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Journal of Neuroscience and Neurological Surgery
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Review Article

Agraphia: definition, clinical contexts, neurobiological profiles and clinical treatments

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Received date: August 17, 2020; Accepted date: August 31, 2020; Published date: September 07, 2020.

Citation: Perrotta G., (2020) Agraphia: definition, clinical contexts, neurobiological profiles and clinical treatments. J Neuroscience and Neurological Surgery. 6(4); **DOI:10.31579/2578-8868/134**

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Abstract

Starting from the general concept of Agraphia, the present work focuses on the clinical, neurobiological, and functional aspects of the morbid condition, suggesting a multidimensional treatment between physiotherapy, psychotherapy, and rehabilitation exercises for lost skills.

Keywords: Agraphia; neurobiological

Contents of the manuscript:

L'agraphia is an acquired neurological and neuropsychological disorder [1] that causes a loss in the ability to communicate through writing and is often associated with another neuropsychological disorder, in the area of the mechanisms involved in the form of writing (whether they are linked to language and motor deficits), such as alexia, aphasia, dysarthria, agnosia, and apraxia. [2] In summary, agraphia consists in the difficulty in producing a previously learned and known written language, with damage to various related cognitive processes, such as language processing, spelling, visual perception, visual orientation for graphic symbols, motor planning and motor control of writing). [3]

In 1553, within Thomas Wilson's book, entitled "Arte of Rhetorique", there is a trace of the first description of acquired agraphia, but only in the second half of the 19th century, the loss of the ability to produce the known written language and previously learned finally receives adequate clinical attention. In this period the idea arises that certain cortical lesions of the brain can involve a dissociation between written and spoken language, as well as altering both reading and writing skills, such as the studies of Broca and Wernicke. [12-13]

During the 1850s, Armand Trousseau and John Hughlings Jackson maintained the opinion that the same linguistic deficit occurs both in writing, both in speech and in reading, while in 1856 Louis-Victor Marcé argued the opposite: written and spoken language are independent of each other. He discovers that in many patients with language disorders, there is an impairment of both spoken language and writing. In these same patients, however, the recovery of an adequate written and spoken linguistic production does not always take place in parallel, suggesting that these two modes of expression are independent. [12-13]

Louis-Victor Marcé comes to believe that the ability to write not only involves the control of motor skills, but also the memory of signs and their meaning. Even Benedikt in 1865 comes to suggest that the areas of the brain that control writing and spoken language may have different anatomical locations, coining for the first time the clinical term, then followed in 1867 by William Ogle who laid the foundations for the first classifications nosographic, distinguishing the different dissociation models that can be found between written and spoken language. He showed that some patients with writing deficits were perfectly capable of copying the written letters, but they struggled or found it completely impossible to organize those same letters to form words of complete meaning. Ogle knew that aphasia and agraphia often occur together, but he also stressed that the deficit of the two different types of language (spoken and written) could vary in type and severity. Ogle himself distinguished between two different types of agraphia: in subjects suffering from mnemonic agraphy the written letters appeared well-formed, but often used incorrectly or completely randomly, and the words could indifferently take the place of each other; in patients with atraphic agraphia, on the other hand, the letters appeared poorly formed, intelligible and sometimes completely unrecognizable. Although Ogle's observations contributed significantly to scientific progress towards understanding writing disorders, there was still no documented case of pure agraphia. [12-13]

In 1884, Albert Pitres, strongly influenced by the modular approach to the memory of Théodule-Armand Ribot, made an important discovery by contributing to the publication of the first report of a case of pure agraphy. Pitres' reading and writing models consisted of three main components: visual (the memory for the letters and how the letters form syllables and words), auditory (the memory for the sounds represented by each letter),

and motor (motor-graphic memory for letters). Based on this three-dimensional model, Pitres proposed his classification of the agraphia: a) agraphia from blindness for words: inability to copy a model, but the individual can write spontaneously and under dictation; b) agraphia from deafness for words: the inability to write under dictation, but the subject can copy a model and write spontaneously; c) motor agraphia: no ability to write, but the individual has no language deficit. [12-13]

The agraphia [1], today, is classified according to two precise forms:

1) "central" ("aphasic"), which includes the lexical form, the phonological form, the deep form and the semantic writing;

2) "peripheral" ("non-aphasic"), which includes the allographic, apraxic, motor, hemianoptic, and afferent forms.

"Central agraphia" [4-7] occurs when there is a deficit of spoken language and a deficit of the various motor and visualization skills involved in writing, distinguished in fluent and not fluent. In the "agraphia with fluent aphasia" (for example receptive aphasia) they normally write well-formed letters, but are unable to write significant words; those who have "agraphia with non-fluent aphasia", do not have an impairment of the spoken language and can write short sentences, but their writing is difficult to read, requires great physical effort, lacks correct syntax and often characterized by poor spelling (e.g. expressive or modifying aphasia):

a) the "deep Agraphia" affects the phonological ability and the spelling memory of individuals and is often the result of an injury involving the left parietal region (supramarginal or insula convolution). The subjects do not remember what the words look like even if they are spelled correctly, nor do they have any idea what sound determines the correct spelling. Individuals usually rely on their damaged spelling memory for writing, which results in frequent errors, usually semantic. Individuals have more difficulty with abstract concepts and uncommon words. Reading and spoken language is also often compromised. Deep agraphia involves damage to the same areas of the brain (as happens with lexical agraphy), which is associated with some damage to the perisylvian linguistic areas as well. Major and massive damage to the left hemisphere can result in a global agraphia.

b) the "global agraphia" instead also damages the spelling memory of a subject, even if to a greater extent than the deep writing. In global agraphia, spelling knowledge is lost to such an extent that the individual can only write a few significant words, or he cannot write any words. Reading and spoken language are significantly impaired.

c) "Gerstmann's syndrome agraphia" is the loss of value of written language production associated with the following structural symptoms: difficulty in discriminating between one's fingers, difficulty in distinguishing the right from the left, difficulty in making calculations. All four of these symptoms result from lesions of a nerve pathway. Gerstmann syndrome can also be associated with alexia and mild aphasia and is caused by a dominant parietal lobe lesion (usually the left one), normally an angular convolution injury.

d) the "lexical and structural agraphia" is caused by damage to the spelling memory; affected individuals cannot view the spelling of a word, even if they retain the ability to pronounce it. This altered spelling memory can result in the loss or degradation of knowledge or simply the inability to access it efficiently. There is a regularity effect associated with lexical letter-writing as individuals are less likely to spell words correctly without regular and predictable spelling. Also, the ability to write a word tends to be less compromised for commonly used words. People also have difficulties with homophonic terms. The linguistic competence in terms of grammar and writing of the sentence tends to be preserved. Lexical agraphia is associated with damage to the left angular convolution and/or posterior temporal cortex. The damage is typically posterior and less than in the Perisylvian language areas.

e) "phonological agraphia" is the opposite of lexical writing because the ability to pronounce words is compromised, but the spelling memory of words can be completely intact. Often it is associated with a lexical effect as there is a difference in the ability to write words with meaning compared to words that are devoid of them. Individuals with this form of agraphia depend on their spelling memory. Moreover, for these subjects, it is often difficult to access more abstract words, without a strong semantic representation (for example, they find it much more difficult to make explicit prepositions than names that represent an object). This type of agraphia is linked to damage in the areas of the brain involved in the phonological processing capacity (the sound of words), in particular the linguistic areas around the Silvian fissure, such as the Broca area, the Wernicke area, and the supramarginal convolution.

f) the "pure agraphia" finally consists in the loss of value of the written language production, without any other linguistic or cognitive disturbance.

"Peripheral agraphia" [3-4, 8-9] occurs when there is damage to the various motor and visualization skills that are necessarily involved in the writing activity:

a) "apraxic agraphia" is the loss of value of written language production associated with a disorder of the motor system. This type of disorder results in the tiring and slow formation of distorted, incomplete and/or inaccurate letters and words. Although the letters and words that are written by these subjects are often so poorly formed that they are almost illegible, the ability to spell out loud is often preserved. This pathological form is caused specifically by a loss of motor planning specialized in letter formation and not by a dysfunction affecting the writing hand. It can present with or without ideomotor apraxia, but other neurological conditions are often associated, such as paralysis, Huntington's chorea, Parkinson's disease (micrograph), and dystonia (writer's cramp). Apraxic ataphia with ideomotor apraxia is usually caused by damage to the upper parietal lobe (in which the skills and graphomotor planning are stored) or the premotor cortex (where the planning is converted into motor commands). Also, some individuals with cerebellar lesions (more typically associated with non-apraxic motor dysfunction) may develop apraxic agraphia. Apraxic ataphia without ideomotor apraxia can be caused by damage to one of the parietal lobes, the dominant frontal lobe, or to the dominant thalamus.

b) "hysterical agraphia" is the loss of value of written language production caused by a conversion disorder.

c) "reiterative agraphia" is found in individuals who repeat letters, words or sentences, in the production of written language, an abnormal number of times. Preservation, paragraph, and ultrasound itself (understood as an eco-phenomenon of automatic imitative action, without an explicit awareness) are examples of reiterative agraphy.

d) “visuospatial agraphia” is the loss of value of written linguistic production defined by a tendency to neglect a portion (often an entire side) of the page, oblique lines upwards or downwards, leaving an abnormal spacing between letters, syllables and words. The orientation

and correct sequence of writing also appear to be compromised. It is often associated with a lack of attention to the left hemispace, difficulties in the construction or assembly of objects, as well as other spatial difficulties. Visuospatial agraphia generally is associated with a pathology of the right hemisphere. Damage to the right frontal region of the brain can cause multiple types of motor deficits, while damage to the posterior part of the right hemisphere mainly leads to spatial defects in writing.

Etiologically, agraphia is the consequence of a series of morbid neurological (vascular, infectious, traumatic lesions, dementia) or systemic (autoimmune and tumor) conditions that neurobiologically interfere in whole or in part with twelve brain areas associated with writing [20-27]:

a) the upper left frontal area, consisting of the middle frontal convolution (1) and the upper frontal sulcus (2), the upper left parietal area (3), composed of the lower parietal lobule, the upper parietal lobule and the intraparietal sulcus and finally from the primary motor cortex and the somatosensory cortex (4).
b) the associative areas: the right anterior cerebellum (5), the left posterior nucleus of the thalamus (6), the lower left frontal convolution (7), the right posterior cerebellum (8), the upper right frontal cortex (9), the right lower parietal lobule (10), the left fusiform convolution (11) and the left putamen (12).

The specific type of agraphia, resulting from trauma and consequent brain injury, will, therefore, depend on which area of the brain is damaged.

Very often then it is observed the writing in association with Alzheimer's disease. Writing disorders can be an early manifestation of Alzheimer's disease. The first indicative sign of involvement of the writing ability is the selective syntactic simplification of the handwriting of these patients. These patients begin to write with fewer descriptions, less detail, and complexity. Other indicators may also emerge, such as some grammatical errors. As Alzheimer's disease develops, different types of agraphia may emerge and develop. In the initial stages of the disorder, the subjects affected by it show signs of allographic agraphia and apraxic agraphy. The allographic ethic in subjects with Alzheimer's dementia is represented by the tendency to mix uppercase and lowercase in words. Egyptian Ataxia is represented by the tendency of patients to build poor, poorly constructed, or frankly illegible letters or a continuous repetition of the same traits of the letters. As the disease progresses, the severity of the writing process also progresses. Patients begin to manifest spatial agraphia, that is, the inability to write on a straight horizontal line, with the tendency to leave unnecessary spaces between letters and words. A connection between Alzheimer's disease and agraphia is the role of memory in normal writing skills. Those who can spell normally have access to a lexical spelling system that is based on a whole word: when it works correctly, it allows you to recall the spelling of a complete word, not of single letters or sounds. This system also uses an internal memory archive, where the spelling of hundreds of words is kept. This system is called a graphemic elaborate lexicon and is aptly named about the graphemic buffer, which represents the short-term memory ring for many of the functions involved in writing. When the spelling system cannot be used, for example because we are dealing with unknown words, non-words, or words whose spelling is not recognized, some people can use the phonological process called "sub-lexical spelling system". The latter system is used to probe a word and be able to write it. In individuals with Alzheimer's disease, the memory stores that are used for everyday writing are lost as the disease progresses. [14]

From a rehabilitative point of view, agraphia cannot be treated directly, but individuals affected by it can be rehabilitated to regain some of their previous writing skills: in the absence of neurological symptoms of another nature, such as amnesia [15], epilepsies [16] or dementia [14, 17-18], the techniques of memorization of keywords, the use of technology for word processing, manual writing, artistic and abstract drawing and physiotherapy are extremely functional, combining a targeted psychological therapy for cognitive-behavioral or strategic support [19].

References

1. Perrotta G., *Psicologia clinica*, Luxco Ed., 1th ed., 2019.
2. *De Smet H.J. et all., Cerebellar-induced apraxic agraphia: a review and three new cases, in Brain Cogn, vol. 76, n. 3, Ago. 2011, pp. 424-34,* doi:10.1016/j.bandc.2010.12.006, PMID 21507544.
3. *Sinanović O. et all., Post-stroke language disorders, in Acta Clin Croat, vol. 50, n. 1, Mar. 2011, pp. 79-94,* PMID 22034787.
4. *Lorch M., Written language production disorders: historical and recent perspectives., in Curr Neurol Neurosci Rep, vol. 13, n. 8, Ago. 2013, p. 369,* doi:10.1007/s11910-013-0369-9, PMID 23793932.
5. *Mariën P. et all., Apraxic dysgraphia in a 15-year-old left-handed patient: disruption of the cerebello-cerebral network involved in the planning and execution of graphomotor movements, in Cerebellum, vol. 12, n. 1, Feb. 2013, pp. 131-9,* doi:10.1007/s12311-012-0395-1, PMID 22752975.
6. *Rusconi E. et all., The enigma of Gerstmann's syndrome revisited: a telling tale of the vicissitudes of neuropsychology. In Brain, vol. 133, Pt 2, Feb. 2010, pp. 320-32,* doi: 10.1093/brain/awp281, PMID 19903731.
7. *Zukic S. et all., Gerstmann'S syndrome in acute stroke patients, in Acta Inform Med, vol. 20, n. 4, Dic. 2012, pp. 242-3,* doi:10.5455/aim.2012.20.242-243, PMID 23378691.
8. *Grigorenko E.L. et all., Writing: a mosaic of new perspectives, Psychology Press, 2012,* ISBN 978-1-84872-812-7.
9. *Ardila A., Rosselli M., Spatial agraphia, in Brain Cogn, vol. 22, n. 2, Lug. 1993, pp. 137-47,* doi: 10.1006/brcg.1993.1029, PMID 8373568.
10. *Planton S. et all., The handwriting brain: a meta-analysis of neuroimaging studies of motor versus orthographic processes., in Cortex, vol. 49, n. 10, pp. 2772-87,* doi:10.1016/j.cortex.2013.05.011, PMID 23831432.
11. *Beeson P.M., Remediation of written language, in Top Stroke Rehabil, vol. 11, n. 1, 2004, pp. 37-48,* PMID 14872398.
12. *Arcolini I. & Zardini G., I disturbi di apprendimento della lettura e della scrittura, Franco Angeli, 2012.*
13. *Bellato F., Fornaciari G. & Giuffrà V., Storia della medicina e della psicologia, Felici Ed., 2012.*
14. Perrotta G., *Alzheimer's disease: definition, contexts, neural correlates, strategies and clinical approaches*. Journal of

Aging Studies and Therapies, J Aging Stud Ther, 1(1), 8 pages, doi.org/10.16966/jast.104, August 2019.

15. Perrotta G., *Amnesia: definition, main models, classifications, neurobiological profiles and clinical treatments*. Review article, Author. Archives of Depression and Anxiety, Arch Depress Anxiety, 6(1): 037-044, 8 pages, doi: http://dx.doi.org/10.17352/2455-5460.000050, June 2020.
16. Perrotta G., *Epilepsy: from pediatric to adulthood. Definition, classifications, neurobiological profiles and clinical treatments*. Review article, Author. Archives of Depression and Anxiety, J Neurol Neurol Sci Disord 6(1): 014-029, 16 pages, doi: https://dx.doi.org/10.17352/jnnsd.000039, July 2020.
17. Perrotta G., *General overview of "human dementia diseases": definitions, classifications, neurobiological profiles and clinical treatments*. Review article, Author. Journal of Gerontology & Geriatrics Studies, Gerontol & Geriatric stud. 6(1). GGS.000626. 2020, 8 pages, doi: 10.31031/GGS.2020.06.000626, May 2020.
18. Perrotta G., *Parkinson's disorder: definition, contexts, neural correlates, strategies and clinical approaches*. Journal of Neuroscience and Neurological Surgery. J Neurosci Neurol Surg 4(5), doi: 10.31579/2578-8868/079, 7 pages, August 2019.
19. Perrotta G., *The strategic clinical model in psychotherapy: theoretical and practical profiles*. Journal of Addiction and Adolescent Behaviour, j Addi Adol Beh, 3(1), 5 pages, doi: 10.31579-007/2688-7517/016, February 2020.
20. Estañol B. et all., *Alexia Without Agraphia Due to the Lesion in the Right Occipital Lobe in a Right-Handed Man. Detection of Hemispheric Lateralization of Handedness and Language in a Right-Handed Patient*. In Rev Neurol, 1999 Feb, 1-15; 28(3): 243-5. PMID: 10714287.
21. Petrillo S. et all., *Neurolinguistic Analysis of a Case of Pure Agraphia*, In Riv Neurol. Nov-Dec 1990;60(6):219-20. PMID: 2100045.
22. Leiguarda R., Carrea R., *Alexia Without Agraphia (Clinicotomographic Correlation)*. In Acta Neurol. Latinoam1977;23(1-4):89-101. PMID: 582231.
23. Piccirilli M., *Alexia Without Agraphia, Optical Ataxia and Simultanagnosia*. Description of a Case. In Riv Neurol. Nov-Dec 1982; 52(6): 392-9. PMID: 7156793.
24. Sfondrini E., Cazzola G., *Clinical Findings in the Study of Agraphia*. In Rass Neuropsichiatr, 1958;12(1):75-7. PMID: 13554934.
25. Da Silva A.B., *Alexia Without Agraphia: A Clinico-Tomographic Study*. In Arq Neuropsiquiatr, 1986 Sep; 44(3): 267-74, doi: 10.1590/s0004-282x1986000300007. PMID: 3593036.
26. Jović N. et all., *Dysconnection Syndrome in a Boy With Tumorous Lesion of Corpus Callosum*. In Srp Arh Celok Lek, Jan-Feb 1995;123(1-2):38-41. PMID: 17974475.
27. Marti-Massó J.F. et all., *Alexia Without Agraphia, Right Homonymous Hemianopsia, and Color Anomia. Report of Four Cases With Location of the Lesion by Computerized Axial Tomography of the Brain (Author's Transl)*. In Med Clin (Barc), 1981 Jun 25;77(2):64-8. PMID: 7321628.

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