Neurologic Emergencies at the Extremes of Age

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KEYWORDS

- Pediatric • Altered mental status • Geriatric • Trauma • Encephalitis • Stroke

KEY POINTS

- In a child with fever and altered mental status, consider anti–*N*-methyl-D-aspartate receptor encephalitis and acute disseminated encephalomyelitis in the differential diagnosis.
- Nonconvulsive status epilepticus is associated with higher mortality, longer pediatric intensive care unit stays, and increased long term disability.
- Age alone is not a contraindication for intravenous tissue-type plasminogen activator administration within 3 hours.
- Acute worsening in Parkinson's disease is usually due to a medication change, infection, or missed subdural hemorrhage.
- Meningitis in older adults presents atypically and has worse outcomes than the general population.

INTRODUCTION

Neurologic conditions, both common and emergent, present to the emergency department (ED) daily. The diagnosis and management of such conditions can be more complex at the extremes of age than in the average adult. In the pediatric population, neurologic emergencies are somewhat rare and some may require emergent consultation, as summarized in **Box 1**.

On the opposite end of the age spectrum, older adults disproportionately require emergency care, and the combination of geriatric physiologic changes with increased comorbidities leads to atypical presentations and worsened outcomes.[1,2] The unique considerations regarding emergency department (ED) presentation and management of stroke and altered mental status (AMS) in both age groups, in addition to seizures

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Box 1
Pediatric conditions requiring emergent neurology consultation

- High suspicion for stroke, regardless of intent to use tissue-type plasminogen activator
- Autoimmune encephalitis
- Acute demyelinating encephalomyelitis
- Acute peripheral neurologic deficit, that is, acute flaccid myelitis, Guillain–Barré syndrome, acute brachial neuritis
- Transverse myelitis
- Any suspected seizure at <1 year of age
- Reported or observed seizure activity with reports of loss of milestones or other systemic symptoms
- Newly documented neurologic deficits/symptoms or first time seizure with concerns regarding timely follow-up
- Refractory status epilepticus, convulsive or nonconvulsive

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and intracranial hemorrhage in pediatrics, and Parkinson's disease and meningitis in the geriatric population are discussed.

NEUROLOGIC EMERGENCIES IN THE PEDIATRIC PATIENT
Pediatric Neurologic Examination

Although not "little adults," our pediatric patients are not aliens either. Their neurologic systems are, however, still in development and in need of a different approach for examination. The normal range for each component varies by age, environmental exposure, and any underlying congenital malformations or diseases. The physical examination begins from the moment you see the patient. Observe how alert or withdrawn they are, or how they respond to their caregiver's voice. Note the muscle tone and posture of the infant or child, and their response to touch. Understand the child's baseline behavior from the accompanying caregiver and compare it with your assessment to uncover any abnormalities. An effective mnemonic for a gross neurologic evaluation of the child is *TICLS*: *T*one, *I*nteraction, *C*onsolability (irritability), *L*ook (gaze), *S*peech (including jargon or cry), as one arm of the Pediatric Assessment Triangle[3] (**Table 1**).

Table 1
Pediatric neurologic examination: the TICLS mnemonic

	Element	Exam Questions
T	Tone	Do they appear floppy or limp? Head lag? Response when touched?
I	Interaction	Resistance to exam? Smile or grin? Acknowledgment of providers?
C	Consolability	Are they irritable or fussy? Consoled by caregiver presence or affection? Pitch of the cry?
L	Look	Pupillary size? Eye contact? Fixed or roaming gaze? Conjugate or disconjugate? Tracking?
S	Speech	Age appropriate babble or jargon? Age appropriate sentences? Oriented or confused? Speech slurring?

Data from Horeczko T, Enriquez B, McGrath NE, Gausche-Hill M, Lewis RJ. The Pediatric Assessment Triangle: Accuracy of Its Application by Nurses in the Triage of Children. *J Emerg Nurs.* 2013;39(2):182-189.

Pediatric Acute Ischemic Stroke

Clinical presentation

In the pediatric population, stroke is rare and can be more difficult to recognize than in the adult population. Children younger than 5 years of age are the most likely to experience acute ischemic stroke (AIS) among pediatric patients. AIS in young children is more likely to present with global neurologic impairment, such as seizure or AMS, than in adults.[4] Up to 50% of infants and young children with AIS may present with AMS, fever, and/or seizure. Older children and teens are more likely to present with focal neurologic deficits similar to those in adults. Considering AIS in the list of differential diagnoses of seizure or AMS, especially in infants and younger children, can improve time to diagnosis. Delay in diagnosis up to 24 hours from the time of presentation is common, even though patients often present within 6 hours from time of onset.[4]

Examination should begin promptly with observation of the patient and quantification of neurologic deficits as soon as they are identified or reported by the caregiver. A noncontrast computed tomography (CT) scan of the brain and point-of-care glucose are just as important in children as in adults. Rapid use of the Pediatric National Institute of Health Stroke Scale should be included in patients ages 6 years and younger to quantify neurologic deficits and guide the decision to use thrombolytics, particularly if the score is greater than or equal to 6.[4,5] The Pediatric National Institute of Health Stroke Scale is partially completed by the physician's observation of the child's behavior and responses in the ED given that it is difficult for many younger children to follow the instructions of the examination. Other age-specific modifications are summarized in **Box 2**.

Thrombolytics

After ruling out intracranial hemorrhage using a CT scan, tissue-type plasminogen activator (tPA) can be given in consultation with the pediatric neurologist and/or

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Box 2
National Institutes of Health Stroke Scale Modifications in Pediatric Patients

- Orientation – Ask age and point to family member
- Commands – Blink eyes and touch nose
- Gaze – Assessed with horizontal gaze only
- Visual fields – Finger counting if >6 years old, or visual threat if <6 years old
- Facial palsy – Scored same as adults
- Motor strength – If unable to follow commands, score by spontaneous or elicited movement, that is, assisted weight bearing may demonstrate 1 leg weaker than the other
- Ataxia – Ask to reach or kick toy in examiner's hand
- Sensation – Pinprick testing, or observe for spontaneous response to pinprick
- Language – Assess with stated words, naming pictures[a]
- Dysarthria – Repeating words or sentences, ask about baseline speech impediment
- Extinction/inattention – Scored similarly to adults

[a] Must understand baseline verbal skills.

Data from Ichord RN, Bastian R, Abraham L, et al. Interrater reliability of the Pediatric National Institutes of Health Stroke Scale (PedNIHSS) in a multicenter study. *Stroke.* 2011;42(3):613-617.

pediatric intensivist, if available, who will be accepting the patient. MRI of the brain is the preferred imaging for detailed examination of brain parenchyma; however, it is not required before tPA administration. Thrombolytics should not be delayed for an MRI if the physician's suspicion for AIS is reasonably high, but certain institutional algorithms may require demonstration of acute vascular occlusion before giving tPA.[4] Goal administration time is of less than 4.5 hours from the time of onset, and early administration is more likely to result in improved outcomes. The pediatric tPA dosing is the same as that in adults: 0.9 mg/kg with 10% administered as a bolus followed by 90% as an infusion over 60 minutes.[4] In vitro data show that the thrombus composition of children's plasma contains less fibrin as compared with adults, resulting in higher recanalization rates with tPA.[4] A small study to review efficacy and safety of tPA in children was published in 2019; there was no clinically significant intracranial hemorrhage after tPA administration in 26 children. One patient experienced severe epistaxis requiring intubation, but details regarding epistaxis evaluation and management are of limited description. There were 2 patients with cerebral petechiae found on follow-up MRI within the area of infarction, but this did not seem to limit their recovery. Overall, the estimated risk profile of tPA in pediatric stroke is 2.1% when given within 4.5 hours from time of onset.[5]

Thrombectomy

There is limited evidence supporting thrombectomy in patients under 18 years of age; however, it may be performed on patients under certain circumstances. Expert opinion, based on extrapolated adult data, encourages its consideration within 6 hours of symptoms onset in select patients. Technical limitations include the smaller size of peripheral and intracranial vessels, as well as increased concerns for radiation risk and contrast load.[4]

Stroke in sickle cell disease

Sickle cell disease accounts for a large portion of disease burden in pediatric stroke, particularly in African American children. The time of greatest risk for stroke in a patient with sickle cell disease is between 2 and 5 years of age.[4] Given that many of these strokes are silent in nature, the only clinical signs may be early hand dominance, speech delays, or learning disabilities. When AIS in sickle cell disease is symptomatic, it is theorized that, owing to the development of higher pain tolerance, headache is a less frequently reported symptom of AIS in sickle cell disease as compared with AIS patients without sickle cell disease.[6] In patients presenting with an acute neurologic deficit, immediate treatment is needed (**Box 3**). In patients with a baseline hemoglobin (Hgb) of less than 10 g/dL, urgent transfusion of 10 mL/kg of packed red blood cells for a goal Hgb of 10 g/dL or sickle cell percentage of less than 15% is indicated, to be given within 6 hours of symptom onset.[4] If the baseline Hgb is greater than 10 mg/dL, exchange transfusion must be performed. Repeat transfusion may be needed but Hgb should not go above 11 g/dL, and post-transfusion Hgb measured at approximately 2 hours after to assess risk hyperviscosity syndrome, which may occur with rapid overcorrection of the Hgb.[4] Hyperviscosity syndrome may lead to cerebral venous sinus thrombosis or multifocal infarcts. Pain and hydration status should be addressed as they would in other vasoocclusive crises. The emergency physician should also look for additional underlying precipitating factors contributing to the acute presentation, such as infection, dehydration, trauma, hypoxia, or acidosis. Ultimately, the patient will require emergent transfer to higher level care and consultation with pediatric hematology if available.

Box 3
Stroke management in pediatric patient with sickle cell disease

Special considerations in sickle cell disease

- When AIS suspected, prompt transfusion is needed
- Goal time to transfusion: <6 hours from onset of symptoms
- Start with 10 mL/kg of packed red blood cells, goal Hgb 10 g/dL or <15% sickled Hgb
- May need exchange transfusion if baseline Hgb is close to 10 g/dL
- Avoid hypervolemia and hyperviscosity syndrome from overtransfusing
- For Hgb >12 g/dL, consider phlebotomy to decrease total blood volume (consult with hematology for recommended volume removal)
- Consider aggravating factors that may contribute to acute sickling or clinical presentation

Abbreviation: Hgb, hemoglobin.

Data from Ferriero DM, Fullerton HJ, Bernard TJ, et al. Management of stroke in neonates and children: A scientific statement from the American Heart Association/American stroke association. *Stroke.* 2019;50(3):E51-E96; Guilliams KP, Kirkham FJ, Holzhauer S, et al. Arteriopathy Influences Pediatric Ischemic Stroke Presentation, but Sickle Cell Disease Influences Stroke Management. *Stroke.* 2019;50(5):1089-1094.

Intracranial Hemorrhage

Many principles of adult care should be readily applied in the management of traumatic and nontraumatic intracranial hemorrhage in pediatric patients. Prioritization of the airway, breathing, and circulation, monitoring of vitals, and assessing blood glucose are necessary.[7] Airway management in the setting of profound mental status change is of paramount importance. Patients with suspected intracranial hemorrhage should then undergo a CT scan of the head to assess severity of intracranial bleeding. There should be an ongoing focus to optimize oxygenation, ventilation, and blood pressure.[4] Ideal parameters for arterial oxygen and carbon dioxide levels, as well as glucose, are similar to those for adults; however, target blood pressure is less clear. In cases of cerebral edema or impending herniation, administration of either mannitol or hypertonic saline is equally acceptable, and the physician should consider their variable effects on blood pressure[7] (**Table 2**). Seizure activity should be treated aggressively. Antiepileptics are commonly used prophylactically owing to concerns of transient intracranial pressure spikes during convulsions, but there is little evidence to support the safety and efficacy of this practice.[4] After patient stabilization, identification of intracranial hemorrhage, and initiation of supportive measures, communication with nearest pediatric neurosurgery and intensive care should ensue to arrange for transfer.

Fever and Altered Mental Status

Bacterial meningitis should not be the only diagnosis considered when a pediatric patient presents with fever and AMS. Other important diagnoses are summarized in **Box 4**.

Acute disseminated encephalomyelitis and anti–N-methyl-ᴅ-aspartate receptor encephalitis

Acute disseminated encephalomyelitis (ADEM) is an acute multifocal demyelinating disease presenting with fever, acute AMS, and neuromotor dysfunction, such as

Table 2
Pediatric management of elevated intracranial pressure

Osmotic Agent	Dose	Frequency	Adverse Effects
Mannitol	0.25–1.00 g/kg IV over 20 min	Up to every 4–6 h	Hypovolemia Acute renal failure Diuresis Decreased BP
Hypertonic saline (3%)	2–5 mL/kg IV given over 10–20 min	Can start continuous infusion of 0.1–1.0 mL/kg/h	Rebound cerebral edema Osmotic demyelination syndrome[a] Increased BP

Abbreviation: BP, blood pressure.
[a] Formerly known as central pontine myelinosis.
Data from Kliegman RM, Stanton BF, St Geme JWI, Schor NF, Behrman RE. *Nelson Textbook of Pediatrics.* 20th ed.; 2016; Kochanek PM, Tasker RC, Carney N, et al. Guidelines for the Management of Pediatric Severe Traumatic Brain Injury, Third Edition: Update of the Brain Trauma Foundation Guidelines, Executive Summary. *Neurosurgery.* 2019;84(6):1169-1178; Stopa BM, Dolmans RG, Broekman ML, et al. Hyperosmolar Therapy in Pediatric Severe Traumatic Brain Injury—A Systematic Review. Critical Care Medicine 2019; 47(12): e1022-e1031.

seizures. Patients are commonly young males, ages 5 to 8 years old, with recent viral illness or immunization. The illness can be idiopathic.[8,9] Patients often seem to be quite ill, and symptoms can progress in hours to days, lasting up to several weeks. The clinical picture is extremely similar to acute bacterial meningitis and a distinguishing feature is contrast-enhanced MRI revealing bilateral, multifocal, asymmetric enhancing lesions on T2-weighted or fluid-attenuated inversion recovery images.[10] Lumbar puncture findings are not diagnostic in ADEM and may reveal elevated proteins and lymphocytic pleocytosis. More important, a lumbar puncture should be done to rule out infectious meningoencephalitis and xanthochromia from intracranial hemorrhage.[8,9]

Box 4
Clinical considerations for pediatric AMS and fever

- Infectious meningitis
- Infectious encephalitis
- ADEM
- Hemophagocytic lymphohistiocytosis
- Anti-NDMAR encephalitis
- Other autoimmune encephalitis
- Stroke (infants and toddlers)
- Toxic ingestions
  - Sympathomimetic
  - Anticholinergic
- Thyrotoxicosis
- Paraneoplastic syndromes

* Not all inclusive.

Anti–N-methyl-D-aspartate receptor (NMDAR) encephalitis is insidious in onset; symptoms progress over weeks. It is more common in prepubertal males or postpubertal females. Classified as an autoimmune encephalitis, second most common only to ADEM, it is typically preceded by a febrile viral-like syndrome. It progresses over the following days to week, and develops into light switch acutely fluctuating mental status changes, dyskinesia, seizures, fever, and autonomic instability.[9] Caregivers may report that the patient does not remember events of the day, has a delay or decrease in expressive language, seems to be restless with tremors or repetitive movements, and may hallucinate. Young children may present with increased frequency of unprovoked tantrums.[11] Given the overlap in symptoms between anti-NMDAR encephalitis and initial presentation of schizophrenia or other psychoses, a thorough history is critical. Early behavioral and mental status changes are often mistaken for a new behavioral or psychiatric disorder, which highlights the importance of a thorough assessment to exclude an underlying organic etiology for the presentation of such patients before psychiatry consultation. When suspected, a full assessment should include a brain MRI, to rule out other entities such as ADEM, and lumbar puncture, to test specifically for anti-NMDAR antibodies.

Both ADEM and anti-NMDAR encephalitis require a high index of suspicion, aggressive seizure control, and early initiation of steroids. Empiric treatment for bacterial and viral meningitis should be given as well. Second- and third-line therapies such as intravenous immunoglobulin, plasmapheresis, and biologic agents such as cyclophosphamide and rituximab are effective, however not indicated in acute ED management. Postpubertal girls (or girls >14 years old) suspected of having anti-NMDAR encephalitis should undergo abdomen and pelvis MRI for the evaluation of possible ovarian mass, because an ovarian teratoma is discovered in up to 40% of these patients.[9]

Extracranial causes of altered mental status

Given the sensitivity of children's nervous systems, the astute physician must also consider that fever and AMS may be a consequence of disease outside of the central nervous system. One of the most common to remember is hypoglycemia, which is not a direct cause of fever, but may be a consequence of febrile illness and concomitant hypermetabolic state. Point-of-care glucose testing should be considered the sixth vital sign for any ill-appearing, altered, or vomiting child. Inborn errors of metabolism such as fatty acid oxidation disorders, carnitine transport disorder, or mitochondrial disease may also present with AMS owing to the metabolic derangements of even a minor febrile illness. Endocrinopathy-like thyrotoxicosis and pheochromocytoma should also be considered, particularly in the setting of tachycardia and hypertension for age. Finally, any anticholinergic or sympathomimetic toxidrome can also present with elevated temperatures and AMS.

Seizures

Seizures are a common clinical entity that emergency physicians must be competent in identifying and managing. Febrile, post-traumatic, and rebound seizures after medication noncompliance are all quite common; however, there are some additional considerations for seizure in pediatrics of which physicians must be aware.

Neonatal seizures

Neonatal seizures occur in 1.0 to 3.5 per 1000 live births, and can have significant consequences if misdiagnosed or mismanaged.[12] The neonatal seizure is defined as seizure activity in a patient less than 28 days old, and can be difficult to recognize owing to the immaturity of the central nervous system. Generalized seizure activity

may present only with mouthing, horizontal eye deviation, blinking, decreased responsiveness, or single limb extension, not with the classic tonic–clonic activity of a more mature brain.[9] Common causes include hypoxic–ischemic encephalopathy, ischemic and hemorrhagic perinatal stroke, electrolyte disturbance, structural brain abnormality, abusive head trauma, or infections. The first-line treatment for neonatal seizures is phenobarbital 20 mg/kg, but if there is a delay in administration, a benzodiazepine can be given.[12] Second-line therapy includes fosphenytoin, levetiracetam, midazolam, and lidocaine (**Table 3**). In refractory neonatal seizures, pyridoxine may be also be effective in some metabolic errors.[9]

Nonconvulsive status epilepticus

Nonconvulsive status epilepticus is another clinical entity of which to be vigilant. Delayed recognition often leads to prolonged hospitalization, prolonged intensive care stays, and long-term morbidity. Nonconvulsive status epilepticus should be in the differential diagnosis when the presenting problem is abrupt onset of lethargy, decreased responsiveness, or AMS, or may be the consequence of inadequate seizure control. Nonconvulsive status epilepticus can also occur after a patient has been intubated for airway protection, has received paralytics, but continues to have epileptiform brain activity. When suspected, a spot electroencephalography is indicated, if available. A trial of a short-acting antiepileptic medication such as midazolam and close observation for change in mental status may also reveal the clinical condition.[12]

NEUROLOGIC EMERGENCIES IN THE GERIATRIC PATIENT
Acute Ischemic Stroke

Prevalence

Stroke is the leading cause of disability in the United States and mostly affects the geriatric population; three-quarters of strokes occur in patients 65 or older,[13] and one-third are in patients greater than 80 years.[14] AIS in older adults is more likely to be secondary to a cardioembolic source (such as atrial fibrillation) than in their younger counterparts.[14,15]

Presentation

Older adults are more likely to have underlying disability at baseline, present atypically, present later to care, and receive less evidence-based care than their younger counterparts.[2,16–18] Some atypical presentations are summarized in **Box 5**. Physicians should maintain a low threshold for suspecting strokes in older adults and obtaining emergent brain imaging for stroke-like and atypical presentations alike.

Table 3
Medications for neonatal seizures

Drug	Dose	Common Adverse Effects
Phenobarbital	20 mg/kg loading dose (max 40 mg/kg)	Respiratory depression, hypotension
Phenytoin	20 mg/kg loading dose	Arrhythmia, central nervous system depression, hypotension
Midazolam	Depends on additional medications being used. Maximum dosing 0.15 mg/kg	Respiratory depression, hypotension May increase length of stay and risk of mortality
Levetiracetam	20 mg/kg	Somnolence

Data from El-Dib M, Soul JS. The use of phenobarbital and other anti-seizure drugs in newborns. *Semin Fetal Neonatal Med*. 2017;22(5):321-327.

Box 5
Atypical AIS presentations in older adults

- AMS
- Dizziness
- Falls
- Headache
- Nausea and vomiting
- Reduced mobility or difficulty walking
- Seizure
- Syncope
- Urinary incontinence

Data from Arch AE, Weisman DC, Coca S, Nystrom K V., Wira CR, Schindler JL. Missed Ischemic Stroke Diagnosis in the Emergency Department by Emergency Medicine and Neurology Services. *Stroke*. 2016;47(3):668-673; Muangpaisan W, Hinkle JL, Westwood M, Kennedy J, Buchan AM. Stroke in the very old: clinical presentations and outcomes. *Age Ageing*. 2008;37(4):473-475; Pare JR, Kahn JH. Basic neuroanatomy and stroke syndromes. *Emerg Med Clin North Am*. 2012;30(3):601-615; Goyal M, Menon BK, van Zwam WH, et al. Endovascular thrombectomy after large-vessel ischaemic stroke: a meta-analysis of individual patient data from five randomised trials. *Lancet*. 2016;387(10029):1723-1731.

Treatment

Treating older adults with systemic tPA or endovascular therapy is a complex decision; they are more likely to have stroke-related death and disability than their younger counterparts, whether they receive treatment or not.[19] The improvement in outcome owing to revascularization treatment seems to be preserved in appropriately selected older adults,[18,20,21] although they may be more likely to have hemorrhagic complications after treatment.[22] Age alone is not a reason to withhold revascularization treatment in older adults.

Systemic thrombolytics

Intravenous tPA has been extensively studied and is standard of care for AIS up to 4.5 hours of last known well with certain exclusions.[23] The data for safety and efficacy of tPA within the 3-hour window are relatively robust, even in patients 80 years or older.[21] However, the evidence is not as clear for the extended time window from 3.0 to 4.5 hours in patients greater than 80 years of age. Although recommended by the American Heart Association as a Level IIa recommendation,[23] based mostly on the Third International Stroke Trial data, experts caution against the use of tPA for octogenarians within the extended window.[24–26] As noted elsewhere in this article, older adults are more likely to have worse outcomes after a stroke and it may be reasonable to quote a higher rate of complications when discussing tPA with the patient and/or family, because the rate of symptomatic intracranial hemorrhage seems to be closer to 10% to 13%, in contrast with 5% to 8% in the younger patients.[22]

Endovascular thrombectomy

AIS secondary to a large vessel occlusion is unlikely to respond to IV tPA.[27] Endovascular thrombectomy (EVT) is the standard of care in patients presenting with anterior circulation large vessel occlusions within 6 hours, and up to 24 hours in those having a

mismatch between the infarct and ischemia on imaging.[23] The American Heart Association guidelines do not cite an upper age limit for EVT,[23] because studies have shown no heterogeneity of treatment effects by age.[18] The patients included in these studies were carefully selected; they all had a good premorbid function with little or no disability. Additionally, some studies excluded older patients (>80 years of age) with larger infarcts because they are more likely to have a poor outcome.[28] Patients with moderate to severe disability at baseline are currently not candidates for EVT, given the scarcity of data in this population.[19] This criterion effectively excludes one-third of octogenarians presenting with AIS.[29] The strict selection criteria of the EVT studies informing the guidelines limits the translation of the positive effects of EVT into clinical care.[30] It is worth noting that the procedure itself may be more technically difficult in older adults given the tortuosity of vasculature and impairment of collaterals. However, it is unclear if this difficulty increases the risk of complications such as dissection.

Altered Mental Status

AMS, a disturbance in brain function manifested by altered consciousness and/or cognition, is common in older ED patients. One-quarter of older adults in the ED have some form of AMS, whether acute or chronic,[31] a percentage that doubles in patients 85 years or older.[32]

Dementia

Dementia is a gradual and progressive significant impairment of one or more cognitive domains, severe enough to interfere with independence in everyday activities.[33] When this impairment is modest and does not limit independence, it is termed "mild cognitive impairment."[33] There are many causes for dementia (**Table 4**) that may coexist at times.[33]

Although dementia is usually diagnosed in the outpatient setting, a patient's first presentation may be in the ED. It is estimated that the number of older adults living with Alzheimer disease, the most common cause of primary dementia, will triple between 2010 and 2050.[14] Furthermore, one-half of the cases of dementia are missed in the outpatient setting.[34] Formal cognitive testing is necessary to exclude dementia, especially in high-functioning individuals, because AMS may not be readily apparent. The Mini-Mental Status Exam is the most widely known cognitive test and may not be feasible in the ED setting owing to its length and complexity.[35] Alternative cognitive tests that have been validated in the ED are summarized in **Box 6** and have varying

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Table 4
Causes of dementia

Primary (Neurodegenerative)	Secondary
Alzheimer disease	Vascular (multi-infarct) dementia
Dementia with Lewy Bodies	Trauma
Frontotemporal lobar degeneration	Infectious
Parkinson's disease	HIV
Huntington disease	Syphilis
	Prion disease
	Drugs and toxins
	Vasculitis
	Intracranial mass/hemorrhage

Abbreviation: HIV, human immunodeficiency virus.
Data from American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* Fifth Edit. Arlington, VA: American Psychiatric Association; 2013.

Box 6
Cognitive tests that may be used in the ED

- Mini-Mental Status
- Mini-Mental Status-2: Brief Version
- Quick Confusion Scale
- Brief Alzheimer's Screen
- Short Blessed Test
- Ottawa 3DY
- Caregiver Completed AD8

Data from Carpenter CR, Bassett ER, Fischer GM, Shirshekan J, Galvin JE, Morris JC. Four sensitive screening tools to detect cognitive dysfunction in geriatric emergency department patients: brief Alzheimer's Screen, Short Blessed Test, Ottawa 3DY, and the caregiver-completed AD8. *Acad Emerg Med*. 2011;18(4):374-384; Folstein MF, Folstein SE, McHugh PR. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*. 1975;12(3):189-198. Folstein MF, Folstein SE, White T, Messer MA. Mini-Mental State Examination, 2nd edition. 2010. Psychological Assessment Resources, Florida; Stair TO, Morrissey J, Jaradeh I, Zhou TX, Goldstein JN. Validation of the Quick Confusion Scale for mental status screening in the emergency department. *Intern Emerg Med*. 2007;2(2):130-132.

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sensitivity, specificity, and usefulness in the ED. Dementia-like illness may occur secondary to depression, hypothyroidism, vitamin B_{12} deficiency, or normal pressure hydrocephalus.[36] If a safe disposition can be ensured and no other acute medical conditions are uncovered (including superimposed delirium, see below), the workup for chronic AMS can be continued as an outpatient.

Delirium

Delirium is another form of cognitive impairment commonly encountered in the ED and is easily missed.[37,38] Delirium is an acute medical emergency and should be treated as such, because it is associated with worsening morbidity (including long-term cognitive effects) and mortality, especially if undiagnosed.[37,39] Delirium may be hypoactive, hyperactive, or mixed, with the hypoactive subtype most frequently missed and therefore associated with the worst outcomes.[40] The hallmark of delirium is inattention, and diagnostic criteria are listed in **Fig. 1**.[33]

Delirium occurs in vulnerable patients with predisposing factors, who are then exposed to 1 or more precipitating insults (**Table 5**). Obtaining a collateral history and a comprehensive medication list are essential. At-risk older adults should be screened for delirium during their ED visit using a validated delirium screening tool such as the Delirium Triage Screen[41,42] (**Fig. 2**). Those who screen positive should then have the diagnosis confirmed using the brief Confusion Assessment Method, which is a stepwise approach through the diagnostic criteria of delirium, or other highly specific tool.[41,42] Flowsheets and videos for both tools are available on the ED Delirium Website (http://eddelirium.org/).

Patients with delirium should have the underlying cause uncovered using a thorough assessment and focused investigations. In addition to treating the underlying cause, there are many nonpharmacologic methods to manage delirium and prevent it, such as providing frequent reorientation, increasing familiar interactions with loved ones, avoiding unnecessary tethering to monitors, and ensuring that patients have their glasses and hearing aids.[42] In the case of hyperactive delirium imminently affecting

All of the following criteria:

Disturbance in Attention

Reduced ability to direct, focus, sustain, and shift attention

Disturbance in Cognition

One or more of the following:
- Memory deficit
- Disorientation
- Language disturbance
- Disturbance in visuospatial ability
- Disturbance in perception

Disturbance in Awareness

Reduced orientation to the environment

- Acute (hours to days), is a change from baseline attention and awareness, and fluctuates in severity during the course of a day.

- Not better explained by a pre-existing established neurocognitive disorder.

- Does not occur in the context of a coma or severely reduced level of arousal.

- Is a direct physiological consequence of another medical condition, substance intoxication or withdrawal, exposure to a toxin, or is due to multiple etiologies.

Fig. 1. Diagnostic criteria for delirium. (*Data from* the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, (Copyright 2013). *American Psychiatric Association.*)

the safety of the patient and/or health care staff, low-dose atypical antipsychotics, such as risperidone or quetiapine, are recommended over other medications.[43] Most patients with delirium will require admission to the hospital for further management and patient safety. Differentiating between delirium and dementia can be

Table 5
Causes of delirium

Precipitating Insults	Predisposing Factors
Infections	Age
Medications/toxins	Dementia
Iatrogenic	Underlying neurologic or psychiatric disorder
Intracranial diseases	(eg, stroke, seizure, schizophrenia)
(eg, stroke, infection, seizure)	Hearing or visual impairment
Cardiovascular diseases	Alcohol or drug use disorder
(eg, acute coronary syndrome)	
Metabolic disorder (eg, uremia)	
Endocrine disorder (eg, thyroid disorder)	
Dehydration	

Data from Wilber ST, Ondrejka JE. Altered Mental Status and Delirium. *Emerg Med Clin North Am.* 2016;34(3):649-665.
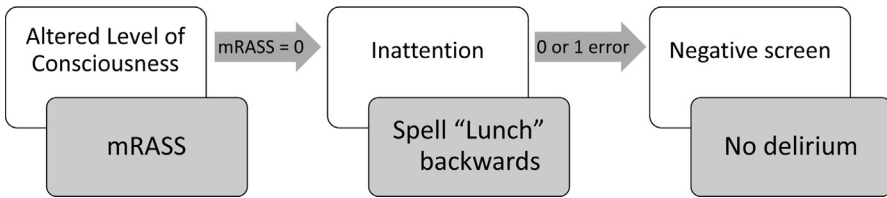
Fig. 2. Delirium triage screen. mRASS, modified Richmond Agitation and Sedation Scale. (*Data from* Geriatric emergency department guidelines. *Ann Emerg Med.* 2014;63(5):e7-25; Han JH, Wilson A, Vasilevskis EE, et al. Diagnosing delirium in older emergency department patients: validity and reliability of the delirium triage screen and the brief confusion assessment method. *Ann Emerg Med.* 2013;62(5):457-465.)

complex, especially because dementia is the most common condition predisposing to delirium.[44] A simple comparison is presented in **Table 6**.

Parkinson's Disease

Parkinson's disease is a neurodegenerative disorder affecting 1% of the population above the age of 60 years.[45] Parkinson's disease is due to a dopaminergic deficiency in the striatum owing to Lewy bodies, which manifests as a variety of motor, autonomic, and psychiatric symptoms,[46,47] and are summarized in **Table 7**.

Patients with Parkinson's disease may be seen in the ED for falls and trauma, medication side effects, acute worsening of symptoms, and psychiatric issues.[48] Falls are common in patients with Parkinson's disease; two-thirds of patients with Parkinson's disease have fallen at least once and 40% have fallen recurrently.[49] Falls occur secondary to a combination of postural instability, postural dizziness, and unstable gait. After excluding significant injuries from the fall, gait stability should be assessed before discharge to ensure a safe disposition,[42] which may require an assessment by a physical therapist.

Medications frequently used for Parkinson's disease treatment and their side effects are summarized in **Table 8**. Levodopa, the mainstay of Parkinson's disease treatment, has a short half-life and fluctuations in its level can cause noticeable symptom worsening. A patient's scheduled home dose should be administered while in the ED for any reason, unless otherwise contraindicated. This factor is also relevant in patients receiving levodopa as a continuous intestinal infusion through a percutaneous endoscopic gastrostomy with a jejunal extension tube who present with malfunction of the tube. Taking a careful medication history is necessary in older adults with Parkinson's

Table 6 Comparison between dementia and delirium		
	Dementia	**Delirium**
Time course	Chronic (months to years)	Acute (hours to days)
Course	Progressive	Fluctuating
Consciousness	Intact (except in late stages)	Altered
Autonomic disturbance	Absent	May be present
Reversibility	Usually irreversible	Usually reversible

Data from American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition.* Washington, DC: American Psychiatric Association; 2013.

Table 7
Symptoms and signs of Parkinson's disease

Motor	Autonomic	Psychiatric
Resting tremor[a]	Postural dizziness (hypotension and tachycardia)	Hallucinations
Rigidity[a] (smooth [lead pipe] or oscillatory [cog-wheel])	Sweating	Dementia
Bradykinesia/akinesia[a]	Constipation	Depression
Postural instability[a]	Urinary incontinence or retention	Sleep disorders
Dystonia	Sialorrhea	Impulse control disorder
Dysphagia		
Stuttering		
Gait disorder		
Masked facies		

[a] Hallmark symptoms, commonly summarized as TRAP (tremor, rigidity, akinesia, postural instability).

Data from Armstrong MJ, Okun MS. Diagnosis and Treatment of Parkinson Disease: A Review. *JAMA.* 2020;323(6):548-560; Zesiewicz TA. Parkinson Disease. *Continuum.* 2019;25(4):896-918.

Table 8
Medications commonly used to treat Parkinson's disease

Class	Examples	Side Effects
Dopamine replacement[a]	Levodopa[b]	Dyskinesia Motor fluctuation (on–off phenomenon) Wearing off
Dopamine agonists[a]	Ropinirole Pramipexole Apomorphine (parenteral)	Hallucinations Somnolence or sleep attacks Edema Orthostatic hypotension Dizziness
Monoaminoxidase B inhibitors	Selegiline Rasalgiline Safinamide Zonisamide	Nausea Sleep disturbance
Anticholinergics	Trihexyphenidyl Benztropine	Delirium Urinary retention Constipation Dry mouth
Catechol-o-methyl-transferase Inhibitors	Tolcapone Entacapone Opicapone	Liver toxicity Diarrhea
NMDAR agonist	Amantadine	Hallucinations Edema Orthostatic hypotension

[a] Similar side effects with differing frequency.
[b] Frequently combined with a peripheral decarboxylase inhibitors (eg, carbidopa) to decrease nausea and prolong half-life.

Data from Armstrong MJ, Okun MS. Diagnosis and Treatment of Parkinson Disease: A Review. *JAMA.* 2020;323(6):548-560; Miyasaki JM, Martin W, Suchowersky O, Weiner WJ, Lang AE. Practice parameter: Initiation of treatment for Parkinson's disease: An evidence-based review: Report of the quality standards subcommittee of the American Academy of Neurology. *Neurology.* 2002;58(1):11-17.

disease, given the propensity of polypharmacy in older adults and the relatively high likelihood of adverse drug events from anti-Parkinsonian medications.[42,50,51] Dopamine receptor blockers, such as antipsychotics and some antiemetics, are best avoided in a patient with Parkinson's disease because they worsen the movement disorder.[52]

Sudden deterioration in a patient with Parkinson's disease occurs secondary to changes in the medication regimen or the addition of new medications, metabolic derangements, concurrent infection (such as urinary tract infection or pneumonia), malfunction of a deep brain stimulator (if present), or a chronic subdural hematoma from a missed fall.[48] Patients present with worsening of rigidity, bradykinesia, and postural instability, and may be completely akinetic. This finding may be associated with dysphagia and dysautonomia as well.[48]

A unique side effect of acutely discontinuing dopaminergic medications is Parkinsonism hyperpyrexia syndrome, which presents similarly to neuroleptic malignant syndrome and should be considered in patients with Parkinson's disease presenting with AMS and a fever. It is clinically indistinguishable from neuroleptic malignant syndrome, with the only difference being the absence of exposure to neuroleptics. Treatment is supportive and includes the reintroduction of antiparkinsonian medications.[48]

Meningitis

Immunosenescence, the decreased ability to fight infections owing to age, renders geriatric patients more prone to infections, different organisms, atypical presentations, and worse outcomes.[53] Therefore, meningitis is more likely to occur in older adults than in their younger counterparts[54,55] and is more likely to be bacterial.[56] Older adults present with less typical symptoms of meningitis when compared with the general population; they are less likely to have a fever,[55,57] neck stiffness,[55,58] rash,[55] or leukocytosis[59] and are more likely to have AMS.[55,60] Older adults are more likely to have a delay in receiving antibiotics[55] and a worsened outcome.[54,55,59,60] Common organisms are listed in **Box 7**.[54,55,60,61] Antibiotics should be started as soon as the diagnosis is suspected, even before completing an lumbar puncture. Empiric antibiotics should include vancomycin and a third-generation cephalosporin (such as ceftriaxone)

Box 7
Common causative organisms for bacterial meningitis in older adults

Streptococcus pneumoniae[a]

Neisseria meningitidis[b]

Listeria monocytogenes[a]

Haemophilus influenzae

Group B streptococcus

Gram-negative rods (eg, *Escherichia coli*, *Klebsiella*)[a]

[a] More likely to occur in older adults than their younger counterparts. [b] Less likely to occur in older adults than their younger counterparts.

Data from Domingo P, Pomar V, de Benito N, Coll P. The spectrum of acute bacterial meningitis in elderly patients. *BMC Infect Dis*. 2013;13:108; Thigpen MC, Whitney CG, Messonnier NE, et al. Bacterial Meningitis in the United States, 1998–2007. *N Engl J Med*. 2011;364(21):2016-2025; Tunkel AR, Hartman BJ, Kaplan SL, et al. Practice Guidelines for the Management of Bacterial Meningitis. *Clin Infect Dis*. 2004;39(9):1267-1284; Weisfelt M, Van De Beek D, Spanjaard L, Reitsma JB, De Gans J. Community-Acquired Bacterial Meningitis in Older People. *J Am Geriatr Soc*. 2006;54(10):1500-1507.

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as in younger patients, in addition to ampicillin, to cover for possible *Listeria*,[61] and acyclovir, to cover for herpes simplex virus.[62] Older adults will require a head CT scan before obtaining a lumbar puncture to exclude an abscess, mass, or other etiologies of their presentation.[61,63] Cerebrospinal fluid findings will be nonspecific in herpes encephalitis and the etiology will only be revealed with targeted polymerase chain reaction testing.[62]

CLINICAL CARE POINTS

- The management of pediatric stroke is in the 3 *T*s: tPA, transfusion, and transfer.
- Use standard weight-based dosing of tPA in pediatric stroke, with limited research thus far demonstrating both safety and efficacy.
- In fever and AMS, consider alternative diagnoses such as anti-NMDA encephalitis or ADEM in children.
- Nonconvulsive status epilepticus is associated with higher mortality, longer pediatric intensive care unit stays, and increased long-term disability.
- Advanced age alone is not a contraindication for IV tPA within 3 hours or EVT
- Delirium is frequently missed and should be screened for using the Delirium Triage Screen.
- Acute worsening in Parkinson's disease is usually due to a medication change, infection, or missed subdural hemorrhage.
- Meningitis in older adults presents atypically and has worse outcomes than the general population.

DISCLOSURE

The authors have nothing to disclose.

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