

Thi-Qar University
College of Medicine DEPARTMENT
OF MEDICINE
2021-2022



Ministry of Higher Education and Scientific Research



PREVALENCE OF APHASIA IN STROKE PATIENTS IN THI_QAR

DONE BY:

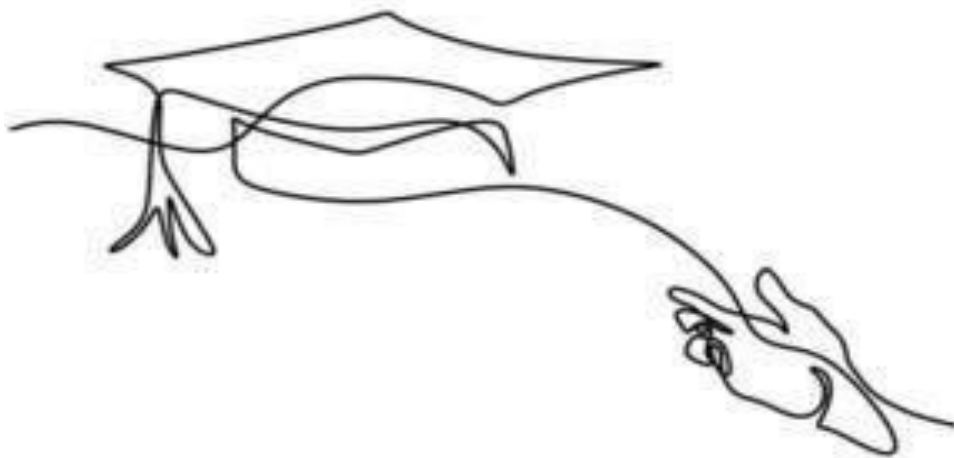
ZAINAB HASAN KARAM

SAEAH KHUDHAIR NAEEM

SUPERVISOR: DR. HAZIM ALI MARRAH

Dedication:

- To our families who was first supportive to us every time.
- TO my father who help me a Lot to complete my graduation project:
assistant lecture "Hassan karam Redhewy “
- TO THIQAR MEDICAL COLLAGE.
- TO our supervisor dr. Hazem Ali Marah
MBChB. -F.I.B.M.S. (Neurology)
- TO our doctor for their effort to teach us a true medicine make us effective doctors .
- TO the administration of Nasiriyah Teaching Hospital.



Stroke sometimes called a brain attack, **occurs when** something blocks blood supply to part of the brain or when a blood vessel in the brain bursts. In either case, parts of the brain become damaged or die. A stroke can cause lasting brain damage, long-term disability, or even death.

A **COMPLETED STROKE** is defined as a neurologic deficit due to occlusive cerebrovascular disease which may persist for hours or days. Cerebral infarction may be presumed if the neural deficit lasts for several days

What are the types of stroke?

There are two types of stroke:

- [Ischemic stroke](#).
- [Hemorrhagic stroke](#).

A [transient ischemic attack \(TIA\)](#) is sometimes called a “mini-stroke.” It is different from the major types of stroke, because blood flow to the brain is blocked for only a short time—usually no more than 5 minutes.¹

Ischemic stroke

Most strokes are ischemic strokes.² An ischemic stroke occurs when blood clots or other particles block the blood vessels to the brain.

Fatty deposits called plaque can also cause blockages by building up in the blood vessels.

Hemorrhagic stroke

A hemorrhagic stroke happens when an artery in the brain leaks blood or ruptures (breaks open). The leaked blood puts too much pressure on brain cells, which damages them.

High blood pressure and aneurysms—balloon-like bulges in an artery that can stretch and burst—are examples of conditions that can cause a hemorrhagic stroke.

Transient ischemic attack (TIA or “mini-stroke”)

:

- Blood clots (deep vein thrombosis or pulmonary embolism)
- Brain swelling.
- Seizures.
- Memory loss.
- Vision and hearing problems.
- Muscle weakness.
- Bed sores.

Aphasia

Definition of aphasia represents an acquired central disorder of language that impairs a person's ability to understand or/and produce spoken language, often associated with impairment in reading (alexia) and

writing (agraphia). Aphasia may supplementary affect the person's ability to use musical notation, mathematical operations, etc.; in consequence, the aphasic may present difficulties to generate and use symbol systems. Aphasia is different from a peripheral (sensory-motor) disorder of language that may mimic aphasia (such as weakness of the muscles of articulation). In the same time, it is an acquired phenomenon that appears after the language has already been learned [1, 2, 3, 4].

Introduction

Aphasia is one of the most common and also frustrating disabilities secondary to stroke; over 25% of the patients who suffer an acute ischemic stroke are dealing with this complex syndrome in their evolution. It is also

**APHASIA is
when your brain holds
your words hostage.**



aphasia
NATIONAL
APHASIA
ASSOCIATION

considered an important stroke severity marker, being associated with a higher risk of mortality, poor functional prognosis, and augmented risk of vascular dementia. This syndrome is a real challenge not only for the patients or their relatives but also for the specialists (neurologists, speech therapists, psychologists, and physiotherapists) involved in the diagnosis and treatment of those patients.

The assessment of aphasias in clinical practice is based on classical analysis of oral production and comprehension. The language disturbances are frequently combined into aphasic syndromes (nonfluent/fluent aphasias), which are constituents of different vascular syndromes, being accompanied by motor deficit of the right limbs or visual deficit (hemianopia). The main determinant of the type of vascular aphasia is the infarct location (especially left middle cerebral artery territory).

Types of aphasic syndromes

The main determinants of the type of aphasias are *the site and size of the lesion* [2, 11].

According to site"array affected" :

Arteries	Clinical symptoms
Anterior cerebral artery	Behavior alterations (frontal lobus stroke) Contralateral hemiparesis, more severe in lower limb Contralateral psychomotor disorders Contralateral sensorial losses Functional alterations in bladder and anal sphincter Hemiplegia with crural predominance Homolateral ataxia in the arm Mental manifestations
Middle cerebral artery	Alexia Aphasia (dominant hemisphere lesion) Apraxia Atherosclerosis Contralateral hemiplegia and/or hemiparesis Homonymous hemianopsia
Posterior cerebral artery	Anton syndrome Ataxia Cortical blindness associated with agnosia (bilateral lesion) Dyslexia without agraphia Hemiplegia Homonymous hemianopsia Memory disorders (bilateral lesion) Thalamic sensorial syndrome

Types of aphasic syndromes (nonfluent/fluent aphasias) [1, 2, 8] are:

1. Broca's aphasia
2. Wernicke's aphasia
3. Conduction aphasia
4. Transcortical aphasias:
 1. Transcortical motor aphasia
 2. Transcortical sensory aphasia
 3. Mixed transcortical aphasia

5. Global aphasias

6. Anomic plus aphasias

The global aphasia (24–38%) and anomic plus aphasia (20%) are more frequent in acute ischemic stroke; Broca (10–15%), Wernicke (15%), and transcortical motor aphasias (15–20%) present an intermediate frequency, and other aphasias are rare [1, 2, 5].

About 10% of aphasias remain unclassifiable, especially in patients with a previous ischemic stroke (atypical aphasias: mixed aphasias, thalamic aphasias, and capsule-striatal aphasias) [2, 12, 13, 14].

Aphasia type	Signs
Transcortical Motor	<ul style="list-style-type: none"> -difficulty is speaking fluently (primarily word retrieval) -language comprehension may be intact (more than Broca's) -may have difficulty with writing -may have difficulty initiating sentences
Broca's	<ul style="list-style-type: none"> -primary difficulty is speaking fluently -language comprehension may be intact or mildly impaired -may be able to read but not write -often labeled "expressive aphasia"
Mixed Transcortical	<ul style="list-style-type: none"> -primary difficulty is speaking fluently -may have difficulties with understanding spoken language -reading and writing often impaired -strength with repetition
Global	<ul style="list-style-type: none"> -difficulty speaking fluently -understands little to no spoken language -injuries to multiple language areas of the brain -most severe form of aphasia
Anomic	<ul style="list-style-type: none"> -primary difficulty with word retrieval -language comprehension is intact -reading is intact -considered mild form of aphasia
Conduction	<ul style="list-style-type: none"> -difficulty repeating words/phrases -some difficulty retrieving words -usually can speak fluently -usually can read and write
Transcortical Sensory	<ul style="list-style-type: none"> -difficulty understanding spoken language -repetition is intact -usually can speak fluently -similar to Wernicke's aphasia
Wernicke's	<ul style="list-style-type: none"> -difficulty understanding spoken language -difficulty repeating words/phrases -usually speech is fluent but without meaning -often labeled "receptive aphasia"

Wernicke Aphasia (Receptive)

- The lesion is located in the Wernicke area (Brodmann area 22), which is the center for comprehension and planning of words.[\[11\]](#)[\[12\]](#) Although patients are unable to understand written and/or spoken words, their speech remains fluent. At the same time, however, their speech is meaningless and can include paraphasia, both phonemic and semantics, neologism, and jargon. Phonemic paraphasia involves words that sound alike (i.e., sap and map), while semantics paraphasia pertains to words that relate to each other (i.e., sofa for couch). Neologisms are made-up words. Jargon is a string of made-up words in combination with real words that do not make sense together in context. These patients are unaware of their errors and do not recognize that their speech lacks meaning.
 - Example:
 - Examiner: Hi, how are you doing today?
 - Patient: I'm happy, are you pretty.
 - Examiner: What are you doing today?
 - Patient: We stayed with the water today.

Broca's Aphasia (Expressive)

- The lesion is located in the Broca area, which is the center for the motor part of speech and sentence formation.[\[12\]](#) Patients are unable to formulate grammatically correct spoken and written language properly. Although their speech is non-fluent and lacks words with grammatical significance such as prepositions and articles, patients are able to include important content words such as nouns, verbs, and some adjectives. Therefore, patients are usually able to convey the message that they are trying to express.
 - Example:
 - Examiner: Hi, Mr. Smith, when did you graduate college?
 - Patient: I was umm... 7 years... ago
 - Examiner: What did you use to do?
 - Patient: well... work... um... work... on desk... uh... marketing... very good...

Conduction Aphasia

- The lesion is located at the arcuate fasciculus, which is the neural pathway connecting the Wernicke area to the Broca area.[\[1\]](#) Patients have difficulties or are unable to repeat what is spoken to them. They recognize their paraphasic errors and try to correct them.
 - Example:
 - Examiner: Please repeat after me; boy.
 - Patient: Boy.

- Examiner: Seventy-nine.
- Patient: Ninety-seven...no... seventy nine... seventy-nine

Transcortical Sensory Aphasia

- The lesion is located around Wernicke area, however, sparing Wernicke area and isolating it. Patients have an inability to comprehend but can repeat with fluent speech. At the same time, patients display the presence of semantic paraphasia.

Transcortical Motor Aphasia

- The lesion is located around Broca area, however, sparing Broca area and isolating it. Patients have a non-fluent speech but are able to repeat long, complex phrases. Patients tend to remain silent but may speak with 1 to 2 words.

Mixed Transcortical Aphasia

- The lesion is located around Wernicke area, Broca area, and arcuate fasciculus, but these areas are spared and are isolated. Patients would have severe speaking and comprehension impairment; however, they can repeat long, complex sentences.

Global Aphasia

- Lesions vary in size and location but tend to follow the left middle cerebral artery distribution. This is the most severe form of aphasia. Patients can only produce a few recognizable words and have little to no understanding of written or spoken language. Patients are unable to read or write.

Anomia

- The lesion is at the angular gyrus and is the mildest form of aphasia. Patients have difficulties with word finding.

Related Behaviors

- Self-repairs are disruptions in fluent speech resulting from mis-attempts to repair erred production of speech.
- Speech disfluencies include repetitions and prolongations at the phonemic, syllable, and word level with pathological frequency.
- Preserved and automatic language are when some language or language sequences that were utilized frequently prior to onset are still produced with more ease.
- Struggle in non-fluent aphasia is a severe limitation in the number of words expressed

Fluent Aphasia Syndromes

- Wernicke- fluent, impaired comprehension, unable to repeat
- Transcortical sensory- fluent, impaired comprehension, able to repeat
- Conduction- fluent, intact comprehension, unable to repeat

- Anomic- fluent, intact comprehension, able to repeat

Non-fluent Aphasia Syndromes

- Broca- non-fluent, intact comprehension, unable to repeat
- Transcortical motor- non-fluent, intact comprehension, able to repeat
- Mixed transcortical- non-fluent, impaired comprehension, able to repeat
- Global- non-fluent, impaired comprehension, unable to repeat

causes

Aphasia can happen with any condition that damages the brain. It can also happen with problems that disrupt your brain's functions. Possible causes for this include:

1. Alzheimer's disease.
2. Aneurysms.
3. Brain surgery.
4. Brain tumors (including cancer).
5. Cerebral hypoxia (brain damage from lack of oxygen).
6. Concussion and traumatic brain injury.
7. Dementia and frontotemporal dementia.
8. Developmental disorders and congenital problems (conditions that you have when you're born because of a problem while you developed in the womb)
9. Epilepsy or seizures (especially if these causes permanent brain damage).
10. Genetic disorders (conditions you have at birth that you inherited from one or both parents, such as Wilson's disease).
11. Inflammation of your brain (encephalitis) from viral or bacterial infections, or autoimmune conditions).
12. Migraines (this effect is temporary).
13. Radiation therapy or chemotherapy.
14. Toxins and poisons (such as carbon monoxide poisoning or heavy metal poisoning).

Prognosis

Recovery of aphasia varies depending on the type, severity, cause, motivation of the patients, etc. Most improvement can be seen two to three months after onset and tends to peak at six months, with recovery rates drastically decreasing thereafter. Boca's aphasia has better recovery when compared to global aphasia, and global aphasia has better recovery compared to Wernicke aphasia

Aim of study

Aphasia intervention research aims to improve communication and quality of life outcomes for people with aphasia. the primary goal is for patients to regain their greatest level of independence. To achieve this goal, the patient's physical comorbidities, mental health, and deficits need to be addressed and properly managed. In addition, caregiver education and social support greatly impact a patient's recovery outcome.

Method

We collected patients by using primary quantities method by touring on patients who diagnosed with stroke in Nasiriyah Teaching Hospital, we have examined them, took a patient history, and reviewed the CT scan for each case. We collected approximately 37 case in a period of time approximately 30 days by using methods such as human observation, open-ended surveys.

Result

Broca's aphasia was 22 patients (59.45%) 3 patients was Smoker while (8.1%) HT was 10 patients(27%) ,DM 6 (16.2%)patients and 3(8.1%)patients have them together. Global aphasia was 15 patient. (54.%) One patient HT(2.7%) and 3(8.1%) patients with DM And 10(27%) case was have both of them.

The study was resulting 100% motor aphasia patients and due to few patients that admitted to first floor medicine floor in AL NASIRIAH TEACHING HOSPITAL and because decrease time of study .

Conclusions

Vascular aphasia is a term that covers complex syndromes, and it is considered not only a stroke severity marker outcome (it is associated with a higher risk of mortality) but also a poststroke poor functional outcome (can have a dramatic impact on person's ability to communicate and increased risk of developing poststroke dementia). Taking into consideration the unpredictable evolution of all mentioned aphasic syndromes and the lack of treatment strategies, next researches should focus on combined methods of improving patients' language after acute and even chronic stage of stroke (such as transcranial magnetic stimulation and speech therapy applied in consecutive, consequent, and sustained sessions).

Statistics

		AGE	TYPE	RISK_FACTOR	CT_FINDING
N	Valid	35	35	35	35
	Missing	0	0	0	0

TABLE: Incidence of aphasia according to TYPE

	Frequency	Percent	Valid Percent	Cumulative Percent
Valid BROCA'S	19	54.3	54.3	54.3
GLOUBAL	16	45.7	45.7	100.0
Total	35	100.0	100.0	

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
TYPE	35	1.4571	.50543	.08543
RISK_FACTOR	35	2.7429	1.29121	.21825
CT_FINDING	35	2.0286	.61767	.10440
AGE	35	72.3143	12.78734	2.16145

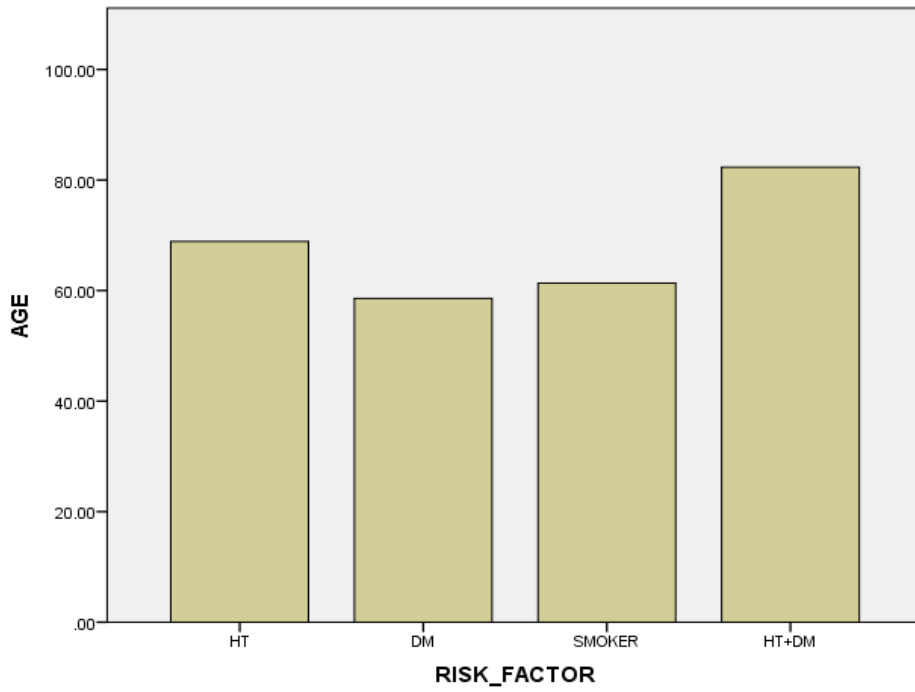
TABLE: Incidence of aphasia according to RISK_FACTOR

	Frequenc y	Percent	Valid Percent	Cumulative Percent
Valid HT	9	25.7	25.7	25.7
DM	7	20.0	20.0	45.7
SMOKE R	3	8.6	8.6	54.3
HT+D M	16	45.7	45.7	100.0
Total	35	100.0	100.0	

TABLE: Incidence of aphasia according to CT_FINDING

	Frequenc y	Percent	Valid Percent	Cumulative Percent
Valid ACA	6	17.1	17.1	17.1
MCA	22	62.9	62.9	80.0
PCA	7	20.0	20.0	100.0
Total	35	100.0	100.0	

Relation between age and risk factor:



Case Summaries :
Table : relation between type
of aphasia and age

			AGE
TYPE	BROC A'S	1	50.00
		2	55.00
		3	55.00
		4	55.00
		5	59.00
		6	60.00
		7	65.00
		8	66.00
		9	66.00
		10	69.00
		11	69.00
		12	70.00
		13	75.00
		14	75.00
		15	76.00
		16	77.00
		17	77.00
		18	83.00
		19	85.00
	Tot N	19	
	GLOU BAL	1	40.00
		2	58.00
		3	66.00
		4	72.00
		5	77.00
		6	77.00
		7	77.00
		8	79.00

	9	80.00
	10	82.00
	11	85.00
	12	88.00
	13	90.00
	14	90.00
	15	90.00
	16	93.00
	Tot	N
	al	16
Total	N	35

a. Limited to first 100 cases.

Frequency Table

		TYPE			
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	BROCA'S	19	54.3	54.3	54.3
	GLOUBAL	16	45.7	45.7	100.0
	Total	35	100.0	100.0	

		RISK_FACTOR			
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	HT	9	25.7	25.7	25.7
	DM	7	20.0	20.0	45.7
	SMOKER	3	8.6	8.6	54.3
	HT+DM	16	45.7	45.7	100.0
	Total	35	100.0	100.0	

		CT_FINDING			
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	ACA	6	17.1	17.1	17.1
	MCA	22	62.9	62.9	80.0
	PCA	7	20.0	20.0	100.0
	Total	35	100.0	100.0	

TABLE: Incidence of aphasia according to age

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	40.00	1	2.9	2.9	2.9
	50.00	1	2.9	2.9	5.7
	55.00	3	8.6	8.6	14.3
	58.00	1	2.9	2.9	17.1
	59.00	1	2.9	2.9	20.0
	60.00	1	2.9	2.9	22.9
	65.00	1	2.9	2.9	25.7
	66.00	3	8.6	8.6	34.3
	69.00	2	5.7	5.7	40.0
	70.00	1	2.9	2.9	42.9
	72.00	1	2.9	2.9	45.7
	75.00	2	5.7	5.7	51.4
	76.00	1	2.9	2.9	54.3
	77.00	5	14.3	14.3	68.6
	79.00	1	2.9	2.9	71.4
	80.00	1	2.9	2.9	74.3
	82.00	1	2.9	2.9	77.1
	83.00	1	2.9	2.9	80.0
	85.00	2	5.7	5.7	85.7
	88.00	1	2.9	2.9	88.6
	90.00	3	8.6	8.6	97.1
	93.00	1	2.9	2.9	100.0
Total		35	100.0	100.0	

References

1. 1. Abou Zeki D, Hillis A. Chapter 12. Acquired disorders of language and speech. In: Masud H, Schott JM, editors. *Oxford Textbook of Cognitive Neurology and Dementia*. UK: Oxford University Press; 2016. pp. 123–133
2. 2. Croquelois A, Godefroy O. Chapter 7. Vascular aphasia. In: Godefroy O, editor. *The Behavioral and Cognitive Neurology of Stroke*. 2nd ed. UK: Cambridge University Press; 2013. pp. 65–75
3. 3. Alajouanine T. *L'aphasie et le langage pathologique*. Paris : J.B. Baillière et fils éditeurs; 1968. p. 19
4. 4. Benson FD, Geschwind N. Chapter 5. Aphasia and related disorders, a clinical approach. In: *Principles of Behavioral Neurology*. 1989
5. 5. Jianu DC. *Elemente de afaziologie (Romanian)*. Timisoara, Romania: Mirton; 2001
6. 6. Ardila A. Chapter 10. Assessment of aphasia. In: *Aphasia Handbook 2*. Miami, Florida, USA: Florida International University; 2014. pp. 171–188
7. 7. De Renzi E, Faglioni P. Normative data and screening power of a shortened version of the token test. *Cortex*. 1978;7:41–49
8. 8. Goodglass H, Kaplan E, editors. *The Assessment of Aphasia and Related Disorders*. 2nd ed. Philadelphia, PA: Lea and Febiger; 1983
9. 9. Kertesz A. *The Western Aphasia Battery*. New York: Grune & Stratton; 1982
10. 10. Nespoulous JL, Lecours AR, Lafond D, editors. *Protocole Montreal–Toulouse de l'examen de l'aphasie: Module Standard Initial (Version Beta)*. Montreal: L'Ortho Edition; 1986
11. 11. Kertesz A, Sheppard A. The epidemiology of aphasic and cognitive impairment in stroke: Age, sex, aphasia type and laterality differences. *Brain*. 1981;104:117–128
12. 12. Nadeau SE, Crosson B. Subcortical aphasia. *Brain and Language*. 1997;58:355–402
13. 13. Verstichel P. Thalamic aphasia. *Revue Neurologique (Paris)*. 2003;122:947–957
14. 14. Lhermitte F, Derouesné J. *L'aphasie amnésique*. *Revue Neurologique*. 1976;132:669–685
15. 15. Kearns KP. Chapter 8. Broca's aphasia. In: La Pointe LL, editor. *Aphasia and Related Neurogenic Language Disorders*. 3rd ed. New York, Stuttgart: Thieme; 2005. pp. 117–141

16. 16. Alexander MP, Naeser MA. Broca' s area aphasics. *Brain and Language*. 1992;43(3):215-227
17. 17. Haartmann HJ, Kolk HHJ. The production of grammatical morphology in Broca' s and Wernicke' s aphasics. *Cortex*. 1992;28:97-112
18. 18. Jianu DC, Bednar M, Zolog A. Diagnosticul diferențial între tulburările fonetice și fonologice la afazici (Romanian). *Neurologie, Psihiatrie, Psihologie, Psihoterapie*. (Revista Societății de Neurologie și Psihiatrie pentru Copii și adolescenți din România), Bucuresti, Romania. 1999;2:55-58
19. 19. Jianu DC. Managementul afazicilor romani consecutiv infarctelor cerebrale (Romanian). Timisoara, Romania: Mirton; 2011
20. 20. Damasio AR. Aphasia. *New England Journal of Medicine*. 1992;326:531-539
21. 21. Kertesz A. Clinical forms of aphasia. *Acta Neurochirurgica. Supplementum (Wien)*. 1993;56:52-58
22. 22. Kory Calomfirescu S, Kory Mercea M. Afazia în accidentele vasculare cerebrale (Romanian). Cluj-Napoca, Romania: Casa Cărții de Știință; 1996
23. 23. Thompson CK. Chapter 2. Functional neuroimaging. In: La Pointe LL, editor. *Aphasia and Related Neurogenic Language Disorders*. 3rd ed. New York-Stuttgart: Thieme; 2005. pp. 19-38
24. 24. Hanna D. Chapter I. Neuroimaging contributions to the understanding of aphasia. In: Boller F, editor. *Handbook of Neuropsychology*. Vol. 2. Amsterdam, Netherlands: Elsevier Science Publishers, B.V.; 1989
25. 25. Poeck K, de Bleser R, von Keyserlink DG. Computed tomography localization of standard aphasic syndromes. In: Rose FC, editor. *Advances in Neurology*. Vol. 42: Progress in Aphasiology. New York: Raven Press; 1984. pp. 71-89
26. 26. Caspari I. Chapter 9. Wernicke' s aphasia. In: La Pointe LL, editor. *Aphasia and Related Neurogenic Language Disorders*. 3rd ed. New York-Stuttgart: Thieme; 2005. pp. 142-154
27. 27. Gainotti G, Ibba A, Caltagirone C. Perturbations acoustiques et semantiques de la compréhension dans l' aphasie. *Revue Neurologique (Paris)*. 1975;131(9):645-659
28. 28. Jianu DC, Petrica M, Matcău L, Zolog A. Observații anatomo-clinice asupra unor cazuri de afazie fluentă Wernicke (Romanian). *Neurologia Medico-Chirurgica*. 2001;6(1):69-79
29. 29. Simmons-Mackie N. Chapter 10. Conduction aphasia. In: La Pointe LL, editor. *Aphasia and Related Neurogenic Language Disorders*. 3rd ed. New York-Stuttgart: Thieme; 2005. pp. 155-

