

# Altered mental status: Cause determines treatment

The workup for AMS can be tough because of the myriad of conditions associated with this disorder. This article provides clinicians with a review and a road map.

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The patient with altered mental status (AMS) poses a challenge to clinicians in a variety of settings, including the family practice office, the emergency department (ED), and the ICU. Because various conditions may produce changes in mental status, providers must have an understanding of the common causes and treatments of AMS. Prompt evaluation and treatment are essential to decrease the morbidity and mortality associated with this condition.

## AMS defined

AMS is an umbrella term closely linked to a variety of other descriptors of mental status, including confusion, delirium, obtundation, stupor, and coma.<sup>1</sup> The term *delirium* carries the most precise medical meaning and will be the primary word used to describe AMS in this article. The diagnosis of delirium is clinical and includes the key features listed in Table 1 (page 18).<sup>2,3</sup>

Not a disease, delirium is rather a clinical entity due to an underlying medical condition and, by definition, resolves with treatment of the underlying disorder. The prevalence of delirium in hospital populations is 10% to 20% and increases among the elderly.<sup>2</sup> A primary analysis found a 35% incidence of delirium among elderly persons living at home and a 33% incidence among those receiving home medical care.<sup>4</sup> Unfortunately, the diagnosis of delirium is often missed. Non-detection rates are in the 33% to 60% range.<sup>5</sup> Because delirium can have both hyperactive and hypoactive forms, it is often mistaken for dementia, epilepsy, depression, or psychosis.<sup>5,6</sup> For example, the hypoactive form of delirium is often characterized by lethargy and psychomotor retardation and may mimic depression. In contrast, the hyperactive form may involve disorientation, memory impairment, psychomotor agitation, and

disturbances in the sleep-wake cycle, which may be interpreted as dementia, hypomania, or frank psychosis. To further complicate the diagnosis, most patients exhibit signs of both forms of delirium.

Certain risk factors predispose a person to develop delirium—most commonly, advanced age, dementia, polypharmacy, bacterial infection, surgery, chronic disease, and the use of eyeglasses or hearing aids (patients not using their eyeglasses or a hearing aid during an evaluation may appear to be delirious). Dementia is a known risk factor, and delirium may unmask a previously unrecognized dementia. However, the clinician should always consider delirium when a patient's mental status has recently changed. The diagnosis of dementia usually requires symptoms to have been present for 6 months or longer.<sup>6</sup>

Those at risk for delirium fall into three categories:

- Patients in whom delirium is primary and no predisposing conditions are present
- Those who have a preexisting neurologic problem, such as Parkinson's disease, that predisposes them to develop delirium
- Patients with another general medical problem that causes them to develop delirium. This is the most common clinical situation, particularly in elderly patients.<sup>2</sup>

## What causes delirium or AMS?

Delirium is a disturbance in consciousness. Consciousness consists of content and arousal. Content relies on the communication of the cerebral hemispheres to integrate and organize information and processes, while the reticular activating system (RAS) and upper brain stem govern arousal. Disturbances in consciousness occur when disease processes interrupt the functioning of content, arousal, or the communication between them.<sup>1,7</sup>

The pathophysiologic mechanism of AMS is unknown, although if a lesion is present, the particular deficits that accompany a change in mental status can

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## KEY POINTS in this article

- ▶ The patient with altered mental status (AMS) can present a challenge to clinicians in a variety of settings, including the family practice office, the emergency department, and the ICU.
- ▶ Patients of different ages present with different types of AMS.
- ▶ Prompt evaluation and treatment can decrease the morbidity and mortality associated with AMS.

yield clues to its location. Acute mental status changes can be crudely classified as either structural or toxic/metabolic in nature. In general, the most acute insults to cerebral functioning produce the quickest changes in mental status. Unilateral mass lesions may produce focal neurologic deficits—for example, left hemiplegia resulting from a right-sided hemorrhagic stroke. Lesions below the tentorium cerebelli cause brain stem ischemia because of compression and may manifest as progressive drowsiness, dilation of the ipsilateral pupil, and oculomotor nerve CN III palsy. Metabolic derangements will usually become evident more slowly and lack localizing signs.<sup>1</sup>

### Who develops AMS?

In a primary retrospective study based in Virginia, Kanich and colleagues describe a population of 317 patients with AMS presenting to the ED.<sup>8</sup> There was a bimodal distribution for the frequency of age occurrence, with one peak in a middle-aged adult about 45 years old and another peak in elderly adults near 78 years old. The mean age of all patients presenting in the study was 49 years. The investigators observed a trend for the most likely diagnosis at discharge; younger patients were more likely to present with traumatic or toxic etiologies, while the elderly patients more likely had suffered a neurologic insult. In addition, patients who were unidentified at time of admission—the so-called “John Doe” syndrome—were more likely to have increased rates of morbidity and mortality associated with their AMS.<sup>8</sup>

### Diagnostic tools

Without proper focus and direction, evaluating the patient with AMS can be overwhelming. The Kanich study evaluated the most useful agents in the workup of patients who presented to the local ED with AMS.<sup>8</sup>

The most valuable tools were history of present illness, medical history, physical examination, and patient’s response to treatment. However, these tools were helpful in only 40% to 50% of cases.

Tests were of less diagnostic value and included radiology ECG, CBC with differential, coagulation studies,

and urinalysis. Clinicians should bear in mind that a negative test result often carries as much weight as a positive test result when confirming or excluding a suspected diagnosis.<sup>8</sup>

Mental status screening tests are integral to the workup of the patient with AMS. Screening evaluations are similar in that they all detect AMS to some degree, but they differ in the amount of time required for assessment as well as in the level of training required to administer the test. Cognitive assessment alone is not reliable for making a diagnosis of delirium because a “below normal” score can be caused by dementia, unwillingness to cooperate, or inability to communicate. The Mini-Mental State Examination (MMSE) takes more than 5 minutes to administer and will generally alert the practitioner only to cognitive impairment. The Confusion Assessment Method (CAM), listed in Table 2 (page 19), is the only rating scale for delirium that has been validated using psychiatric assessment as a reference standard.<sup>6</sup> Completion takes less than 5 minutes, and the CAM has a sensitivity of 94% to 100% and a specificity of 90% to 95%.<sup>5,6</sup> A positive diagnosis requires that both Features 1 and 2 be present, along with either Feature 3 or Feature 4.<sup>2,6</sup>

A prospective interventional study by Hustey and colleagues looked at the effect of screening examinations for mental status impairment on the care of elderly patients presenting to the ED.<sup>9</sup> Adults with delirium were 1.5 times more likely to be hospitalized than those patients without delirium, though the results of mental status screening surveys did not alter care plans for these patients when physicians were notified of their scores. The article did not address the reasoning behind the increased incidence of hospitalization for patients with delirium. The authors demonstrated that emergency physicians often fail to recognize delirium,

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### Learning objectives

- List the risk factors for delirium
- Describe the initial physical assessment of a patient who presents with altered mental status
- Review the most common causes of altered mental status and their prevalence
- Discuss the most appropriate diagnostic tests

as the physicians in this study correctly recognized and diagnosed delirium only 13% of the time. Delirium is a condition that is associated with increased rates of morbidity and mortality. For example, in this study, five patients with unrecognized delirium were discharged home; of these patients, one returned after a fall, two returned to the same ED 3 days later and were admitted, and one with a history of colon cancer was given a new diagnosis of metastatic colon cancer on return to the ED 4 days later. This article demonstrates that clinicians must be familiar with and willing to use appropriate screening tools to assess impaired mental status because they may play an important part in reducing morbidity and mortality.<sup>9</sup>

### Initial assessment and workup

The initial focus should be on stabilizing the patient and assessing the ABCs (airway, breathing, and circulation). Some patients may present with alterations in level of consciousness. The rapid sequence intubation protocol is a safe, quick method of securing the airway before further evaluation.<sup>10</sup> If trauma is suspected, the cervical spine should be immobilized. Next, the assessment should include empiric treatment and a battery of laboratory tests.

The most common empiric treatments are thiamine, naloxone (Narcan), and glucose. The clinician can give thiamine if Wernicke's encephalopathy is suspected in cases of alcoholism; however, glucose should always be administered first. Always obtain a rapid fingerstick glucose test before administering glucose for suspected hypoglycemia, as glucose can increase cerebral metabolism and worsen the damage of a stroke. Naloxone is used in cases of suspected opioid intoxication.

Draw blood for laboratory studies, including a CBC with differential, electrolytes (including magnesium and calcium), coagulation studies, BUN, and creatinine.<sup>10</sup> Other tests may include urinalysis, chest radiography, blood cultures, and 12-lead ECG.<sup>2</sup> Depending on

level of consciousness, other options include placement of arterial lines, Foley catheter, or nasogastric tube to monitor blood gases and urinary output or allow stomach decompression.<sup>10</sup>

### History and physical examination

Once the patient has been stabilized, the clinician must obtain the history of present illness and perform an initial physical examination. The patient with AMS often will not be able to offer a reliable history; family, friends, or caregivers may provide the most important and helpful information regarding onset of symptoms and the patient's recent behavior. Key elements of the history include onset of symptoms; associated symptoms, such as delusions or hallucinations; impairment of functional status; and fever or headache. The most important features of the medical history include current medications, allergies, any chronic illnesses or recent infections, psychiatric history, substance or alcohol use, or recent life-altering events.<sup>11</sup>

A thorough but focused physical examination should follow the history. Vital sign abnormalities, specific odors, and evidence of trauma or neglect should be noted, and a cognitive assessment, such as the MMSE or CAM, should be performed. The mnemonic JIM A MOTSIG (judgment, intelligence, memory, affect, mood, orientation, thought [process and content], speech, insight, and grooming) lends itself to a brief assessment of the patient's mental functioning.

Lastly, perform a complete neurologic examination, paying attention to level of consciousness, cranial nerve testing, muscle tone and strength, presence of tremor, sensory response to pain, deep tendon reflexes, and gait.<sup>11</sup> The Glasgow Coma Scale (GCS) is not used to assess delirium but to determine the patient's level of consciousness (see Table 3, page 21).<sup>2,5</sup> Be vigilant for any focal neurologic deficits and signs of brain herniation (asymmetrical dilated pupils) or meningitis (nuchal rigidity). Less obvious signs of AMS such as mild confusion may occur in the outpatient setting, and cognitive screening tools such as the MMSE may be helpful. In all settings, the history and physical examination should be rapid but complete, as they determine the direction of the workup and allow for the development of a more focused differential diagnosis.<sup>11</sup>

### Differential diagnosis and treatment

The differential diagnosis of AMS is lengthy and complex. There are five major causes of AMS. From the most to the least common, they are drug toxicity/overdose, metabolic derangement, structural abnormality, infectious disease, and psychiatric illness. The remaining causes span a range of organ systems and include endocrine, pulmonary, oncologic, cardiovascular, GI,

TABLE 1

#### Key features of delirium

- Disturbance of consciousness with reduced ability to sustain or focus attention
- Change in cognition, such as memory deficit or perceptual disturbance that is not better accounted for by preexisting or evolving dementia
- Disturbance that developed over a short period of time, usually hours to days, and has a fluctuant nature
- Disturbance likely to be a direct consequence of a general medical condition, based on evidence from the history, physical examination, and laboratory tests

and renal derangements; a detailed discussion involving these etiologies is beyond the scope of this article.<sup>8</sup>

According to O’Keefe and Sanson, the most common causes of AMS in elderly patients were metabolic/toxic (65%), structural (33%), and psychiatric (2%).<sup>11</sup> Kanich and colleagues reported that for 317 patients presenting with AMS in the ED, the most common diagnoses at discharge were neurologic (28%), toxicologic (21%), traumatic and psychiatric (14%), and infectious (10%). The variation between studies may be a result of different study populations, the different ages of subjects, and the likely inclusion of neurologic causes within the structural category in the O’Keefe and Sanson study.<sup>8</sup>

### Drug toxicity or overdose

One of the most common causes of AMS is drug toxicity or overdose. Forty percent of drug intoxication cases result from ingestion of prescribed drugs, so an accurate medication history is of utmost importance.<sup>1</sup> Patients may be unwilling to confirm a drug exposure, so obtaining a urine sample may be helpful.

The clinician must also be aware of various “tox-idromes” and the features seen on presentation. For example, narcotic overdose results in miosis, decreased respirations, and hypotension, while cholinergic toxicity tends to result in bradycardia, salivation, lacrimation, loss of bowel and bladder control, and diaphoresis.<sup>10</sup> Ethanol intoxication is often accompanied by ingestion of other drugs, head injury, liver dysfunction, vitamin deficiency, or Wernicke’s encephalopathy. Clinical signs of Wernicke’s include oculomotor paralysis, nystagmus, ataxia, and signs of malnutrition. Drug intoxication rarely produces focal neurologic signs or seizures, with the major exception being theophylline intoxication or use of drugs that cause hypoglycemia.<sup>1</sup> However, overdoses of tricyclic antidepressants or calcium channel blockers may lead to arrhythmias visible on ECG.<sup>10</sup>

Treatment of drug toxicity should consist of supportive measures as well as administration of a specific antidote, if available. Naloxone may be given for opioid toxicity, N acetylcysteine for acetaminophen toxicity, and bicarbonate for tricyclic overdose. Activated charcoal remains the mainstay of treatment. When treatment is started just a few hours after ingestion of a toxic substance, gastric lavage may be worthwhile. Do not give syrup of ipecac, as it does not result in a significant degree of gastric content removal and the continuous vomiting it produces can prevent proper administration of activated charcoal.<sup>10</sup>

### Metabolic abnormalities

The metabolic abnormalities that lead to AMS can result from various types of underlying pathology. Labo-

TABLE 2

### Confusion assessment method (CAM)

Feature 1: Acute onset and fluctuating course

Feature 2: Inattention

Feature 3: Disorganized thinking

Feature 4: Altered level of consciousness

**Note:** For a positive diagnosis, features 1 and 2 must both be present, and either feature 3 or feature 4 must be present.

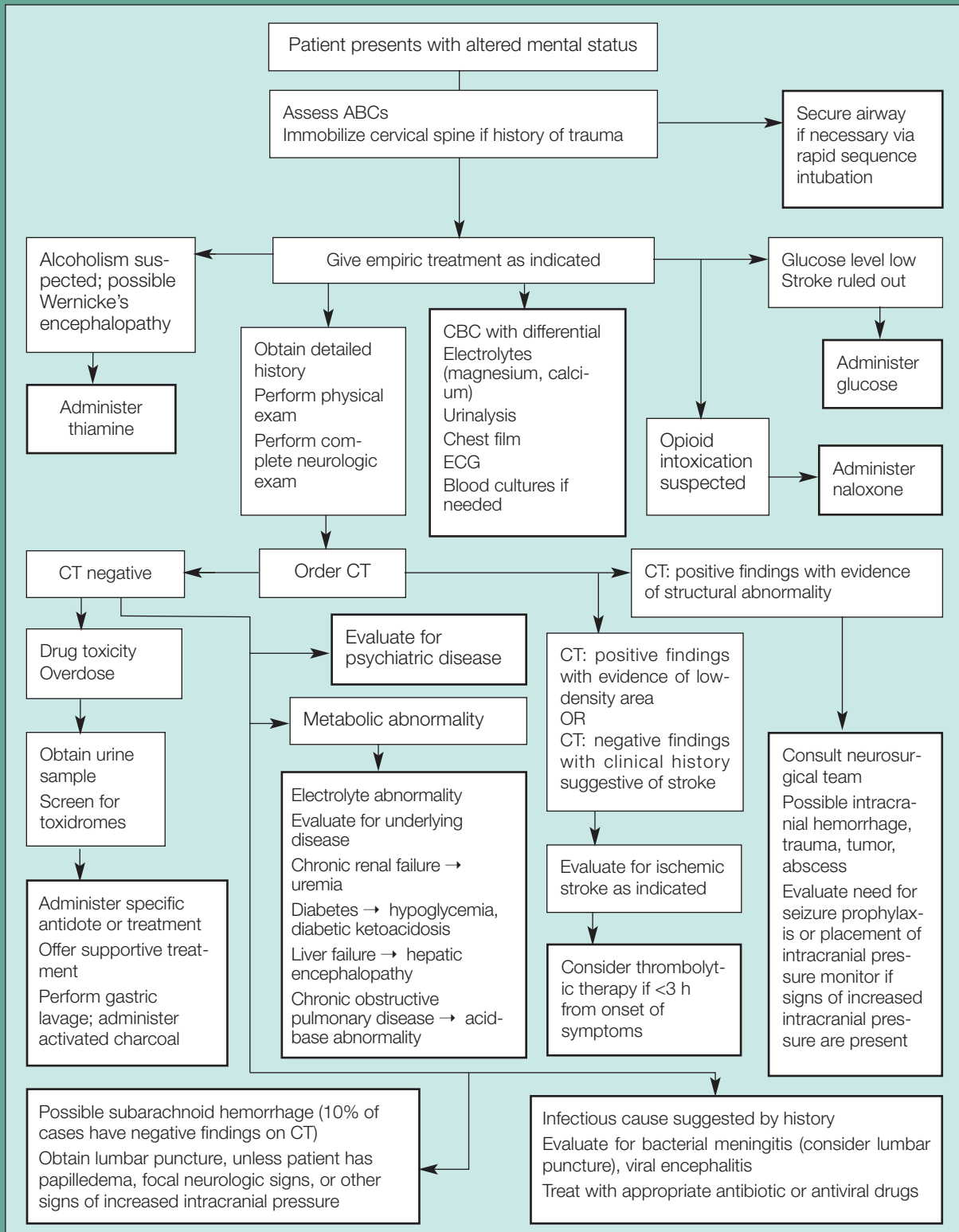
ratory studies drawn at admission should reveal any electrolyte imbalances. Patients with conditions such as chronic renal failure (CRF), chronic liver disease, chronic obstructive pulmonary disease (COPD), and diabetes mellitus are at increased risk for development of metabolic disturbances. Uremia may be encountered in patients presenting at all acute care practices, and affected persons may display confusion, asterixis, muscle twitching, hallucination, and, in some cases, seizures.<sup>1</sup> Hepatic encephalopathy resulting from liver disease may mimic uremia, but an elevated ammonia level precludes that diagnosis. Acid-base disturbances may affect patients with COPD; these persons may display intermittent neurologic symptoms such as drowsiness, inattention to environment, or forgetfulness.<sup>1</sup>

Hypoglycemia may also imitate uremia and hepatic encephalopathy and can actually result from CRF and liver disease, but most patients have a history of diabetes mellitus. The prevalence rate of hypoglycemia is 20% to 30%, but episodes are increasing with patients exercising tighter glycemic control. Most cases of hypoglycemia result from use of hypoglycemic drugs such as insulin or sulfonylureas. Symptoms of hypoglycemia include both adrenergic symptoms (sweating, palpitations, tachycardia, and nausea) and neuroglycopenic symptoms (headaches, visual disturbances, weakness or paresthesia, and seizures or coma). The clinician should be aware that beta-adrenergic antagonists such as propranolol may mask the adrenergic symptoms of hypoglycemia. Treatment of hypoglycemia consists of 50 mL of 50% glucose IV after a glucose level has been drawn, and the patient should be reassessed every 10 to 15 minutes.<sup>12</sup>

Diabetic patients may also develop diabetic ketoacidosis (DKA). Unlike hypoglycemia, which occurs in minutes to hours, DKA takes hours or days to develop and urinalysis reveals the presence of acetone and glucose.<sup>1</sup> Treatment of DKA requires correction of dehydration and hypovolemia, hyperglycemia, ketonemia, and electrolyte imbalances. Most importantly, the provider must find and treat the underlying cause of the DKA. The variety of chronic illnesses that can cause metabolic abnormalities requires a detailed med-

FIGURE 1

## Managing the patient with AMS



ical history, as the information gained this way may give important insight to the cause of problem.

### Structural lesions

A significant number of cases of AMS are caused by structural lesions. Emergent CT is warranted in any patient with a suspected head injury or focal neurologic signs. CT accurately identifies bleeding sources as well as increased intracranial pressure. The clinician should evaluate trauma patients for Cushing's response (bradycardia and hypertension resulting from increased intracranial pressure) and calculate the GCS score. If signs of increased intracranial pressure are present and the CT findings are abnormal, a neurosurgeon should be consulted; the patient may need placement of an intracranial pressure monitor and prophylactic treatment for seizures.<sup>10</sup>

Typical causes of increased intracranial pressure include intracranial bleeding, trauma, tumors, or hydrocephalus. The classic symptoms of intracranial hemorrhage are sudden onset of severe headache, nausea, vomiting, and rapid progression to loss of consciousness; in contrast, those symptoms associated with subdural hematoma may involve less severe headache and evolve over several days.<sup>1</sup> The practitioner should recognize that patients on chronic anticoagulation therapy are at increased risk for intracranial bleeding following moderate head trauma.<sup>13</sup>

If the CT findings are positive but there are no signs of increased intracranial pressure, the practitioner should evaluate the patient for stroke or hypertensive encephalopathy. Severe headache, dizziness, nausea, and vomiting often accompany hypertensive encephalopathy, which may be due to an underlying medical condition such as preeclampsia. A funduscopic examination frequently reveals papilledema.<sup>11</sup> IV labetalol (Trandate) or nitroprusside is the treatment of choice for hypertensive encephalopathy and hemorrhagic stroke; so as to minimize the risk of organ hypoperfusion, BP should be reduced 20% to 25% initially.<sup>10,11,14</sup>

About 10% of patients with subarachnoid hemorrhage (SAH) will have normal findings on CT scan. If the clinical presentation is suspicious for SAH and the findings on CT scan are negative, lumbar puncture (LP) is indicated. If xanthochromia is found, calcium channel blocker therapy with nimodipine (Nimotop) should be initiated to prevent vasospasm. The goal of treatment of SAH-induced hypertension should be to reach pre-morbid levels.<sup>10</sup> In addition, patients with SAH may display unusual ECG findings such as arrhythmias, ST segment abnormalities, and marked U waves, which may help to distinguish the diagnosis.<sup>15</sup>

Cerebral embolism is the most common cause of

ischemic stroke. Any patient with suspected stroke should be evaluated for risk factors, including atrial fibrillation, coronary artery disease, hypertension, and diabetes mellitus. The onset of stroke symptoms may develop over several minutes, whereas an extremely rapid onset of symptoms is associated with cerebral hemorrhage. Unlike hypertensive encephalopathy, ischemic stroke often produces focal neurologic signs.<sup>1</sup> BP associated with ischemic stroke usually returns to pre-morbid levels in a few days. However, systolic BP and diastolic BP should be maintained at less than 185 mm Hg and 110 mm Hg, respectively, to reduce the risk of transformation to hemorrhagic stroke.<sup>14</sup>

If the findings on the CT scan are negative, a metabolic, toxicologic, or infectious etiology is likely. Patients who have normal findings on CT scan on admission should be reevaluated if there is an acute change in mental status in the following 24 hours. Arterial hypoxemia, an uncommon condition, is the classic etiology associated with this circumstance. However, if the patient is adequately oxygenated, the practitioner should consider three other less common etiologies for deterioration of mental status: cerebral fat emboli syn-

TABLE 3  
Glasgow Coma Scale

|                         |      |
|-------------------------|------|
| <b>Eye opening</b>      |      |
| Spontaneous             | 4    |
| To verbal command       | 3    |
| To pain                 | 2    |
| None                    | 1    |
| <b>Verbal response</b>  |      |
| Oriented                | 5    |
| Confused                | 4    |
| Inappropriate words     | 3    |
| Incomprehensible sounds | 2    |
| None                    | 1    |
| <b>Motor response</b>   |      |
| Obeys commands          | 6    |
| Localizes pain          | 5    |
| Withdraws from pain     | 4    |
| Flexion to pain         | 3    |
| Extension to pain       | 2    |
| None                    | 1    |
| <b>Score</b>            | 3-15 |

drome, blunt carotid artery injury, or vertebrobasilar artery thrombosis. Doppler imaging or MRI studies be the tests of choice for these conditions.<sup>16</sup>

Infectious etiologies are less common than structural lesions but equally dangerous for patients presenting with AMS. The most common CNS infections are bacterial meningitis, viral encephalitis, or abscess or empyema. Meningitis and encephalitis may look similar, with symptoms of headache, fever, nuchal rigidity, photophobia, confusion, lethargy or seizures.<sup>1</sup> It may not be easy to demonstrate meningismus in elderly or neonatal populations.

The causative agent of bacterial meningitis varies with age. Pathogens such as *Escherichia coli*, *Klebsiella* species, Group B streptococci, and *Listeria* species are commonly isolated in neonates, while *Streptococcus pneumoniae* and *Nisseria meningitidis* are dominant pathogens in children and adults. Obtunded or comatose patients with fever, recent upper respiratory tract infection, otitis, or known exposure to meningitis should be given empiric therapy pending LP; a good initial choice is ceftriaxone (Rocephin) and ampicillin. The clinician should order CT to exclude mass lesions or brainstem herniation before LP. Two sets of blood cultures should be obtained, and antibiotic therapy should be tailored accordingly, pending the results of CSF cultures.

In contrast to meningitis, most cases of encephalitis are viral; patients with encephalitis display more severe diffuse or focal neurologic signs than do patients with meningitis. The most common cause of viral encephalitis is herpes simplex virus (HSV), followed by arboviruses such as West Nile virus or St. Louis virus. Classic symptoms of HSV encephalitis include personality changes, psychotic behavior, hallucinations, anosmia, and temporal lobe seizures. The association of symptoms reflects the virus' predilection for the frontal and temporal lobes.<sup>1</sup> A 2-week course of acyclovir (Zovirax) reduces the mortality of HSV encephalitis by 30%. Treatment for arbovirus-related encephalitis remains supportive.<sup>10</sup>

Cerebral abscesses and subdural empyemas are focal suppurative collections within the brain that arise from such remote sources as dental or otologic infections, endocarditis, sepsis, or meningitis. Symptoms are specific to the location of the lesion but may include headache, fever, nausea/vomiting, or focal neurologic signs. The diagnosis is made by CT with contrast; LP is not indicated because of the risk of herniation. The common pathogens associated with abscesses/empyemas include anaerobes as well as *S pneumoniae*, *Moraxella catarrhalis*, and Group A streptococci. Treatment consists of broad-spectrum IV antibiotics such as penicillin, ceftriaxone, and metronidazole for 4 to 6 weeks.<sup>10</sup>

## Psychiatric disease

A smaller but significant percentage of patients present with AMS due to psychiatric disease. Persons with schizophrenia may display symptoms of disorganized thoughts and behaviors, hallucinations, and delusions. The clinician should take care to identify the type of hallucination: Auditory hallucinations suggest a psychiatric condition, whereas visual and other sensory hallucinations are more likely to indicate an organic brain disorder.

The practitioner should thoroughly assess a patient for dementia and delirium using screening tools such as CAM and MMSE before diagnosing a new psychiatric illness. As a general rule, psychiatric conditions are more likely in patients younger than 40 years, and alternative diagnoses should be explored for patients older than 50 years with no history of psychiatric disease.<sup>11</sup> Dementia and delirium may mimic psychiatric disease, while the reverse also holds true. Depression may be mistaken for the hypoactive form of delirium, while polypharmacy, a common occurrence with elderly patients, may cause anxiety, hallucinations, confusion, or psychosis.<sup>2</sup>

## Conclusion

A plethora of conditions can result in acute changes in mental status. Initial management of the patient with such changes consists of stabilization followed by a careful history, physical examination, and laboratory studies. "Managing the patient with AMS" (page 20) offers a flow chart to aid in assessment and treatment. □

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