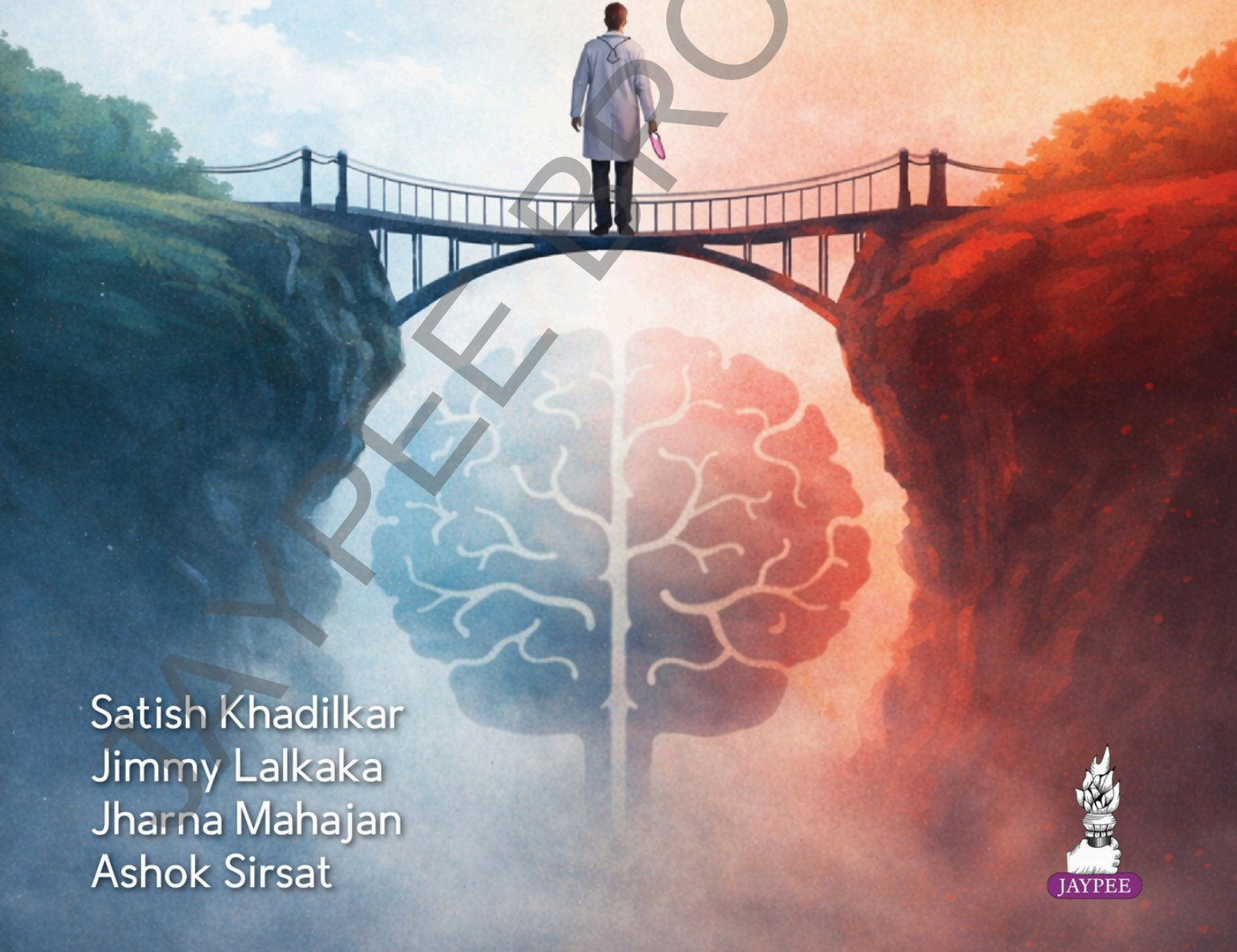
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Between Common *and* Critical

A GP's Guide to Neurology



Satish Khadilkar
Jimmy Lalkaka
Jharna Mahajan
Ashok Sirsat

JAYPEE

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SECTION 3

Speech and Language

Section Introduction

Speech and language disturbances are among the most striking neurological symptoms encountered in clinical practice. Patients may present with slurring of speech, difficulty articulating words, or problems in expressing or understanding language. Although these complaints are often grouped together by patients and caregivers, they arise from very different mechanisms and anatomical locations within the nervous system.

Speech disturbances may result from dysfunction of the articulatory apparatus—tongue, lips, palate, and their neural control—leading to dysarthria. In contrast, language disorders arise from damage to specialized cortical networks responsible for the comprehension and production of meaningful speech. Acute onset of slurred speech may signal life-threatening conditions such as stroke or encephalopathy, whereas chronic or progressive speech disturbances may point toward neuromuscular disorders, neurodegenerative diseases, demyelination, or intracranial lesions.

This section examines speech and language complaints through their major clinical patterns: Acute slurring of speech, chronic articulation problems, and language disturbances affecting expression or comprehension. Distinguishing between these patterns helps localize the neurological lesion and determine the urgency of investigation and treatment.

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3.1 Acute Slurring of Speech

"This is usually a medical emergency."

SERIOUS DISEASES

- *Acute stroke*: Acute slurring of speech is often associated with weakness of the right hand and sometimes the leg as well. Such a stroke is left hemispheric. Here, the speech problem is termed dysphasia as it relates to the language dysfunction, and the speech output dysfunction is often associated with defects of comprehension and repetition. Acute slurring of speech can come with imbalance and bilateral deficits in brainstem strokes.
- *Myasthenia gravis*: The slurring fluctuates. As patients continue to speak, the speech deteriorates. It is associated with chewing difficulty, ptosis, and double vision.
- *Encephalopathy*: These patients have altered sensorium and seizures. Focal deficits are less common.

COMMON DISEASES

- *Intoxications*: Due to alcohol, other inebriants, and drug toxicity
- Dental problems and mouth ulcers
- *Bells's palsy*: Acute lower motor neuron facial nerve palsy usually affecting eye and mouth.

WHICH INVESTIGATIONS ARE NEEDED?

- Serum electrolytes, serum calcium, EEG, and MRI brain for encephalopathic patients
- Acetylcholine receptor antibodies, MuSK antibodies, decrementing response in electrophysiology, and CT thymus for thymoma
- Urgent MR angiogram brain with diffusion imaging for stroke

WHICH SYMPTOMATIC TREATMENTS CAN BE GIVEN ON PRIORITY?

- Pyridostigmine for myasthenia (pyridostigmine 60 mg ½ tab TDS/QID, slowly titrate by 30 mg every 2–4 days to find the lowest effective dose, titrate if needed up to 60 mg QID over 5–10 days and experiment with timing. Maximum up to 300 mg/day as divided doses)
- Suggest urgent admission to the hospital

WHICH DEFINITIVE THERAPY CAN BE GIVEN?

- For acute stroke therapy, IV thrombolysis with injection alteplase (0.9 mg/kg IV; not to exceed 90 mg total dose), injection tenecteplase (0.25–0.5 mg/kg). Secondary prophylaxis with antiplatelets such as tablet aspirin (75–150 mg/day PO OD) and tablet clopidogrel (75 mg/day PO OD), ticagrelor (180 mg/day PO q12h), and atorvastatin (40–80 mg/day PO)
- *Myasthenia gravis*: Gradual increasing dose of steroids with steroid sparing agents such as mycophenolate mofetil (start with 500 mg/day to maximum dose of 2 g/day) and azathioprine (1 mg/kg/day not to exceed 2.5 mg/kg/day)
- For encephalopathy, correction of electrolyte imbalance and vitamins and cofactor supplementation can help.
- *Bell's palsy*: Prednisolone 1 mg/kg/day till max of 80 mg for 7 days then taper steroids over 5 days, tablet valacyclovir 1 g TDS for 7 days or tablet acyclovir 400 mg five times a day for 10 days, eye protection—glasses, goggles, and artificial tears drop.

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Approach to acute slurring of speech

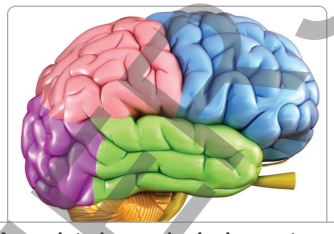
Step 1: Initial approach



Clarify
- Dysarthria (Slurred articulation)
- Aphasia (Language impairment)
- Both



Onset pattern
- *Sudden:* Stroke more likely
- *Fluctuating:* Myasthenia Gravis most likely
- *With confusion:* Encephalopathy
- *Toxins:* Alcohol/drug



Associated neurological symptoms
- Facial asymmetry
- Limb weakness
- Imbalance/vertigo
- Seizure
- Diplopia/fatiguability



Red flags of

Acute slurring of speech

Ha...h-ow a...are yyour??

Ha..hhow a...are yyou???

Warning signs requiring urgent medical evaluation

1 Sudden onset slurring ± facial droop or limb weakness



Acute stroke (Hemispheric/Brainstem) | Emergent stroke protocol → | MRI/CT brain



2 Slurring + ptosis + fatigability + fluctuating weakness



Myasthenia Gravis | Start pyridostigmine, check antibodies; urgent admission



3 Slurring after heavy alcohol/drug intake or suspected overdose



Intoxication/ Drug toxicity | Toxicology screen, supportive care; monitor airway protection

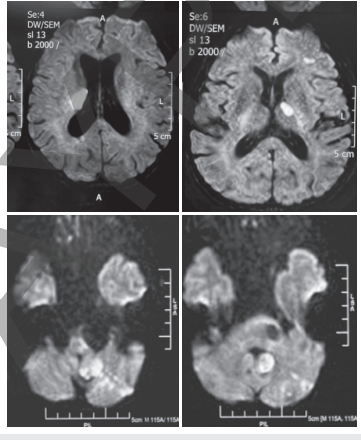
4 Slurring + inability to handle secretions/ risk of aspiration



Bulbar failure from neuromuscular disease | Immediate admission for airway pro



Immediate medical evaluation is critical for these warning signs



Acute stroke: Different territory acute infarct that can present as dysarthria.

Bell's palsy

What

Paralysis
- Acute
- Idiopathic
- Unilateral ↓ face

Signs
- Face
- Eye
- Mouth
- Drool



Treat
- Steroids
- Eye care
- Antivirals



Clues
- Ear
- Taste
- Sound
- Tears



Red flags
- Gradual
- Recurrent
- Bilateral
- Other CN

Bell's palsy: Acute lower motor neuron facial nerve palsy.

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3.2A Chronic Speech Problems: Articulation

"Articulation problems are termed as dysarthrias. They result from dysfunction of the articulatory apparatus (tongue, lips, palate, and their control from the cerebrum and pyramidal systems)."

SERIOUS DISEASES

- *Motor neuron disease*: The bulbar and pseudobulbar involvement results in dysarthria. It is accompanied by difficulty in swallowing, inability to swallow saliva, emotionality leading to uncontrolled crying or laughing, and a combination of upper and lower motor neuron signs in the limbs.
- *Brainstem demyelination/space-occupying lesion*: Chronic slurring of speech can be associated with imbalance and bilateral deficits due to brainstem plaques. Space-occupying lesions such as ependymoma, glioma, lymphoma, or secondaries can also cause dysarthria.
- *Myasthenia gravis (fluctuating slurring of speech)*: As patients continue to speak, the speech deteriorates. It is associated with chewing difficulty, ptosis, and double vision.

COMMON DISEASES

- *Parkinson's disease*: Low volume speech, which is spoken rapidly, accompanied by tremors, rigidity, slowing, and postural reflex changes
- *Tongue tie*: Defects of lingual phonation, malocclusion of jaws, or tooth pain
- Early-life hearing loss

WHICH INVESTIGATIONS ARE NEEDED?

- EMG NCV studies of the cervical, thoracic, and lumbar segments and the tongue for motor neuron disease
- Acetylcholine receptor antibodies, MuSK antibodies, decrementing response in electrophysiology and CT thymus for thymoma
- Urgent MRI with contrast for demyelination/SOL

WHICH SYMPTOMATIC TREATMENTS CAN BE GIVEN ON PRIORITY?

Pyridostigmine for myasthenia (pyridostigmine 60 mg ½ tablet TDS/QID, slowly titrate by 30 mg every 2–4 days to find the lowest effective dose, titrate if needed up to 60 mg QID over 5–10 days and experiment with timing. Maximum up to 300 mg/day as divided doses.

WHICH DEFINITIVE THERAPY CAN BE GIVEN?

- Riluzole for motor neuron disease (50 mg PO q12h)
- Immunomodulation for myasthenia gravis, plasma exchange, and IVIG followed by a course of cortico-steroids, along with steroid-sparing agents. Gradual increasing dose of steroids with steroid-sparing agents such as mycophenolate mofetil (start with 500 mg/day to maximum dose of 2 g/day) and azathioprine (1 mg/kg/day not to exceed 2.5 mg/kg/day)
- Surgery for tongue tie **(Videos 5 and 6)**

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Approach to chronic speech problems: Articulation

Step 1: Initial approach

Define the speech disturbance

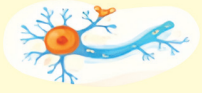
- Imprecise consonant production
- Slurred, "thick", or effortful articulation
- Distortion of phonemes rather than language errors
- Worsens with fatigue (consider neuromuscular cause)

Key associated symptoms

- Dysphagia, choking episodes
- Limb weakness or atrophy (MND)
- Ptosis, fluctuating facial weakness (MG)
- Facial asymmetry or long-standing dental/jaw issues
- Early childhood hearing impairment (speech development impact)
- Early childhood hearing impairment (speech development impact)

Red flags of chronic speech problems: Articulation

Certain speech symptoms may indicate serious underlying neurological conditions requiring urgent evaluation and action

Red flags	Likely etiology	Actions
Progressive dysarthria + limb wasting or fasciculations	Motor neuron disease 	Neuromuscular evaluation; start disease-modifying therapy (riluzole) 
Chronic articulation deficits + spasticity/UMN signs	Chronic demyelination or space-occupying lesions 	MRI brain with contrast 
Fatigable dysarthria ± ptosis/diplopia 	Myasthenia gravis 	Initiate pyridostigmine; confirm with antibodies consider IVIG/PLEX for worsening symptoms 

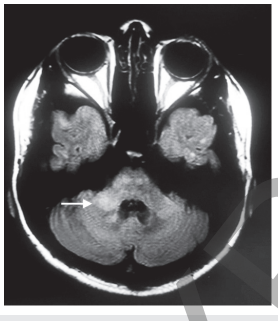
When to refer to a neurologist

Urgent referral

- Progressive dysarthria with limb weakness/wasting
- Dysphagia or aspiration
- Sudden onset or altered sensorium
- Fluctuating articulation with ptosis
- Dysarthria with diplopia or ataxia

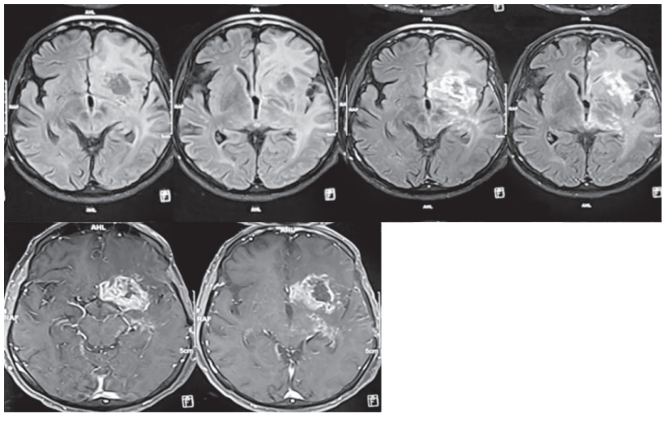
Elective referral

- Normal evaluation by dentist
- Facial or tongue abnormalities



Axial FLAIR MRI of the brain showing a focal hyperintense lesion in the left cerebellar hemisphere (arrow), suggestive of a demyelinating lesion.

MRI brain demonstrating a left frontal mass lesion. Axial *FLAIR images (top row)* show a heterogeneous hyperintense lesion in the *left frontal lobe* with surrounding vasogenic edema and mild mass effect. *Postcontrast T1-weighted images (bottom row)* demonstrate *irregular ring enhancement with central necrosis*, suggestive of a high-grade neoplastic process.



Scan the QR code to access the above videos

Video 5 **Video 6**

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3.2B Chronic Speech Problems: Expression

"Speech is the clarity and sound of talking; language is the understanding and forming of meaningful words."

SERIOUS DISEASES

- *Left hemispheric tumors*: Variable speech defects along with headaches, visual disturbances relating to increased intracranial pressure and right-sided limb signs
- *Dementias*: Dementias such as Alzheimer's disease have language problems as part of a multidomain affectation. They are chronic and progressive. Some variants, such as primary progressive aphasia and logopenic variant, have mainly language problems, and other features develop later.
- *Residue of left hemispheric stroke*: A lesion in and around Broca's area results in reduced word output, telegraphic speech and loss of repetition, prosody, and melody of speech. Lesions in and around the Wernicke's areas lead to comprehension deficits with increased word output which is not meaningful, and loss of repetition.

COMMON DISEASES

- There are truly no common diseases that lead to language problems, but head injury involving in the Broca's area and/or Wernicke's areas are seen from time to time.
- *Depression/apathy*: These can superficially resemble dysphasia.

WHICH INVESTIGATIONS ARE NEEDED?

MRI with angiogram sequence and contrast administration.

WHICH SYMPTOMATIC TREATMENTS CAN BE GIVEN ON PRIORITY?

None, suggest urgent admission to a hospital.

WHICH DEFINITIVE THERAPY CAN BE GIVEN?

- Surgeries, radiation, or chemotherapy for tumors, as per the type and extent of the process
- Speech therapy

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Approach to chronic language problems: Expression

Step 1: Initial approach

Define the type of expression difficulty

- Difficulty generating words
- Reduced frequency
- Word-finding pauses
- Effortful or fragmented language
- Mislabeling or incorrect word substitutions

Note: This is language issue—articulation is normal

Step 2: Non-neurological causes

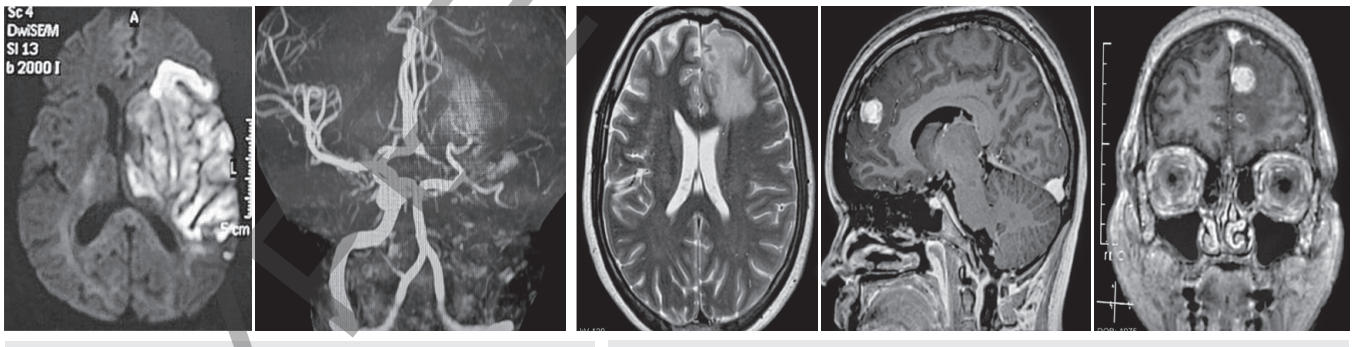
Differentiate dysphasia from non-neurological causes

- Depression/psychomotor slowing (not a true dysphasia)
- Low motivation, reduced speech output
- Normal articulation but decreased expressive effort

Red flags of expressive speech difficulty

Certain symptoms of expressive speech difficulty may indicate serious underlying neurological conditions requiring timely evaluation

Red flags	Likely etiology	Required action
Gradually worsening expressive difficulty	Left hemispheric tumor (Broca region)	MRI brain with contrast and angiogram sequence
Loss of fluency after head trauma	Injury to Broca's area	MRI brain; admit for neurology/ neurosurgery review
Stepwise or progressive speech fragmentation	Left hemispheric stroke (old/subacute)	MRI brain + vascular imaging; secondary stroke prevention



Large hemispheric stroke: Involving left frontoparietal region causing severe speech problems (expression + articulation).

MRI brain demonstrating a well-defined intracranial lesion. Axial, sagittal, and coronal images show a well-circumscribed lesion in the frontal region near the lateral ventricle, with surrounding edema and mild mass effect, suggestive of a space-occupying lesion.

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3.2C Chronic Speech Problems: Comprehension

"These are problems of the understanding of spoken and/or written language."

SERIOUS DISEASES

- *Left hemispheric tumors*: Lesions in and around the Wernicke's areas lead to comprehension deficits with increased word output which is not meaningful, and loss of repetition.
- Residue of left hemispheric strokes (in and around the Wernicke's area) leads to similar language changes as above.
- Head injury around the Wernicke's area

COMMON DISEASE/S

Depression or schizophrenia: In differentiating psychiatric and neurological diseases, neuropsychological evaluation is very helpful. This test generates cognitive scores and depression scores.

WHICH INVESTIGATIONS ARE NEEDED?

- MRI brain with angiogram sequence and diffusion sequences for strokes
- MRI with contrast administration for brain tumors

WHICH SYMPTOMATIC TREATMENTS CAN BE GIVEN ON PRIORITY?

None: Suggest hospital admission.

WHICH DEFINITIVE THERAPY CAN BE GIVEN?

- For Acute stroke therapy, IV thrombolysis with injection alteplase (0.9 mg/kg IV; not to exceed 90 mg total dose), injection tenecteplase (0.25–0.5 mg/kg). Secondary prophylaxis with antiplatelets such as tablet aspirin (75–150 mg/day PO OD) and tablet clopidogrel (75 mg/day PO OD), ticagrelor (180 mg/day PO q12h), and atorvastatin (40–80 mg/day PO)
- Surgeries, radiation, or chemotherapy for tumors, as per the type and extent of the process
- Speech therapy

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Approach to chronic language problems: Comprehension

Step 1: Initial approach

Define the type of comprehension difficulty

- Difficulty understanding spoken or written language
- Misinterpreting simple instructions
- Invention of new, nonsense words (Jargon)
- Responds inappropriately despite fluent speech
- Appears inattentive but comprehension is truly deficit

Note: This is language issue—articulation is normal

Step 2: Non-neurological causes

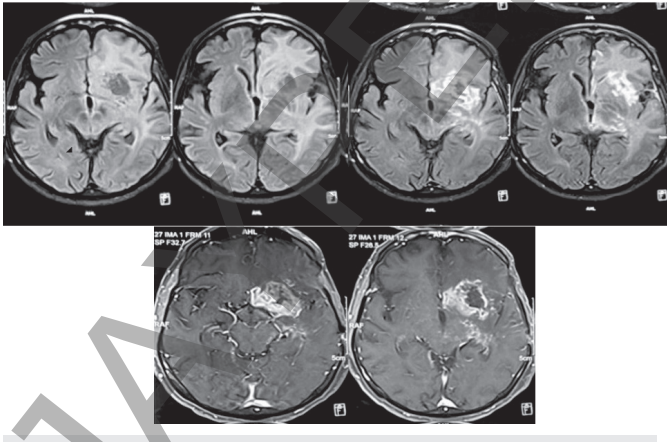
Exclude non-neurological causes

- Depression/psychomotor slowing—reduced engagement
- Schizophrenia/bipolar/mania disorder
- Hearing impairment

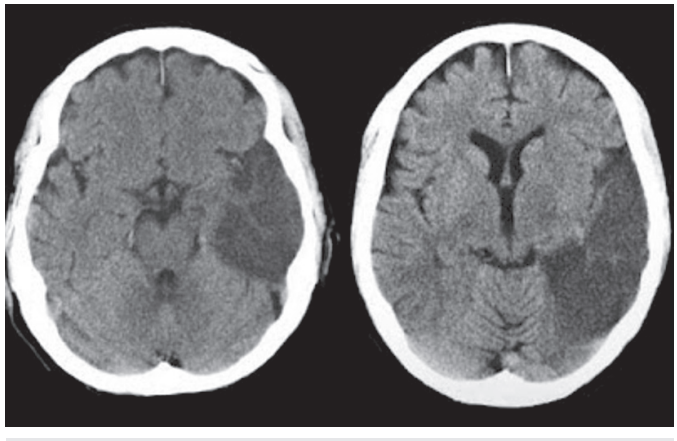
Red flags of comprehension difficulty

A certain comprehension difficulties may indicate serious underlying neurological conditions requiring urgent evaluation

Red flags	Likely etiology	Urgent action
Gradual but progressive decline in understanding spoken/written language	Left hemispheric tumor involving the Wernicke region	MRI brain with contrast + angiogram sequence
Abrupt onset of impaired comprehension with preserved fluency	Left hemispheric (Wernicke territory) stroke	Emergency neuroimaging; stroke pathway activation
Fluctuating comprehension deficits with overall deterioration	Seizure-related aphasia or evolving stroke	Urgent admission and MRI brain
New-onset comprehension difficulty in a patient with headache, seizures, or cognitive changes		MRI brain contrast-enhanced; neurosurgical review



Axial *FLAIR images (top row)* show a heterogeneous hyperintense lesion in the *left frontal lobe* with surrounding vasogenic edema and mild mass effect. *Postcontrast T1-weighted images (bottom row)* demonstrate *irregular ring enhancement with central necrosis*, suggestive of a high-grade neoplastic process.



Left ischemic stroke: Wedge-shaped hypodensity in left temporo-parietal region in MCA territory, involving the Wernicke's region.

WAVE BROTHERS

SECTION CONCLUSION

As we have seen, speech and language symptoms offer valuable clues to neurological localization when carefully analyzed. Slurred speech often reflects impairment of the motor pathways controlling articulation, while language disturbances indicate dysfunction of dominant hemispheric cortical networks responsible for communication. Differentiating dysarthria from aphasia is therefore a critical first step in evaluating patients with speech complaints.

The time course of symptoms provides further diagnostic guidance. Sudden-onset speech disturbance frequently suggests vascular or metabolic causes and requires urgent evaluation, while progressive changes over months or years may reflect neurodegenerative, demyelinating, or structural brain disease. Associated features such as limb weakness, cranial nerve dysfunction, cognitive decline, or fatigability often help narrow the differential diagnosis.

By recognizing these patterns and correlating them with clinical examination findings, general practitioners can rapidly identify neurological emergencies, initiate appropriate investigations, and ensure timely referral for definitive management.

SECTION TAKEAWAY

In patients presenting with speech or language disturbance, distinguishing dysarthria from aphasia and recognizing the pattern of onset are key to identifying neurological emergencies and localizing the underlying lesion.

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A GP's Guide to Neurology

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Satish Khadilkar MD DM(Neurology) DNBE FICP FIAN FAMS FRCP is a pioneering neurologist who established and advanced the field of neuromuscular disorders in India and contributed significantly across Asia. With over three decades of work, he identified key genetic mutations, introduced diagnostic advances, and built essential laboratory and research infrastructure. He has led national and international collaborations, guided research through the ICMR, and trained generations of neurologists. A gifted teacher and nationally awarded academician, he has made enduring contributions to medical education. His academic work includes multiple textbooks and extensive research publications. Beyond academia, he has provided decades of free patient care and supported a variety of patient organizations, leaving a lasting clinical, educational, and humanitarian legacy.

Jimmy Lalkaka MD DM(Neurology) is a highly respected neurologist based in Mumbai, with decades of clinical experience and a reputation for astute clinical judgement. A gifted teacher who has guided and influenced generations of neurologists, he is equally known for his meticulous diagnostic approach and deeply empathetic, patient-centric care.

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Ashok Sirsat MD DM(Neurology) is an accomplished neurologist trained at Grant Medical College, with extensive clinical experience in private practice. Known for his sharp clinical acumen and clear, compassionate communication, he is widely respected by both patients and peers. He has played a significant role in postgraduate training as a dedicated teacher for DNB Neurology at Lilavati Hospital and Holy Spirit Hospital Mumbai, Maharashtra, India. Dr Sirsat has also contributed to the professional community, having served as President of the Bombay Neurosciences Association and the Maharashtra Association of Neurology, reflecting his leadership and commitment to advancing neurological care and education.

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