Hegde's PocketGuide to Communication Disorders

Second Edition

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Preface to the Second Edition

The ever-expanding research base in communication disorders and related medical conditions have necessitated a revision of the first edition of this PocketGuide to Communication Disorders. Therefore, for this new edition, information on all disorders has been updated, along with critical references. Whenever warranted, characteristics of disorders, their classifications, epidemiology, and etiology have been revised to reflect the current state of research.

The second edition of this *PocketGuide to Communication Disorders* is a companion volume to the fourth editions of *Hegde's PocketGuide to Assessment in Speech-Language Pathology* and *Hegde's PocketGuide to Treatment in Speech-Language Pathology*. These three PocketGuides combine the most desirable features of a specialized dictionary of terms, clinical resource books, and textbooks. These three PocketGuides are meant to be quick reference books, like a dictionary, because the entries are alphabetized; but they offer more than most resource books by organizing detailed and specific information on disorders, their assessment, and their treatment. Unlike textbooks that address only one disorder each, these PocketGuides are organized such that detailed information can be quickly retrieved on most disorders of communication.

As a set, these three PocketGuides offer comprehensive information on the characteristics of communication disorders, their epidemiology, etiology, and major theories; assessment approaches and procedures; and treatment approaches and techniques. The three guides serve a dual function: First, they are detailed enough for student clinicians as well as more established practicing clinicians. Second, the guides are succinct enough to provide an overview of the entire range of knowledge in speech-language pathology.

How the PocketGuide Is Organized

This PocketGuide on communication disorders summarizes the available research and clinical information on disorders of communication, epidemiology and ethnocultural factors, symptomatology, onset and development, etiological factors, and major theoretical concepts. Whenever appropriate, neurophysiological and neuropathological factors related to a communication disorder also are described.

Each major disorder of communication is an alphabetized entry in the guide. Within each major entry, types or varieties of that disorder are alphabetically described. For example, under the main entry <u>Aphasia: Specific Types</u>, varieties of aphasia are described (e.g., <u>Broca's Aphasia</u>, <u>Wernicke's</u> <u>Aphasia</u>, etc.). Under the main entry <u>Fluency Disorders</u>, the reader will find entries for <u>Cluttering</u>, <u>Neurogenic Stuttering</u>, and Stuttering.

Entries in the guide are not limited to major communication disorders. Neurophysiological diseases or disorders of relevance and various clinical and related concepts also are defined and described.

How to Use This PocketGuide

The guide may be used very much like a dictionary. A clinician who wants to find out information about a specific communication disorder will access it by its main alphabetical entry. The table of contents will quickly direct the clinician to the major entries in the book; most of these are names of various disorders. Under each main entry, the clinician will find information within a standard format as described. The clinician also may be referred to related concepts and disorders that are cross-referenced. All cross-referenced entries are underlined. Thus, throughout the guide, an underlined term means that the reader can find more about it in its own main alphabetical entry.

About the Author

M.N. Hegde, PhD, is Professor Emeritus of Speech-Language Pathology in the Department of Communicative Disorders at California State University, Fresno. A highly regarded author in speech-language pathology, his books include leading texts in academic courses and valuable resources for clinicians. His books have been used in worldwide in speech-language pathology programs.

He holds a master's degree in experimental psychology from the University of Mysore, India, a post master's diploma in Medical (Clinical) Psychology from Bangalore University, India, and a doctoral degree in Speech-Language Pathology from Southern Illinois University at Carbondale.

Dr. Hegde is a specialist in fluency disorders, language disorders, research methods, and treatment procedures in communicative Disorders. He has made numerous presentations to national and international audiences on various basic and applied topics in communicative disorders and experimental and applied behavior analysis. He also has served on the editorial boards of scientific and professional journals and continues to serve as an editorial consultant to Journal of Fluency Disorders and the American Journal of Speech-Language Pathology.

Dr. Hegde is a recipient of various honors including the Outstanding Professor Award from California State University-Fresno, CSU-Fresno Provost's Recognition for Outstanding Scholarship and Publication, Distinguished Alumnus Award from the Southern Illinois University Department of Communication Sciences and Disorders, and Outstanding Professional Achievement Award from District 5 of California Speech-Language-Hearing Association. Dr. Hegde is a Fellow of the American Speech-Language-Hearing Association.

Acknowledgments

I am pleased to note that this new edition is being published by Plural, whose predecessor, Singular, was the original publisher of the first editions of the other two PocketGuides (*Hegde's PocketGuide to Assessment in Speech-Language Pathology* and *Hegde's PocketGuide to Treatment in Speech-Language Pathology*). I would like to thank Valerie Johns, Executive Editor, Nicole Hodges, Assistant Editor, Linda Shapiro, Production Coordinator, Jessica Bristow, Production Assistant, and Angie Singh, President and CEO of Plural, for their excellent support throughout the preparation of the new edition of this PocketGuide.



Abduct. Pull apart, open, as in *abducted* vocal folds.

Abductor Spasmodic Dysphonia (ABSD). A less common subtype of spasmodic dysphonia, a relatively rare voice disorder, characterized by sudden abduction of vocal folds during speech and the consequent cessation of phonation, bursts of breathiness, increased frequency of vocal spasms, and normal or near-normal voice except for such phonation breaks; see Spasmodic Dysphonia (SD).

- **Abulia.** Extreme lack of motivation, found in some psychiatric and neurological disorders; not an independent disease entity, but a symptom of specific clinical conditions; the person with abulia is uninterested in any kind of activity, including speaking; an extreme form of abulia is called akinetic <u>Mutism</u>, in which the client is alert, does not have neuromotor disorders related to speech, but still lacks motivation to speak; any speech that gets produced is initiated slowly; utterances tend to be brief and prosodically flat; abulia may be associated with severe anterior or mesial frontal lobe damage; see also <u>Psychiatric Problems</u> Associated With Communication Disorders.
- **Acoustic Nerve.** The cranial nerve VIII that conducts sound impulses from the cochlea to the brain's auditory center.
- **Acquired Communication Disorders.** Not due to genetic or neurophysiological conditions; may be learned, may be idiopathic; may be due to neurological events (e.g., strokes and traumatic brain injury).
- Acquired Immunodeficiency Syndrome (AIDS). A syndrome caused by human immunodeficiency virus, which causes the destruction of white blood cells and reduces cell-mediated immunity to diseases; drug treatment has made significant progress in controlling the disease and reducing opportunistic infections; patients' survival rate and duration have improved; untreated or

ineffectively treated AIDS may cause a type of dementia known as the AIDS Dementia Complex.

- **Acrocephaly.** A cranial abnormality found in certain genetic syndromes, resulting in high-domed skull (see <u>Syn</u>-dromes Associated With Communication Disorders).
- **Acute.** A short, suddenly emerged, severe clinical condition.
- Adaptation Effect. Progressive decrease in the frequency of stuttering when a printed passage is orally read repeatedly; maximum decrease observed on the second reading; decrease is progressively less on subsequent readings, with little or no decrease after the fifth reading; the effect is reduced or eliminated by rest pause between readings; there is no transfer of the effect across passages; contrasted with Consistency Effect.
- Adductor Spasmodic Dysphonia. A more common variety of spasmodic dysphonia; a voice disorder of presumed neurological origin; characterized by vocal fold adduction with excessive force and effort; the resulting voice sounds strained and strangled; associated features may include head jerks, eye blinks, and repetition of speech sounds; see <u>Spasmodic Dysphonia (SD)</u> and <u>Voice</u> Disorders.
- **Adjacency Effect.** Occurrence of new stuttering on previously fluently read words because they are adjacent to words that were stuttered in prior oral readings of the same passage; the effect may be recorded by blotting out the previously stuttered words and asking the client to read the passage aloud; some of the words printed before, after, above, and below the blotted words may be stuttered on repeated readings even though they were initially read fluently; shows that stuttering is under stimulus control.

Afferent. Flow of information toward cell body.

Affricates. Speech sounds that include stops and fricatives.

African American English (AAE). A dialect of American English shaped by the cultural and historical forces of African American people.

Agenesis. Absence of an organ due to a genetic defect.

Agnosia. A group of disorders in which recognition of sensory stimuli is impaired to varying extents due to central nervous system dysfunction in the absence of impaired sense organs; the individuals can see, hear, or feel stimuli or objects but are unable to grasp their meaning; rare in its pure form; to diagnose agnosia, it should not be a function of intellectual deterioration or dementia that also may give the impression of agnosia; once they recognize the stimulus, they can name it; a person who cannot recognize an object presented visually may recognize it in some other modality (e.g., tactile or auditory); the major types of agnosia include the following:

- Auditory agnosia: Difficulty recognizing the meaning of auditory stimuli including language, in the absence of peripheral hearing loss as assessed by pure tone audiometry; bilateral damage to the auditory association areas is the typical cerebral pathology; the individuals
 - Can hear a sound but cannot understand its meaning
 - May visually recognize an object but cannot match the object with its sound
- Auditory verbal agnosia: Difficulty understanding the meaning of spoken words the person hears well; also known as *pure word deafness*, a rare form of agnosia; bilateral temporal lobe lesions that isolate Wernicke's area from the other parts of the brain are thought to cause it; the individuals
 - May have significant problems understanding the meaning of what others say
 - May recognize nonverbal sounds
 - May recognize printed or written words
 - May have intact spontaneous speech, reading, and writing

- Prosopagnosia: Difficulty recognizing familiar faces due to right hemisphere damage. The persons
 - May fail to recognize the faces of family members, friends, and other familiar persons
 - May recognize the face when the person begins to speak
- Tactile agnosia: Difficulty recognizing or discriminating objects through touch when blindfolded and does not hear the sounds that are associated with them (if any); lesion in the parietal lobe that isolates the somatosensory cortex from other parts of the brain is the most common cause; the individuals
 - Report normal sensation through touch, but cannot name the objects they touch or hold in their hands while they cannot see them
 - May correctly name the objects when they see them or hear the characteristic sounds
- Visual agnosia: This is difficulty recognizing or discriminating visual stimuli, a rare form of agnosia; causes include bilateral occipital lobe lesions, posterior parietal lobe lesions, or damaged fibers that connect the visual cortex to other brain regions; the individuals
 - Cannot name what they see
 - May have no difficulty naming the objects when they hear their characteristic sounds
 - May name the objects when they touch them

Agrammatism. Deficient grammar (missing grammatic features) in spoken or written language, also known as <u>Telegraphic Speech</u>. Sentence length and variety are limited; a characteristic of nonfluent forms of <u>Aphasia</u>, especially Broca's Aphasia (see Aphasia: Specific Types).

Agraphia. Loss or impairment of previously acquired writing skills due to recent brain pathology; writing problems in children are typically associated with learning disorders and are not classified as agraphia; in adults, agraphia is associated with aphasia, dementia, and other neurological disorders; controversially, the foot of the second frontal gyrus has been suggested as the area that controls writing

(Exner's area); several areas in the left hemisphere may be involved in writing; speech-language pathologists assess and treat writing problems in the context of <u>Aphasia</u> and other language disorders; the loci of brain lesions and their consequences include the following:

- Left hemisphere lesions: They lead to structural and syntactic writing problems; the writing
 - Tends to contain morphologic and syntactic errors
 - May include neologistic constructions found in individuals with aphasia
- Right hemisphere lesions: They may affect the spatial aspects of writing, especially in individuals with right hemisphere damage; the individual
 - May fail to give margins and adequate spaces in between words and sentences
 - May neglect the left side of the page in writing
- Apraxic agraphia: Writing problems associated with Apraxia, possibly due to focal brain lesions in the parietal lobe; problems include
 - Disorders of letter formation, numerous spelling errors, and repeated words
 - Each letter of the alphabet may be only a scribble in severe cases
 - Writing only in capital letters—spontaneous writing, copying, and writing to dictation may all be equally affected
- Motor agraphia: Writing problems due to impaired neuromotor control. Upper and lower motor neuron pathology may affect the muscles of the hand and thus lead to motor agraphia. Individuals may
 - Write with very small letters or the size of the letters may progressively decrease (hypokinetic agraphia or micrographia)
 - Write in a highly disorganized manner or may find it impossible to write because of tremors, tics, chorea, and dystonia (hyperkinetic agraphia)
- Pure agraphia: An isolated writing disorder in the context of normal language functions, including normal auditory comprehension; not a part of aphasia, and its existence is in doubt; suggested neuroanatomic sites of

A

lesions include the premotor cortex (Exner's area) and the superior parietal lobe. The individual

- May not write anything although there is no motor involvement
- May produce spontaneous writing full of errors, but automatic writing and copying may be normal or nearly so

Benson, D. E., & Ardila, A. (1996). *Aphasia: A clinical perspective*. New York, NY: Oxford University Press.

Aided System of Communication. Use of such external aids as pictures, written messages, communication boards, and other kinds of devices, including electronic devices, by individuals who have limited oral communication skills.

AIDS Dementia Complex (ADC). Dementia associated with acquired immune deficiency syndrome (AIDS); ADC is one of several infection-induced forms of dementia; also known as *HIV encephalopathy* and *HIV/Aids-Associated dementia;* due to HIV infection itself, not due to opportunistic infections commonly affecting patients with AIDS.

Epidemiology and Ethnocultural Variables

- For the most current statistics, readers should visit https://www.cdc.gov (Centers for Disease Control and Prevention); the numbers tend to change annually; incidence of ADC is correlated with AIDS; the higher the incidence of AIDS in a subgroup of the population, the greater the prevalence of ADC in that population; effective treatment is available for HIV, but not all ethnic groups have equal access to it; at 12% of the general population, African Americans represent 45% of HIV diagnosis; at 18% of the general population, Hispanic Americans represent 24% HIV diagnosis; more men than women are affected with AIDS
- The incidence of ADC was higher before the highly active antiretroviral therapy (HAART) that is effective in treating AIDS (acquired immunodeficiency syndrome). Before HAART was available (1990–1992),

the incidence of aids dementia complex in HIVinfected patients was 21 cases per 1,000 person-years; after the advent of HAART (1996–1998), the incidence decreased to 10.5 cases per 1,000 person-years

- With untreated AIDS in children, the prevalence of HIV-associated progressive encephalopathy (HPE) is 50%; with treatment, HPE prevalence drops to less than 2%. Arrested HPE in children is about 10%
- The incidence of ADC is on the rise again because of the effective HAART treatment, patients with AIDS are living longer and run the risk of ADC
- Incidence of ADC is higher in African Americans and people living in Africa partly because of inadequate access to HAART

Onset and Early Symptoms

- Prominent early symptoms include the following:
 - Forgetfulness, slow thinking, and generally slow response time
 - Apathy, diminished sex drive, and loss of interest in work
 - Social withdrawal and depression
 - Attention deficits relative to speaking and writing
 - Disorientation to time and space
 - Frequent and severe headaches
 - Progressive deterioration in balance and weakness in legs
 - Deterioration in handwriting
 - Reduced verbal output

Progression and Advanced Characteristics

- · Serious and general cognitive deterioration may include
 - Severe memory impairment
 - Severely impaired concentration
 - Confusion and indifference
 - Hallucinations and delusions
 - Disorientation to time, place, and person
- Severe neurological symptoms may include
 - Further deterioration in motor skills and performance
 - More serious balance problems (ataxia)

- Neuromotor disorders including hypertonia, myoclonus, seizures, tremors, facial nerve paralysis, and rigidity of the muscles
- Incontinence
- Opportunistic infections (organisms that can infect only when a client's immune system is weak)
- Slow progression in the beginning but rapid physical and cognitive decline in the advanced stage
- Mutism in the final stage of the disease
- Diagnosis of dementia precede the diagnosis of fullblown AIDS

Etiologic Factors

- Human immunodeficiency virus
- Depletion of white blood cells
- Impaired immune system
- Neural degeneration in subcortical white matter and the basal ganglia
- Degeneration of cortical layers in the advanced stage of the disease

Air Conduction. Sound conducted to the ear through the medium of air.

- **Akinesia.** Absent or reduced voluntary movement, a symptom of several neurological diseases including <u>Parkinson's Disease and Progressive Supranuclear Palsy.</u>
- Alexia. Reading problems in adults of recent origin, due to neuropathological conditions and not attributable to peripheral visual impairments; in children, reading problems are a part of learning disabilities and are called *dyslexia*; speech-language pathologists assess and remediate reading problems because of their frequent association with neurodegenerative diseases that cause communication deficits, including <u>Aphasia</u>; varieties of alexia include the following.
 - *Alexia With Agraphia.* Reading and writing problems due to recent cerebral pathology, also called parietal-temporal alexia; often associated with aphasia;

the severity of reading and writing problems are roughly equal; reading nonalphabetic symbols, such as musical notations and mathematical formulae, also are affected; these problems are typically due to lesions in the angular gyrus and the dominant parietal and temporal lobes; lesions may be caused by strokes, tumors, metastatic tumors, trauma, and gunshot wounds; includes reading and writing problems associated with Wernicke's Aphasia and Broca's Aphasia (see Aphasia: Specific Types); characteristics include the following:

- Reading difficulties reflect typical problems of oral expression
- Reading comprehension deficits are more serious in clients with Wernicke's aphasia and are generally related to the degree of auditory comprehension of the spoken language; individuals may be unable to comprehend words that are spelled out for them
- Better reading of concrete nouns than abstract words by clients with Broca's aphasia
- Better comprehension of read material by clients with Broca's aphasia than by clients with Wernicke's aphasia
- Writing problems parallel an individual's reading problems; letter combinations in writing may be nonsensical
- Letter and word copying skills may be better than spontaneous writing skills

See Agraphia for more on writing problems.

- *Alexia Without Agraphia.* Reading difficulties that are due to recent neuropathological conditions; with relatively intact writing skills; causes include various neuropathologies associated with <u>Aphasia</u> and, more specifically, a disassociation between occipital association cortex (damage to the visual cortex) and the dominant angular gyrus; occlusion of the posterior cerebral artery, malformations of the arteries, and tumors cause this neural damage; the characteristics include
 - Varied reading problems as in alexia with agraphia; persons may be unable to read normally because of lost