Mr. R is a 44-year-old man with no significant medical history who presented to the emergency department complaining of severe headache. Non-contrast head computed tomography revealed a subarachnoid hemorrhage (SAH) in the right basal cistern extending to the sylvian fissure and ventricular system. Upon hospitalization, an external ventricular drain was placed. Three days later, brain magnetic resonance imaging (MRI) revealed T2 prolongation in both hippocampi (greater on the right than left), extending into the posterior subiculum bilaterally.

One week after Mr. R’s admission, the psychiatry consultation service was asked to evaluate for agitation. At that time, Mr. R was in restraints and made inappropriate, profane, and sexual remarks throughout the interview. Mental status was notable for psychomotor agitation, poor attention, and orientation to name only. A diagnosis of delirium was made, and the patient was prescribed quetiapine and valproic acid. Mr. R’s agitation and inappropriate behaviors remitted completely by the third week of hospitalization.

As Mr. R’s sensorium cleared, deficits to short-term memory became more apparent. Specifically, Mr. R was unable to retain new information. He could not recognize his doctors and nurses (whom he saw daily) and asked repeatedly why he was in the hospital. At times, he ate meals twice, forgetting that he had eaten once before. One month into admission, repeat brain MRI revealed the hippocampi to be normal in signal intensity but with interval volume loss; the neuroradiologist hypothesized these findings to be the sequelae of previously seen diffusion abnormalities.

Several days prior to his discharge, Mr. R scored 22 of 30 on a Montreal Cognitive Assessment (normal cognition ≥ 26). He could not recall any of five words after a 3-minute delay and could not remember the date, month, or year. Although his acute medical, neurological, and psychiatric symptoms largely resolved during the first month of admission, Mr. R’s memory loss persisted for the entire 59-day hospitalization. Short-term memory impairment posed several challenges to clinical management. For example, his loss of memory for basic activities of daily living required close supervision. Mr. R’s inability to recall the reason for hospitalization led to irritability and at times agitation, requiring frequent re-direction by sitters and nurses. Disposition planning posed a unique challenge, as amnesia prevented Mr. R from living independently, and the paucity of other deficits made it difficult to seek discharge to a personal care or group home.
The well-known case of Henry Molaison (HM), for whom resection of the medial temporal lobes for management of intractable seizures resulted in permanent memory loss, underscores the profound impact of bilateral hippocampal injuries. Postoperative studies of HM have provided valuable insights to the understanding of neural networks critical to memory. Subsequent research has explored nuances of these types of insults. One case series outlines individuals with similar degrees of hippocampal damage and the considerable variation in their clinical presentations, including several who sustained no memory loss at all. In this case, our patient had complete anterograde memory loss following SAH, which resulted in significant challenges in the general hospital setting in which we saw him.

DISCUSSION
Memory impairment has been recognized as a potential long-standing consequence of SAH, with almost 37% of patients reporting memory deficits and up to 6% reporting severe and persisting memory changes. Although mild memory loss is noted in multiple studies, profound loss of short-term memory and anterograde amnesia are rare. Hippocampal injury after SAH has been reported both in vitro and in animal models, although precise mechanisms are not clearly understood. The hippocampus appears to be more vulnerable to blunt head injury, with post-traumatic microscopic damage evident on autopsy even in the absence of increased intracranial pressure. Ischemia due to vasoconstriction has been hypothesized as one possible etiology. Autopsy data also support the contribution of necrotic injury from intracellular biochemical pathways, to which hippocampal cells are more susceptible.

Despite studies supporting hippocampal susceptibility to intracranial bleeds, few cases have been reported of memory loss in live patients as a direct result of subarachnoid hemorrhage, and what has been mentioned is inconsistent in regard to affected neuroanatomy and observed deficits. Studies have implicated the basal forebrain, with one in particular noting amnesia after basal forebrain lesions in five patients; the authors hypothesize that memory impairments were the result of disruption in frontal-hippocampal memory pathways. Another case report presents a 20-year-old man with persistent memory loss and a variety of other psychiatric syndromes (eg, compulsive eating, hypersonnia, executive dysfunction) after endoscopic third ventriculostomy, although this procedure was not undertaken for a hemorrhage.

A more recent investigation of brain imaging after subarachnoid hemorrhage demonstrated lower hippocampal volumes in post-hemorrhage patients compared with matched controls; although neuropsychological symptoms correlated with hippocampal loss, changes to memory were inconsistent. Similarly, hippocampal volume loss was described in a small sample (n = 37) of patients after traumatic brain injury (for which the mechanism was unspecified), although in this case volume loss was associated with higher rates of mood disorder and not with persistent cognitive deficits.

CONCLUSION
Memory loss after subarachnoid hemorrhage presents unique challenges for treatment providers in the general hospital. As patients who experience post-hemorrhage amnesia often present with other neuropsychiatric symptoms, including delirium, agitation, and lesion-specific behavioral disturbances, the memory loss may not be detected immediately. Delayed identification may lead to further complications, including unintended postponement of rehabilitative treatments and misattribution of agitation in patients who cannot identify how or why they have been admitted to the hospital. In the face of such trials, educating treatment providers, staff, and family about the impacts of subarachnoid hemorrhage and hippocampal injury becomes imperative.

REFERENCES
4. Scoville WB, Milner B. Loss of recent


