Being faced with an unconscious patient in the emergency department can be particularly daunting, so one needs an easy-to-remember, safe and user-friendly approach that can be tailored to each patient. Although coma in its various manifestations is one of the most challenging and interesting subjects in all of clinical medicine, a full academic discussion of the subject is beyond the intended scope of this brief article. For those with a deeper interest in the subject, various chapters in textbooks of medicine or neurology may be consulted, or particularly the classics in the field – *Diagnosis of Stupor and Coma*, by Plum and Posner, and *Coma and Impaired Consciousness*, by Young, Ropper and Bolton.1,2

**Definition.** Coma may be defined as a sleep-like state of diverse etiology and pathogenesis from which the patient cannot be aroused.

**The Mnemonics of Coma.**

**Clinical approach.** Although subdivided into various headings, the clinical approach to coma is really a continuum, which can be guided by several useful mnemonics.

**History.** Comatose patients are, by definition, unable to supply a history in the way that their conscious counterparts might, but the history is no less important — it just has to come from other sources. This first phase of the “detective work” associated with coma management has to be performed simultaneous with, or even after, handling the most immediate threats to the patient’s life, as described in the following paragraphs. Pre-hospital personnel may provide valuable information about gas fumes, drug paraphernalia, pill bottles, suicide notes, or dirty and disorganized conditions in the patient’s home that
would suggest decompensation or gradual decline in function. Also ask them about pre-hospital interventions and the patient’s response to them. Family members and friends may know the progression of the patient’s symptoms, previous illnesses and drug use, both prescribed and otherwise. Medic Alert® bracelets or wallet cards may yield useful information and expedite retrieval of the hospital record may be invaluable.

**Resuscitation and stabilization.** Every comatose patient is potentially in a life-threatening situation, regardless of the cause. Assessment and intervention needs to be done simultaneously, so one should assemble an appropriate team of assistants. An approach that is both easy to remember and perform is the “A-B-C-D₂-E” approach used for trauma patients (Table 1).³ Immediate assessment of the patient’s airway, breathing and circulation is critical to address and correct any deficiencies as they are diagnosed and anticipating deficiencies that may arise if these are left unattended.

The gag reflex, traditionally used as a method of assessing the ability to protect one’s airway, is absent in up to 37% of healthy patients,⁴ and observation of spontaneous periodic swallowing action by the patient is a more reliable means of assessment. The cervical spine cannot be cleared clinically in an unconscious patient, and it is prudent to manage the patient as if they have an unstable cervical spine until such an injury has been excluded. Patients with a Glasgow Coma Scale (GCS) of less than eight, or in whom airway compromise is a concern, should have a definitive airway obtained without delay (Table 2). At the same time as the clinical assessment of the ABC’s is taking place, medical staff need to obtain intravenous access, a high flow non-rebreather oxygen supply, cardiac and oxygen saturation monitoring and, as soon as feasible, a 12-lead electrocardiograph (ECG), urinary catheterization and dip.

“D₂.” The “D’s” of the mnemonic stands for “drugs” and “disability.” The “drugs” used in the resuscitation phase of coma management represent the empiric use of the so-called “coma cocktail,” remembered by the mnemonic “DON’T (dextrose, oxygen, naloxone, thiamine)” (Table 3). Although this approach has received criticism recently for a number of reasons, it remains safe and easy to remember, and may be particularly helpful for the practitioner who does not manage coma frequently.⁵

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**Table 1**

<table>
<thead>
<tr>
<th>The “ABCD₂E” Approach To COMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
</tr>
<tr>
<td>D₂</td>
</tr>
</tbody>
</table>

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Dr. Campbell is assistant professor of emergency medicine, Dalhousie University, and the Queen Elizabeth II Health Science Centre, Halifax, Nova Scotia.

Dr. McCormick is associate professor of neurology, Dalhousie University, and the Queen Elizabeth II Health Science Centre, Halifax, Nova Scotia.
“DON’T:” Dextrose, oxygen, naloxone, thiamine.

Dextrose. Hypoglycemia is one of the commonest causes of coma in the ED, and the majority will respond dramatically to intravenous dextrose. In patients where hypoglycemia has been prolonged, or in whom seizures have occurred, response may be slower.

Evidence in patients who have suffered ischemic strokes suggests that outcome is adversely affected by hyperglycemia, so a high or high/normal blood sugar may allow one to omit this step. However, recoverable ischemic strokes are a less common cause of coma and rapid reagent chemstrips may fail to recognize clinical hypoglycemia in the presence of numerical normoglycemia. In addition, giving dextrose to an already hyperglycemic patient is far less dangerous than withholding it from a hypoglycemic patient, so, if in doubt, dextrose, 50 mL of D50W should be given.

Oxygen. Oxygenation should be maximized in all critically ill patients during the initial assessment. Concerns about suppression of the hypoxic drive in patients with chronic lung disease should never prevent this important step. Hypercapnea is far less likely to cause irreparable brain damage than is hypoxia.

Naloxone (0.4 – 2 mg intravenous [IV]). This opioid antagonist is relatively inexpensive, well-tolerated and easy to administer. With the current high prevalence of opioid use and abuse, opioid overdose is another common cause of coma. In obvious opioid coma, the clinician may prefer to titrate the dose of naloxone to a quantity enough to make the patient breathe without reversing the entire opioid load. This will avoid precipitating acute opioid withdrawal and abrupt (unauthorized) patient departure that may be followed by a recurrence of coma as the naloxone wears off before the opioid. Synthetic opioids may respond only to higher than usual doses of naloxone, so using higher doses in suspicious cases may be indicated. Certain opioids (e.g., meperidine) or encephalopathy due to cerebral hypoxia after any opioid overdose may not cause the classic
pupillary constriction of opioid overdose, so the specific toxidrome need not be present to prompt naloxone administration.

**Thiamine.** The routine administration of thiamine (100 mg IV) to avoid precipitation or treat Wernicke’s encephalopathy following dextrose loading in coma of unknown origin has been questioned. It is very well-tolerated, however, and is relatively inexpensive. Wernicke’s encephalopathy is common, dangerous and frequently misdiagnosed.10

The use of flumazenil (a benzodiazepine antagonist) is mentioned only to warn against its use in coma of unknown origin.11 In cases of mixed overdose, ingested benzodiazepines may protect against seizure and arrhythmias predisposed to by co-ingestants. Numerous reports have described fatal uncontrollable seizures or arrhythmias with the use of flumazenil in patients who had taken mixed overdoses.12,13

“Disability” represents early evaluation of the level of coma, in order to initially identify and follow changes in the patient’s level of consciousness. The GCS, although originally developed for use in head trauma, is widely used to describe the level of consciousness in all types of coma.3

The “E” of the “ABCD2E” stands for “expose,” remembering to consider, and avoid, hypothermia.

**Exposing** essentially means conducting a thorough examination of the patient. The best way to ensure that nothing is missed is to start at the top of the patient and end at the toes, making sure to palpate the entire surface of the body to detect any clues that may explain the condition. This will also help identify injuries that may become life or limb threatening, as well as those that may be left untreated until the patient wakes up after a prolonged stay in the intensive care unit (e.g., broken or dislocated fingers). Each system and every orifice should be systematically examined and, after X-rays where indicated, every joint put through a full range of motion. A log roll to examine the back should be performed once the anterior and sides of the patient have been examined. Particular attention should be paid to the pupillary and other aspects of the central nervous system (CNS) examination to ensure early diagnosis of a herniation syndrome. Other clues to look for include track marks (in IV drug users), petechial bruises on the abdomen or thighs (insulin-dependent diabetics), the smell of the breath (e.g., almonds — cyanide, acetone — diabetic ketoacidosis [DKA], or alcohol, etc). Features suggestive of a particular drug toxidrome may be found.14

The ABCs should be re-evaluated regularly, especially if the patient’s condition changes, or if he/she has been moved.
## Table 4

**“TVINS in the EMD–LEAFS”**

<table>
<thead>
<tr>
<th>Causes of COMA above the neck</th>
<th>Causes of COMA below the neck</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Trauma</strong></td>
<td><strong>Essentials</strong> (vital signs): Temperature $\uparrow$ or $\downarrow$, BP $\uparrow$ or $\downarrow$, Pulse $\uparrow$ or $\downarrow$, RR $\downarrow$, Glucose $\uparrow$ or $\downarrow$, SaO$_2$ $\downarrow$.</td>
</tr>
<tr>
<td><strong>Vascular</strong></td>
<td><strong>Metabolic:</strong> Lytes (Na$^+$, K$^+$, Ca$^{++}$, etc. $\uparrow$ or $\downarrow$)</td>
</tr>
<tr>
<td><strong>Infective</strong></td>
<td><strong>Endocrine</strong> (Thyroid, PTH, Adrenal, etc. $\uparrow$ or $\downarrow$)</td>
</tr>
<tr>
<td><strong>Neoplastic</strong></td>
<td><strong>Acid/base</strong></td>
</tr>
<tr>
<td><strong>Seizures</strong></td>
<td><strong>Failures</strong> (Brain, heart, lung, kidney, liver, etc.)</td>
</tr>
<tr>
<td></td>
<td><strong>Sepsis</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Drugs/Diet</strong></td>
</tr>
</tbody>
</table>

Adapted from Kassen D: St Paul's Hospital, Vancouver. Personal communication.
Further Measures

Differential diagnosis. By the time the ABCs have been secured and the D2 and E completed, a diagnosis will often have become apparent. If not, a wider differential diagnosis must be considered — another opportunity where a mnemonic may help a great deal. Although several are available, a uniquely Canadian version is: “TVINS in the EMD” (emergency medicine department). The “M” is further subdivided into “LEAFS” (a nod to Toronto hockey fans) (Table 4). Use of this mnemonic reminds physicians of diagnoses that need to be ruled out.

By considering the various mechanisms by which each category could render a patient unconscious, the differential can be further narrowed down and one may return to the patient for specific, goal-
directed reevaluation. By this time, relevant consultants may have been contacted. **Empiric treatment.** As soon as a diagnosis is made, appropriate management and disposition decisions can be reached. In many cases, however, in spite of much “detective work,” the cause of the coma remains elusive. Recognizing the potential seriousness of delays in instituting treatment, further empiric therapy (in addition to the coma cocktail) must now be considered in light of possible causes. The goal is to treat the most life-threatening possibilities with the least dangerous treatments, as soon as possible. One will notice that, apart from blood glucose, urinary dip test, oxygen saturation monitoring and ECG, ancillary tests have yet to be mentioned. This is because, although they may have been done consecutively during initial resuscitation and evaluation phases, they should never be allowed to delay indicated empiric therapy. Broad-spectrum antibiotics, which cross the blood-brain barrier, should be initiated immediately if sepsis or meningitis is a possibility. Consider the use of acyclovir for possible herpes encephalitis. Other examples of empiric treatment to consider include dexamethasone for suspected adrenal crisis, judicious thyroxine administration for suspected myxedema, or specific antidotes appropriate for drugs suggested by a particular toxidrome. Toxicologic causes of coma (beyond the scope of this article) are very common, and should always be suspected. Early contact with a poison center should be considered. **Ancillary investigations.** At this stage, after the initial “ABCD$_2$E” phase has been completed, a full physical examination performed and appropriate empiric treatment initiated, further investigations may be carried out, usually in consultation with an appropriate specialist (Table 5).

**Conclusion**

Although the management of comatose patients can be intimidating, it need not be complicated. A simple, systematic approach will go a long way to ensure adequate stabilization, diagnosis and initial empiric treatment. [CME]

References
15. Kassen D: St Paul’s Hospital, Vancouver. Personal communication.