy of Sciences of the United States of America*, *95*(3), 765–772. https://doi.org/10.1073/pnas.95.3.765

Rascovsky, K., Hodges, J. R., Knopman, D., Mendez, M. F., Kramer, J. H., Neuhaus, J., van Swieten, J. C., Seelaar, H., Dopper, E. G., Onyike, C. U., Hillis, A. E., Josephs, K. A., Boeve, B. F., Kertesz, A., Seeley, W. W., Rankin, K. P., Johnson, J. K., Gorno-Tempini, M. L., Rosen, H., Prioleau-Latham, C. E., & Miller, B. L. (2011). Sensitivity of revised diagnostic criteria for the behavioural variant of frontotemporal dementia. *Brain*, *134*(9), 2456–2477. https://doi.org/10.1093/brain/awr179

Ribot, T. (1885). *La psychologie allemande contemporaine (école expérimentale)* (2nd ed.) [Contemporary German psychology. Experimental school]. Alcan. https://archive.org/details/lapsychologieal00ribogoog

Ribot, T. (1909). *La psychologie allemande contemporaine (école expérimentale)* (7th ed.) [Contemporary German psychology. Experimental school]. Alcan. https://archive.org/details/lapsychologieal00ribo

Roelofs, A. (1992). A spreading-activation theory of lemma retrieval in speaking. *Cognition*, *42*(1–3), 107–142. https://doi.org/10.1016/0010-0277(92)90041-F

Roelofs, A. (1997). The WEAVER model of word-form encoding in speech production. *Cognition*, *64*(3), 249–284. https://doi.org/10.1016/S0010-0277(97)00027-9

Roelofs, A. (2003). Goal-referenced selection of verbal action: Modeling attentional control in the Stroop task. *Psychological Review*, *110*(1), 88–125. https://doi.org/10.1037/0033-295X.110.1.88

Roelofs, A. (2008). Dynamics of the attentional control of word retrieval: Analyses of response time distributions. *Journal of Experimental Psychology: General*, *137*(2), 303–323. https://doi.org/10.1037/0096-3445.137.2.303

Roelofs, A. (2014). A dorsal-pathway account of aphasic language production: The WEAVER++/ARC model. *Cortex*, *59*, 33–48. https://doi.org/10.1016/j.cortex.2014.07.001

Roelofs, A. (2018). One hundred fifty years after Donders: Insights from unpublished data, a replication, and modeling of his reaction times. *Acta Psychologica*, *191*, 228–233. https://doi.org/10.1016/j.actpsy.2018.10.002

Roelofs, A. (2021). How attention controls naming: Lessons from Wundt 2.0. *Journal of Experimental Psychology: General*, *150*(10), 1927–1955. https://doi.org/10.1037/xge0001030

Roelofs, A. (2022). A neurocognitive computational account of word production, comprehension, and repetition in primary progressive aphasia. *Brain and Language*, *227*, 105094. https://doi.org/10.1016/j.bandl.2022.105094

Roelofs, A. (2023a). Word production and comprehension in frontotemporal degeneration: A neurocognitive computational Pickian account. *Cortex*, *163*, 42–56. https://doi.org/10.1016/j.cortex.2023.03.003

Roelofs, A. (2023b). Cerebral atrophy as a cause of aphasia: From Pick to the modern era. *Cortex*, *165*, 101–118. https://doi.org/10.1016/j.cortex.2023.05.004

Roelofs, A. (2024). Wernicke's functional neuroanatomy model of language turns 150: What became of its psychological reflex arcs? *Brain Structure and Function*. https://doi.org/10.1007/s00429-024-02785-5

Roelofs, A., & Piai, V. (2017). Distributional analysis of semantic interference in picture naming. *Quarterly Journal of Experimental Psychology*, *70*(4), 782–792. https://doi.org/10.1080/17470218.2016.1165264

San José, A., Roelofs, A., & Meyer, A. S. (2021). Modeling the distributional dynamics of attention and semantic interference in word production. *Cognition*, *211*, Article 104636. https://doi.org/10.1016/j.cognition.2021.104636.

Scaltritti, M., Navarrete, E., & Peressotti, F. (2015). Distributional analyses in the picture-word interference paradigm: Exploring the semantic interference and the distractor frequency effects. *Quarterly Journal of Experimental Psychology*, *68*(7), 1348–1369. https://doi.org/10.1080/17470218.2014.981196

Seeley, W. W., Crawford, R. K., Zhou, J., Miller, B. L., & Greicius, M. D. (2009). Neurodegenerative diseases target large-scale human brain networks. *Neuron*, *62*(1), 42–52. https://doi.org/10.1016/j.neuron.2009.03.024

Seidenberg, M. S., & McClelland, J. L. (1989). A distributed, developmental model of visual word recognition and naming. *Psychological Review*, *96*(4), 523–568. https://doi.org/10.1037/0033-295X.96.4.523

Selnes, O. A., Van Zijl, P. C., Barker, P. B., Hillis, A. E., & Mori, S. (2002). MR diffusion tensor imaging documented arcuate fasciculus lesion in a patient with normal repetition performance. *Aphasiology*, *16*(9), 897–902.

Shallice, T., & Cooper, R. (2011). *The organisation of mind*. Oxford University Press.

Snowden, J. S., Harris, J. M., Saxon, J. A., Thompson, J. C., Richardson, A. M., Jones, M., & Kobylecki, C. (2019). Naming and conceptual understanding in frontotemporal dementia. *Cortex*, *120*, 22–35. https://doi.org/10.1016/j.cortex.2019.04.027

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, *18*(6), 643–662. https://doi.org/10.1037/h0054651

Tesak, J., & Code, C. (2008). *Milestones in the history of aphasia: Theories and protagonists*. Psychology Press.

Ueno, T., Saito, S., Rogers, T. T., & Lambon Ralph, M. A. (2011). Lichtheim 2: Synthesizing aphasia and the neural basis of language in a neurocomputational model of the dual dorsal-ventral language pathways. *Neuron*, *72*(2), 385–396. https://doi.org/10.1016/j.neuron.2011.09.013

Von Monakow, C. (1897). *Gehirnpathologie* [Brain pathology]. Alfred Hölder. https://wellcomecollection.org/works/k5s28vpx

Warrington, E. K. (1975). The selective impairment of semantic memory. *The Quarterly Journal of Experimental Psychology*, *27*(4), 635–657. https://doi.org/10.1080/14640747508400525

Wernicke, C. (1874). *Der aphasische Symptomencomplex: Eine psychologische Studie auf anatomischer Basis* [The aphasic symptom complex: A psychological study on anatomical basis]. Cohn und Weigert.

Wernicke, C. (1886). Die neueren Arbeiten über Aphasie [The new works on aphasia]. *Fortschritte der Medizin*, *4*(11), 371–377.

Wernicke, C. (1893). *Gesammelte Aufsätze und kritische Referate zur Pathologie des Nervensystems* [Collected essays and critical papers on the pathology of the nervous system]. Verlag von Fischer's medicinischer Buchhandlung.

Wernicke, C. (1906). Der aphasische Symptomencomplex [The aphasic symptom complex]. *Die deutsche Klinik am Eingange des zwanzigsten Jahrhunderts*, *6*(1), 487–556.

Wundt, W. (1874). *Grundzüge der physiologischen Psychologie* [Principles of physiological psychology]. Engelmann. http://vlp.mpiwg-berlin.mpg.de/references?id=lit46

Wundt, W. (1896). *Grundriss der Psychologie* [Outlines of psychology]. Engelmann. http://vlp.mpiwg-berlin.mpg.de/references?id=lit779
Wundt, W. (1900). *Die Sprache* [Language]. Engelmann.
Wundt, W. (1902). *Grundzüge der physiologischen Psychologie* [Principles of physiological psychology] (5th ed., Vol. 1). Engelmann. http://vlp.mpiwg-berlin.mpg.de/references?id=lit806
Wundt, W. (1903). *Grundzüge der physiologischen Psychologie* [Principles of physiological psychology] (5th ed., Vol. 3). Engelmann.